Right ventricular false tendon–originating premature ventricular complexes triggering ventricular tachycardia: Identification and ablation

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Introduction

Ventricular false tendons (FTs) are fibroid or fibromuscular endocavitary structures with significant variation in origin, communication with the ventricular free wall, papillary muscles, and conduction tissue fibers.[1] Previous studies have demonstrated premature ventricular complexes (PVCs) originating from left ventricular FTs.[2,3] However, no report is available to show right ventricular false tendons (RV-FTs) as a definitive etiologic role for PVCs during catheter ablation. Herein, we present a case of ventricular tachycardia (VT) and ventricular fibrillation (VF) triggered by RV-FT-originated PVCs, which was identified by intracardiac echocardiogram and, in turn, successfully abolished by radiofrequency ablation. We further provide detailed anatomical considerations and the importance of adjusting the intracardiac echocardiography (ICE) view in detecting RV-FTs.

KEY TEACHING POINTS

- Right ventricular false tendons (RV-FTs) have arrhythmogenic properties, which can cause premature ventricular contractions (PVCs) and even PVCs trigging ventricular tachycardia/ventricular fibrillation.
- RV-FTs vary greatly in morphology and anatomical location. Careful observation is needed to avoid omitting this anatomical structure during the procedure.
- The intracardiac echocardiography (ICE) view should be not perpendicular but parallel to the RV-FTs. It is important to maneuver the ICE catheter to a suitable position and angle of view for detecting the real origin of those rare arrhythmias.

Case report

A 79-year-old male patient presented with repeated VT-correlated dizziness for 1 day. He was diagnosed with symptomatic PVCs (14,404 beats/day, 15.6% of all heartbeats) 1 month prior. The VT episodes were triggered by a monomorphic PVC with a short coupling interval of 280 ms, which was left bundle branch block type, left superior axis, and precordial transition in V_6 (Figure 1). The patient denied any previous history of hypertension, coronary heart disease, diabetes, and family history of sudden cardiac death. His blood pressure at admission was 120/70 mm Hg. No obvious heart murmur was heard. The electrocardiogram showed normal atrial-ventricular and intraventricular conduction with regular QT intervals during sinus rhythm. No abnormalities in the blood test results were observed. An echocardiogram revealed normal left ventricular size and function. No anatomical variant was observed in transthoracic echocardiography. Coronary computed tomography angiography examination did not show any abnormal findings.

After inpatient admission, he had several syncopal episodes owing to VF degenerating from the VT and required defibrillation. Antiarrhythmic therapy including amiodarone and β-blocker, as well as general sedation using dexmedetomidine, and magnesium sulfate failed to control the electrical storm. As a result, an emergency ablation was performed. Considering the late ($>V_4$) precordial transition and a rapid downstroke of the initial part of QRS in the precordial leads, an origin of endocavitary structures, such as the moderator band (MB), was suspected. ICE (Biosense Webster, Diamond Bar, CA) was then used to facilitate the mapping in this operation. The ICE catheter was inserted into the right ventricle for real-time MB and papillary muscles (PM) observation. Three-dimensional mapping showed that the earliest activation site was located not at the MB or PM but at the lower part of the RV free wall (Figure 2A). When we maneuvered the ICE catheter to a more desirable position and adjusted it to a parallel view, the RV-FT was visualized

