

# Postinfarction monomorphic ventricular tachycardia originating from the moderator band



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## Introduction

Ventricular arrhythmias arising from right ventricular (RV) intracavitary structures, consisting of the moderator band and papillary muscles, are generally considered to be idiopathic and often serve as triggers for ventricular fibrillation. The moderator band is a complex structure that houses the right bundle and receives its blood supply from septal perforators of the left anterior descending artery (LAD). We report a case of scar-associated monomorphic ventricular tachycardia arising from the moderator band in a patient with ischemic cardiomyopathy.

## Case report

A 49-year-old man with no prior cardiac history presented with a witnessed out-of-hospital cardiac arrest from rapid ventricular tachycardia (VT) (200 beats/min) and was successfully defibrillated. Transthoracic echocardiogram showed ejection fraction of 20%–25% with apical akinesis and a laminated apical thrombus. Coronary angiogram was performed showing chronic total occlusion of the LAD and no intervention was performed. During the hospital stay, he suffered an in-hospital arrest from sustained VT that progressed to ventricular fibrillation requiring cardiopulmonary resuscitation and external defibrillation. Amiodarone was given intravenously and an intra-aortic balloon pump was placed. A dual-chamber implantable cardioverter-defibrillator (ICD) was implanted. He was transferred to our facility for further management of VT storm.

He underwent repeat coronary angiogram and mid LAD percutaneous coronary intervention was performed owing to absence of collaterals from the right coronary artery. He was taken to the electrophysiology lab and VT was noninducible. Substrate modification was performed using an epicardial approach only, owing to the presence of laminated thrombus. He was discharged on oral amiodarone therapy

**KEYWORDS** Ablation; Electroanatomic mapping; Intracardiac echo; Moderator band; Ventricular tachycardia  
(Heart Rhythm Case Reports 2019;5:445–447)

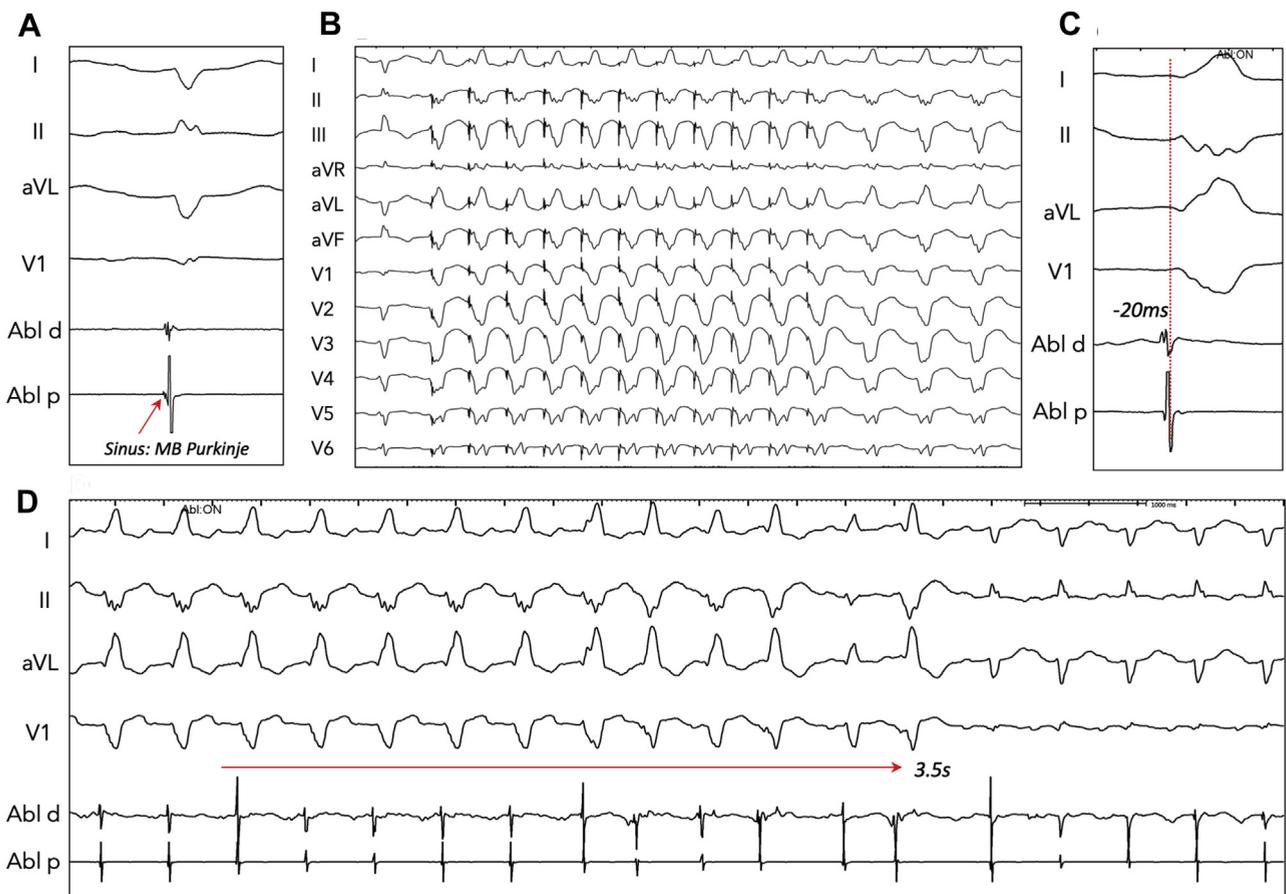
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## KEY TEACHING POINTS

