Idiopathic ventricular arrhythmias originating from the moderator band: Electrocardiographic characteristics and treatment by catheter ablation

Mouhannad M. Sadek, MD,[1] Daniel Benhayon, MD,[1] Ravi Sureddi, MD, William Chik, MD, Pasquale Santangeli, MD, Gregory E. Supple, MD, Mathew D. Hutchinson, MD, FHRS, Rupa Bala, MD, Lidia Carballeira, MD, Erica S. Zado, PA-C, FHRS, Vickas V. Patel, MD, PhD, David J. Callans, MD, FHRS, Francis E. Marchlinski, MD, FHRS, Fermin C. Garcia, MD

From the Section of Cardiac Electrophysiology, Cardiovascular Division, Department of Medicine, Hospital of the University of Pennsylvania, Philadelphia, Pennsylvania.

BACKGROUND The moderator band (MB) can be a source of premature ventricular contractions (PVCs), monomorphic ventricular tachycardia (VT), and idiopathic ventricular fibrillation (IVF).

OBJECTIVE The purpose of this study was to define the electrocardiographic (ECG) characteristics and procedural techniques to successfully identify and ablate MB PVCs/VT.

METHODS In 10 patients with left bundle branch block morphology PVCs/VT, electroanatomic mapping in conjunction with intracardiac echocardiography (ICE) localized the site of origin of the PVCs to the MB. Clinical characteristics of the patients, ECG features, and procedural data were collected and analyzed.

RESULTS Seven patients presented with IVF and 3 presented with monomorphic VT. In all patients, the ventricular arrhythmias (VAs) had a left bundle branch block QRS with a late precordial transition ($>V_4$), a rapid downstroke of the QRS in the precordial leads, and a left superior frontal plane axis. Mean QRS duration was 152.7 ± 15.2 ms. Six patients required a repeat procedure. After mean follow-up of 21.5 ± 11.6 months, all patients were free of sustained VAs, with only 1 patient requiring antiarrhythmic drug therapy and 1 patient having isolated PVCs no longer inducing VF. There were no procedural complications.

CONCLUSION VAs originating from the MB have a distinctive morphology and often are associated with PVC-induced ventricular fibrillation. Catheter ablation can be safely performed and is facilitated by ICE imaging.

KEYWORDS Premature ventricular contractions; Moderator band; Idiopathic ventricular fibrillation; Intracardiac echocardiography; Catheter ablation

ABBREVIATIONS **APM** = anterior papillary muscle; **ECG** = electrocardiogram; **EGM** = electrogram; **ICD** = implantable cardioverter-defibrillator; **ICE** = intracardiac echocardiography; **IVF** = idiopathic ventricular fibrillation; **LBBB** = left bundle branch block; **LV** = left ventricle; **MB** = moderator band; **PF** = Purkinje fiber; **PVC** = premature ventricular contraction; **RB** = right bundle; **RBBB** = right bundle branch block; **RV** = right ventricle; **TTE** = transthoracic echocardiography; **VA** = ventricular arrhythmia; **VF** = ventricular fibrillation; **VT** = ventricular tachycardia

Introduction

Idiopathic premature ventricular contractions (PVCs) and ventricular tachycardia (VT) most commonly originate from the outflow tract region of the heart.[1,2] Other sites of origin include the basal left ventricle (LV), the LV ostium, the right ventricular (RV) and LV intracavitary structures, and the RV inflow region.[3–6] There has been increasing recognition of PVCs as a potential cause of ventricular fibrillation (VF) in patients with no structural heart disease, referred to as idiopathic ventricular fibrillation (IVF).[7–10]

The moderator band (MB) in the RV extends from the septum to the free wall (Figure 1). It is a muscular structure encompassing RV Purkinje fibers (PFs) (Online Supplemental Figure 1). In this report, we describe patients with ventricular arrhythmias (VAs), including IVF, that were mapped to and ablated targeting the MB.

This work was supported in part by the F. Harlan Batrus Research Fund. Drs. Marchlinski, Garcia, and Callans received research grant support and honoraria from Biosense Webster on topics unrelated to the content of this study. **Address reprint requests and correspondence:** Dr. Fermin Garcia, Hospital of the University of Pennsylvania, 9 Founders Pavilion–Cardiology, 3400 Spruce St, Philadelphia, PA 19104. E-mail address: fermin.garcia@uphs.upenn.edu.

[1]Drs. Sadek and Benhayon contributed equally to this work.