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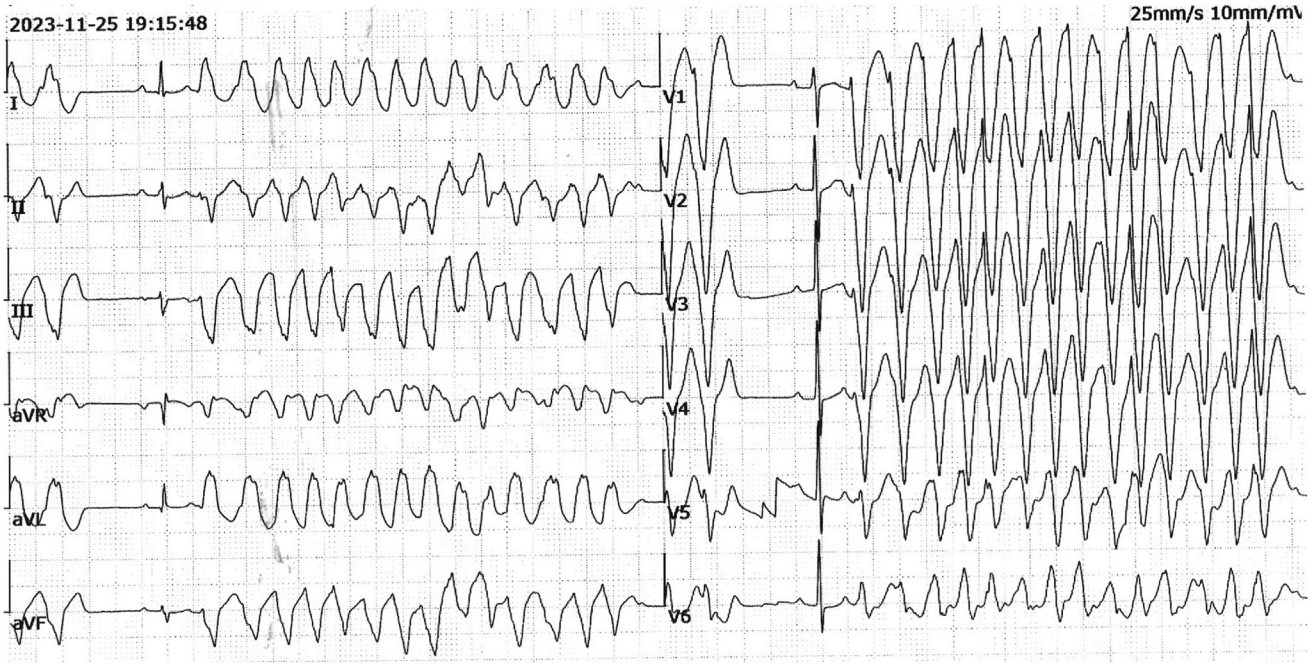

Figure 1 Twelve-lead electrocardiogram of premature ventricular contraction–triggering ventricular tachycardia.

from the lower part of the septal right atrium, with one end connecting the base of the anterior papillary muscle (APM) and the other connecting the free wall (Figure 2B). The target site was located at the attachment of the FT to the RV lateral free wall. The mapping catheter recorded an electrogram showing a near-field pre-systolic potential (red arrow) preceding QRS onset by 30 milliseconds during PVC with no obvious Purkinje potential during sinus rhythm (Figure 2C). Ablation at this site successfully abolished the PVCs and VT. The anatomic relationship between the tip of the ablation catheter and the FT as well as the APM and MB can be clearly identified from the Supplemental Video. The ablation site was located at the attachment of the FT to the RV free wall, while the other end of the FT was connected to the root of the APM. MB could also be observed in this view connecting to the APM from the septal side. Voltage mapping of the right ventricle was performed using the ablation catheter during sinus rhythm. No low-voltage area or fragmented potential was revealed.

Electrocardiographic monitoring showed no PVCs or VT recurrence before discharge. Given that there was no evidence of structural heart disease and the patient refused to receive implantable cardioverter-defibrillator therapy, he did not receive implantable cardioverter-defibrillator implantation. Considering that the average heart rate was 65 beats per minute, the patient was discharged free of any antiarrhythmic therapy and was asked to keep in close touch with cardiologists. To date, there is no evidence of PVCs or VT recurrence through 24-hour Holter and outpatient follow-up.