- Ventricular arrhythmias arising from the moderator band typically occur in patients without structural heart disease.
- The moderator band, a prominent endocavitary band of tissue in the right ventricle, receives its blood supply from the septal perforators of the left anterior descending artery (LAD).
- Occlusion of the LAD with development of septal infarct may result in scar-mediated ventricular arrhythmias with involvement of the moderator band.

and anticoagulation. Four weeks later, he presented with syncope and multiple ICD shocks. Emergent coronary angiography showed a patent LAD stent and coronaries. After resolution of the thrombus was confirmed by transthoracic echocardiogram, endocardial mapping was performed during sinus rhythm and showed dense scarring and late potentials in the apical septum. The baseline HV interval was 39 ms. During mapping of the anteroseptal scar, clinical VT was spontaneously induced with a cycle length of 320 ms, left bundle, left superior axis morphology with dominant S waves across the precordium. ICD interrogation during the VT matched the clinical VT. Pace mapping from multiple regions of pathologic electrograms from the left ventricular apex did not yield a good match with clinical VT. Limited activation mapping also failed to identify any presystolic activation sites from within the left ventricle. Retrograde activation of the His was observed, making bundle branch reentry less likely.

Owing to the left bundle QRS morphology of clinical VT, the right ventricle was mapped. A high-frequency prepotential during sinus rhythm was consistent with a moderator band Purkinje potential and was confirmed with intracardiac echocardiography (Figures 1A and 2A). Pacing from the RV septum on the moderator band resulted in a perfectly matched pace map (12/12) induction of clinical VT (Figure 1B). Activation mapping was performed using a 64-minielectrode



**Figure 1** A: Pre-QRS moderator band (MB) potential noted during sinus rhythm. B: Pace map induction with near-perfect morphologic match of the clinical ventricular tachycardia (VT). C: Activation mapping during VT with -20 ms earliest activation. D: Termination of ventricular tachycardia during the radiofrequency application at 3.5 seconds at the exact site of pace map-matched induction.

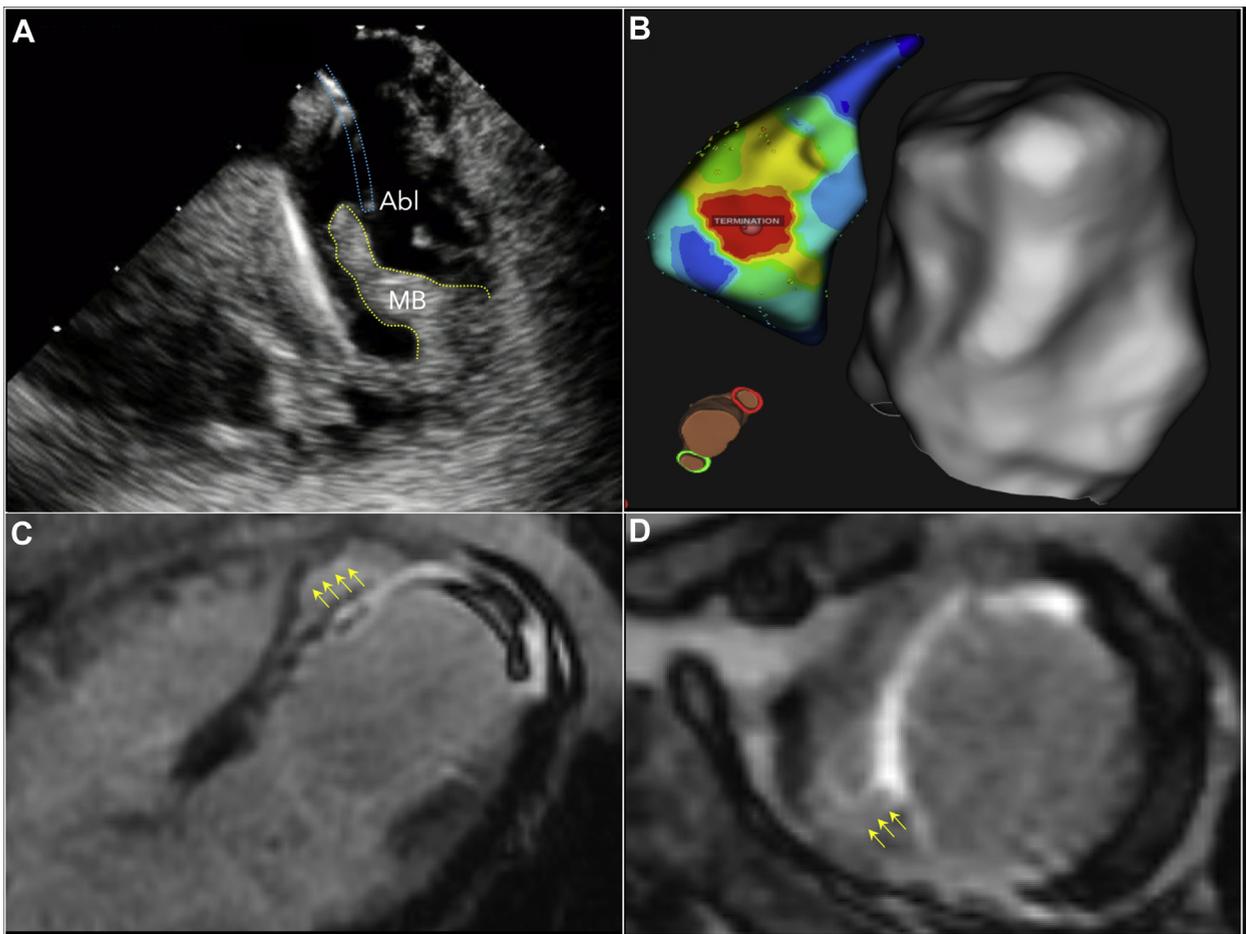
basket catheter (Orion, Boston Scientific, Natick, MA) and the Rhythmia electroanatomic mapping system (Boston Scientific) with earliest activation (-20 ms) at the insertion of the moderator band into the inferior RV free wall (Figures 1C and 2B). Radiofrequency applications were delivered to the moderator band at its RV free wall attachment at 40 watts, resulting in termination of VT in 3.5 seconds (Figure 1D). The patient was rendered noninducible and has remained free of ventricular recurrence at 6-month follow-up off antiarrhythmic medications with normal QRS duration of 104 ms.

