Successful ablation of a narrow complex tachycardia arising from a left ventricular false tendon: Mapping and optimizing energy delivery

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Overview

Ventricular tachycardia (VT) arising in the His-Purkinje system is an important cause of narrow complex tachycardia that is often amenable to catheter ablation. Ventricular arrhythmias may require targeted mapping and ablation of endocavitary structures, such as papillary muscles or false tendons to eliminate symptomatic arrhythmia. We describe a patient with narrow QRS (NQRS) tachycardia who required mapping and ablation of a left ventricular false tendon (LVFT). Intracardiac echocardiography (ICE) was used to identify the target and optimize contact to successfully ablate this unique focus for arrhythmia.

Introduction

Radiofrequency ablation of NQRS tachycardia is a highly effective treatment option for symptomatic patients.[1] Although supraventricular tachycardia (SVT) is the most common form of NQRS tachycardia, VT arising from the fascicles should also be considered. Ablation of arrhythmia focus in endocavitary structures, such as papillary muscles, false tendons, and the moderator band, has been described for the treatment of VT.[2–5] While left fascicular tachycardia is well known to produce typical right bundle branch block morphology tachycardia and thus mimic SVT with aberrancy,[6] NQRS tachycardia requiring ablation of an endocavitary structure has not been reported. We describe a patient with narrow complex tachycardia and ectopy where successful ablation was performed on an LVFT. Correct interpretation of the His bundle electrogram, assessment of multiple sites of early activation in the left ventricle, and the use of ICE to identify and guide ablation on the false tendon is described.

KEYWORDS Narrow complex tachycardia; Fascicular tachycardia; Ventricular tachycardia; False tendon; Intracardiac echocardiography

ABBREVIATIONS **ICE** = intracardiac echocardiography; **LV** = left ventricular; **LVFT** = left ventricular false tendon; **NQRS** = narrow QRS; **SVT** = supraventricular tachycardia; **VT** = ventricular tachycardia (Heart Rhythm 2014;11:321–324)

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Case report

A 52-year-old woman with a 25-year history of paroxysmal tachypalpitations presented with increasing frequency of lifestyle limiting symptoms despite β-blocker therapy. Holter monitoring documented narrow complex tachycardia coincident with her symptoms (Figure 1). The QRS to P-wave interval was short, and tachycardia initiation was either spontaneous or associated with ectopic beats. Single ectopic beats with identical QRS morphology occurred 3000 times over a 24-hour period. Transthoracic echocardiography was normal with preserved ventricular function (ejection fraction 65%) and no evidence of valvular disease. She was brought to the electrophysiology laboratory for catheter ablation.

Electrophysiological study

Mapping catheters were inserted via right internal jugular, right femoral, and left femoral venous access and positioned in the coronary sinus, high right atrium, His bundle region, and right ventricle. A 7-F, 3.5-mm tip, open-irrigated catheter (Biosense Webster, Johnson & Johnson, Diamond Bar, CA) was used for mapping and ablation. Tachycardia and frequent ectopy identical to the clinically documented morphology were induced (Figure 1). The tachycardia had a QRS duration of 90 ms and a V-A interval of 70 ms, and the earliest retrograde atrial activation was in the vicinity of the fast pathway. The differential diagnoses included SVT with aberrancy and VT arising from the His-Purkinje system. The V-A interval was seen to vary without change in the His-His activation time and tachycardia cycle length, making orthodromic reciprocating tachycardia using an accessory pathway unlikely. The intra-Hisian electrogram activation sequence during ectopy was distinct from that noted during sinus rhythm. Furthermore, the H-V interval was shorter