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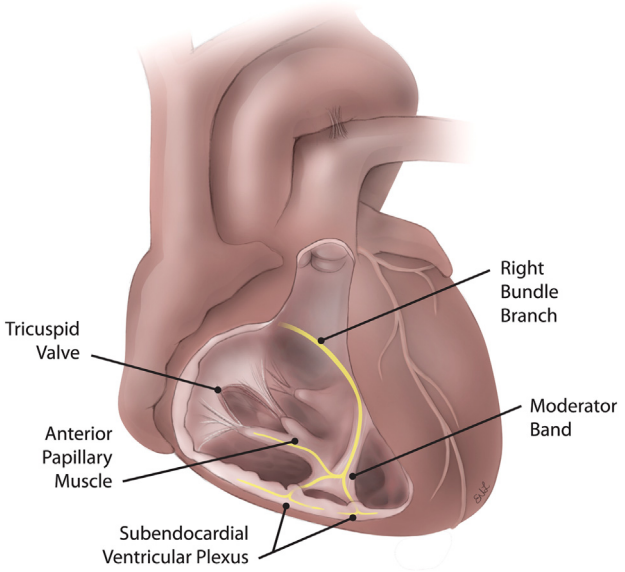

Figure 1 The moderator band crossing from the septum to the free wall of the right ventricle and supporting the anterior papillary muscle of the tricuspid valve.

Methods

Patients with VAs mapped to the MB, directly visualized on imaging with intracardiac echocardiography (ICE) during the electrophysiologic study, are included in this report. Baseline demographic and clinical patient characteristics, including age, sex, LV ejection fraction, presence of structural heart disease, comorbidities, baseline electrocardiogram (ECG), mode of presentation, and PVC/VT morphology were collected. All patients underwent evaluation with transthoracic echocardiography (TTE) and/or cardiac magnetic resonance imaging before electrophysiologic study to assess for structural heart disease and the presence of myocardial fibrosis. The study protocol was conducted in accordance with the University of Pennsylvania research committee guidelines. All data were prospectively entered in a database that was approved by the University of Pennsylvania institution review board.

Electrophysiologic study and ablation

After providing informed consent, patients underwent electrophysiologic study and ablation in the postabsorptive state under minimal sedation. All procedures were performed in accordance with the University of Pennsylvania institutional guidelines. When feasible, beta-blockers, calcium channel blockers, and membrane active antiarrhythmic medications were discontinued for 5 half-lives before the study, or intravenous antiarrhythmic medications were stopped 12 hours before the procedure. Standard surface ECG leads were applied during each procedure.

A 3.5-mm irrigated-tip catheter (ThermoCool, Biosense Webster, Diamond Bar, CA) was used for mapping and ablation. A steerable sheath placed at the tricuspid annulus (Agilis NxT, St. Jude Medical, St. Paul, MN) was used in some patients to assist with catheter manipulation and stability. In some patients, a quadripolar catheter was positioned in the RV and/or high right atrium for pacing. An 8Fr (AcuNav, Siemens Medical Solutions, Mountain View, CA) or 10Fr ICE probe (SoundStar, Biosense Webster) was advanced into the right atrium and RV to define the anatomy, facilitate mapping, and assess contact during ablation in all patients. A CARTO electroanatomic mapping system (Biosense Webster) was used, and online image integration and 3-dimensional anatomic reconstruction (RV, MB, and papillary muscles) was performed in 7 patients using phased-array ICE integration (CartoSound Module, Biosense Webster). Detailed endocardial voltage maps were created using previously established values to define normal RV bipolar and unipolar voltage.[11,12] Bipolar (bandpass filtered at 16–500 Hz) and unipolar (bandpass filtered at 2–240 Hz) electrograms were recorded and displayed at 200 mm/s sweep speed. Wilson central terminal was used as an indifferent electrode to record unipolar electrograms.

Spontaneous PVCs/VT were identified at baseline when present. If none were noted, isoproterenol infusion (2–20 μg/min) and/or burst pacing from the RV apex, RV outflow tract, and/or high RA were performed in attempt to provoke VAs. When PVCs/VT were present or inducible, detailed activation mapping was performed using the ablation catheter and/or a multielectrode mapping catheter (Pentaray, Biosense Webster). Local activation times and pace-mapping were used to approximate the site of origin. The electrogram (EGM) characteristics at the sites of successful PVC elimination were recorded and analyzed. At these sites, radiofrequency applications were delivered at 20–40 W of power with tip temperature limited to 42°C, targeting an impedance drop of 10–15 Ω. Ablation was deemed successful with immediate suppression and in the absence of spontaneous or inducible PVCs/VT after repeating the induction protocol with isoproterenol and/or ventricular pacing. A minimum 60-minute waiting period was observed after the successful elimination of the VA.

ECG analysis

A twelve-lead ECG of the VA was recorded at a sweep speed of 100 mm/s during the procedure and analyzed offline using digital calipers. The morphology was evaluated with respect to QRS duration, amplitude, morphology, and precordial vector transition.

Follow-up

Postprocedure follow-up included inpatient telemetry, extended outpatient rhythm monitoring, implanted device interrogations, and follow-up clinic visits. Patients with arrhythmia recurrence were brought back to the electrophysiology laboratory for repeat study and ablation, which was performed in a similar manner to the first procedure. All patients underwent TTE after the procedure.