## Discussion

We report a case of hemodynamically significant VT that originated from the RV moderator band in a patient with known ischemic cardiomyopathy from prior anteroseptal myocardial infarction. To the best of our knowledge, this is the first report of scar-associated sustained monomorphic VT originating from the moderator band. Prior reports on the arrhythmogenicity of the moderator band have primarily described this structure as a source of PVC triggers for idiopathic ventricular fibrillation and idiopathic sudden death.<sup>1</sup> Sadek and colleagues<sup>2</sup> reported 10 cases of ventricular

arrhythmias arising with moderator band observed in patients with normal ejection fraction without structural heart disease; only 3 of these patients had sustained monomorphic VT and magnetic resonance imaging was normal in these cases. Ventricular arrhythmias originating from the moderator band are relatively uncommon compared to the outflow tract ventricular arrhythmias.<sup>3</sup> Intracavitary structures are increasingly recognized as arrhythmia sources owing to advanced 3-dimensional mapping techniques and with use of intracardiac echocardiography.

The moderator band, also known as trabecula septomarginalis, is a prominent muscular structure that extends from the interventricular septum to the RV anterior wall and helps anchor the anterior leaflet of the tricuspid valve.<sup>4</sup> It carries the right bundle branch and the corresponding Purkinje fibers along with arteries from the left coronary system that contributes to the blood supply of the anterior papillary muscle of the tricuspid valve. Characteristic high-frequency prepotentials indicating Purkinje activation preceding the local ventricular activation are classically observed. Reig and colleagues<sup>5</sup> noted that the blood supply to the moderator band is supplied almost exclusively from the septal perforators, with the majority arising from the midseptal perforators. This finding



**Figure 2** **A:** Intracardiac echocardiography image confirming location of ablation catheter (Abl) positioned on the moderator band (MB). **B:** Location of successful ablation site in the inferior free wall of the right ventricle shown with electroanatomic mapping (Rhythmia, Boston Scientific, Natick, MA). Transmural anteroseptal and apical scar extending into the interventricular septum and the moderator band (yellow arrows) with delayed-enhancement magnetic resonance imaging using wideband sequence in long axis (C) and short axis (D).

was also observed in pediatric angiograms, where in most cases the artery of the moderator band is supplied from the septal perforators of the LAD.<sup>6</sup> While it is possible that the VT from moderator band may be unrelated to the structural heart disease, the mid-LAD occlusion observed in this case is consistent with ischemia or infarct in the moderator band, given the blood supply, which is consistent with mid-septal perforators.

Preprocedural cardiac magnetic resonance imaging performed before the first ablation with wideband sequence, which showed delayed enhancement in the anteroseptal and apical scar with laminated thrombus, was reexamined and showed evidence of infarct extension into the right septum and moderator band (Figure 2C and D). Although entrainment was not performed in this case to confirm reentry as the mechanism, the activation timing is consistent with an exit site from the moderator band from a deeper intraseptal circuit. Alternatively, reentry may be sustained completely within the moderator band owing to compartmentalization between Purkinje and myocardial fibers.<sup>7</sup>

## Conclusion

We report the first case of postinfarction sustained monomorphic VT arising from the moderator band.

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