Discussion

RV-FTs have been systematically described and classified into 5 types by Loukas and colleagues.[4] Referring to their study, Type I (21, 47.7%) of RV-FTs were located between the ventricular septum and the APM; Type II (11, 22.9%) was a connection between ventricular septum and the posterior PM; in Type III (7, 14.5%) the RV-FTs were connected between the anterior leaflet of the tricuspid valve and the RV free wall; in Type IV (5, 10.4%), between the posterior PM and the ventricular free wall; and lastly, in Type V (4, 8.3%), between the APM and ventricular free wall. In our case, the ICE view clearly showed that the tip of the ablation catheter at the target was put on a thin structure connecting the RV anterior PM and the free wall. Meanwhile, the ICE also clearly showed the MB connecting to the PM from the septal side. As such, the structure of RV-FT was unquestionably confirmed by ICE, and could be categorized as Type V.

Our case is unique, as it represents the first delineation of the arrhythmogenicity of RV-FT. PVCs originating from the left ventricular FT and successful radiofrequency ablation using an ICE-guided electroanatomical approach have been described in previous studies, with the number of reported cases significantly lower than that of papillary muscle or even MB-originating PVCs. Nevertheless, there were even fewer studies on the RV-FT than on the left ventricular FT. According to previous anatomical descriptions, RV-FTs greatly vary in morphology and relative positions.[4] Therefore, careful observation is needed to avoid omitting this anatomical structure during the procedure. In our case, at first, the ICE view was perpendicular to the RV free wall, showing the catheter tip located at one of the ventricular trabeculae (Figure 3A1–3A4). Considering that anatomic substrates are usually responsible for idiopathic ventricular arrhythmias in the absence of identifiable structural heart disease, the catheter was pushed forward and rotated clockwise, and both ends of the RV-FT and the base of the APM were

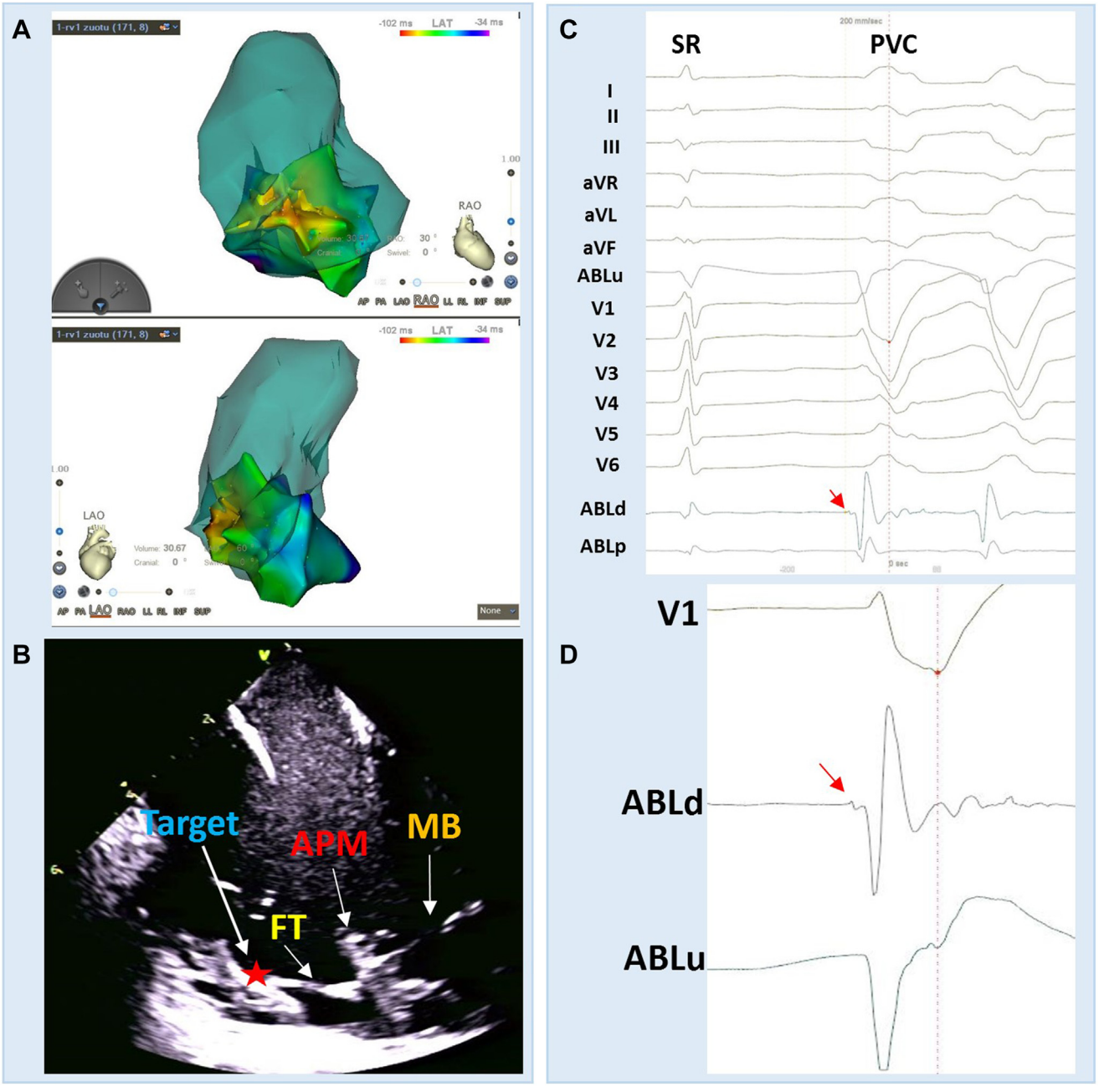

Figure 2 Three-dimensional mapping, intracardiac echocardiography view, and local electrogram characteristics at the successful ablation site. **A:** The earliest activation site was located at the lower part of the right ventricular (RV) free wall. **B:** The RV false tendons (FT) connected one end to the base of the anterior papillary muscle (APM) and the other end to the free wall. The target site was located at the attachment of the FT to the RV lateral free wall (*red star*). **C:** A near-field presystolic potential (*red arrow*) preceding QRS onset by 30 ms during premature ventricular contraction (PVC) was recorded at the target. **D:** Magnification of unipolar and bipolar electrogram. MB = moderator band; SR = sinus rhythm.

observed. When the ICE catheter was positioned at the lower septal part of the right atrium, the view clearly showed the RV-FT, APM, and the ablation catheter tip on the end adjacent to the free wall (Figure 3B1–3B4). RV-FTs can also be visualized by a long-axis view from the right ventricle's upper part (Figure 3C1–3C4). In this sense, the anatomical localization of RV-FT is straightforward to be omitted, especially when the ICE view is perpendicular to RV-FT, by which the RV-FT is viewed from a cross-section perspective (Figure 3A1–3A4). We speculate that the apparent rareness of RV-FT-originated arrhythmia may partly be attributable to the missed diagnosis in clinical practice.

Though FTs are generally considered to be benign, epidemiological studies have demonstrated a significant correlation between FTs and PVCs.[5] Some clinical observations have directly proved the clear relationship between PVCs or idiopathic VT and the left ventricular FTs by combining the finding of intracardiac electrophysiological study and ICE mapping during radiofrequency ablation.[2,6] It has been reported that FTs contain fibrous tissue, myocardial fibers,

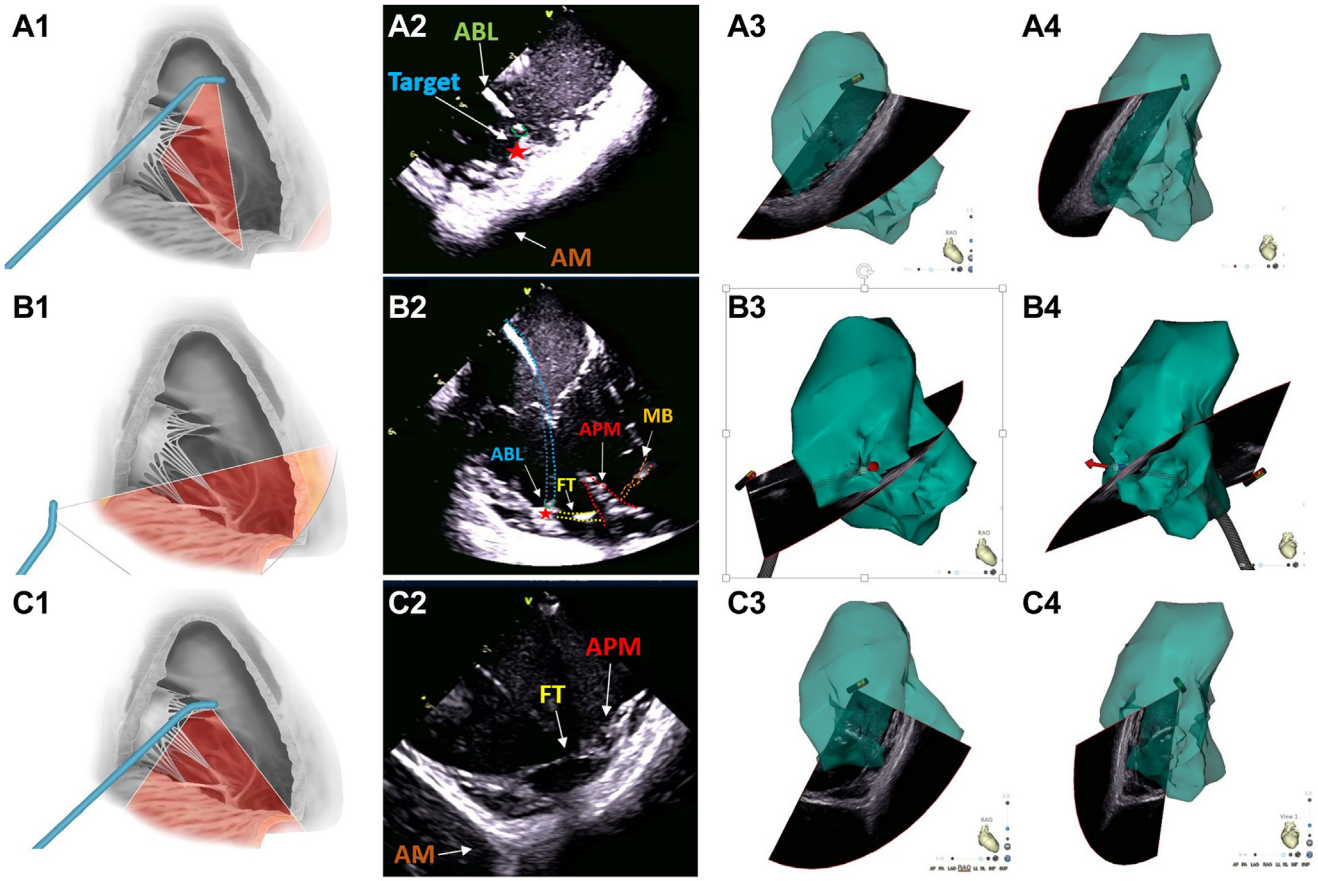

Figure 3 Images demonstrating the relationship between the intracardiac echocardiography view and the anatomy of a right ventricular false tendon (FT). ABL = ablation catheter; AM = acute margin of the heart; APM = anterior papillary muscle; MB = moderator band.

Purkinje fibers, and blood vessels.[7] Increased automaticity of Purkinje cells within muscle fibers and mechanical traction at the FT attachment site were speculated to be accounted for in the mechanisms of PVCs arising from FTs.

PVCs from the ventricular endocavitary structure triggering VT/VF have been reported as an infrequent clinical presentation. Our case expands the clinical scope regarding the originating structure of the arrhythmogenic substrate. In the future, careful prospective observation for RV arrhythmias using ICE from multiple views is needed to clarify the real prevalence, as well as the electrocardiogram and electrophysiological characteristics of the RV-FT arrhythmia.

Conclusion

The present study represents the first delineation of the arrhythmogenicity of RV-FT, which exhibits a short-coupled PVC trigging VT/VF. The case also implicates the importance of maneuvering the ICE catheter to a suitable position and angle of view for detecting the real origin of those rare arrhythmias, which may have been readily omitted in past practice.

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Appendix

Supplementary Data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrcr.2024.03.011.

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