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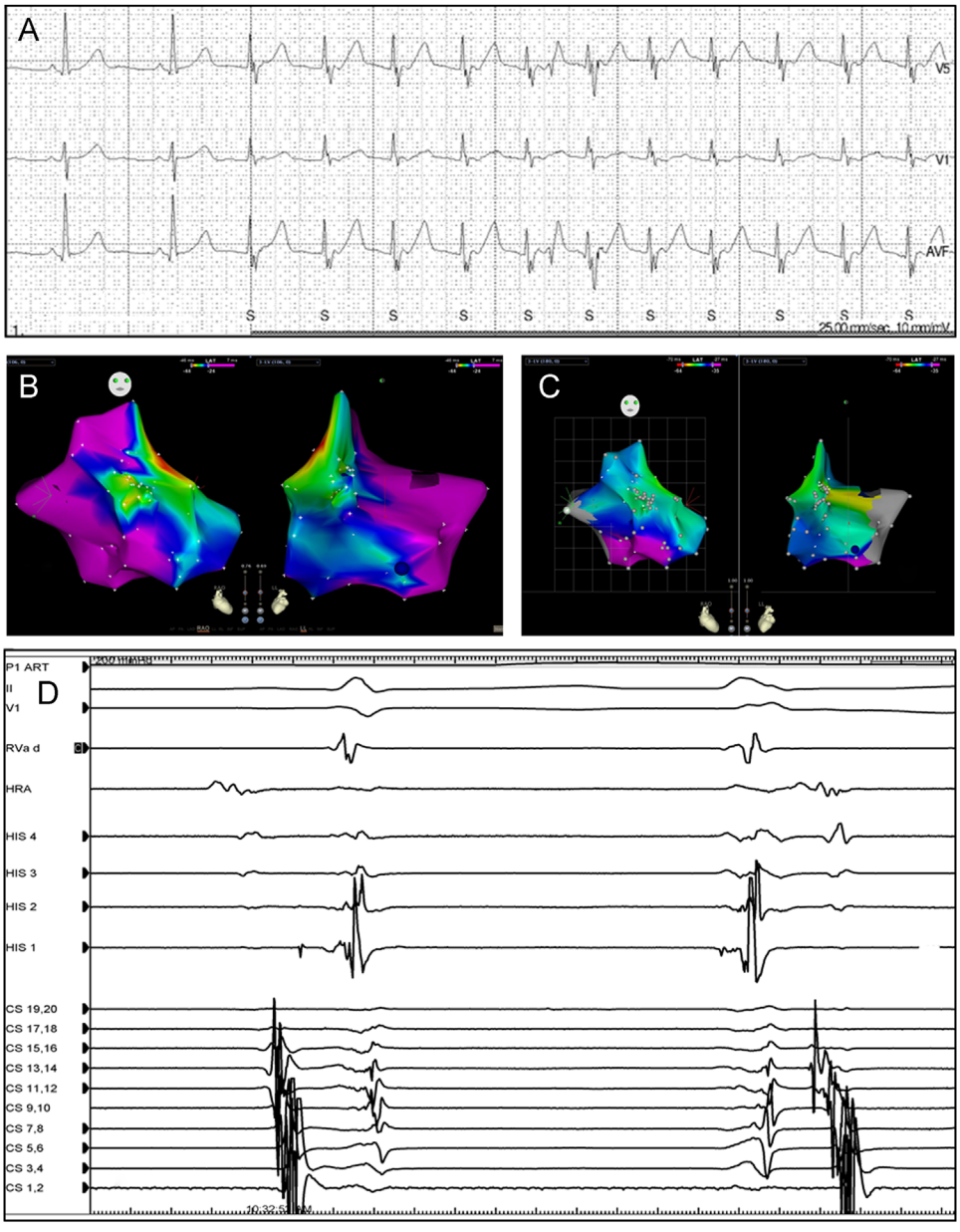

Figure 1 **A:** Holter recording during palpitations showing narrow complex tachycardia with short RP interval (70 ms). **B:** Electroanatomic map of left ventricular (LV) myocardial activation during the ectopic beat. The earliest activation was at the septum with unexpectedly early activation of the lateral wall and anterolateral papillary muscle. Blue dots indicate location of the false tendon identified using intracardiac echocardiography. Note that this is a partial map of the LV. **C:** Electroanatomic map of fascicular potential preceding the local ventricular activation in the LV superimposed on the myocardial activation map. The site of the earliest fascicular activation was near the septal insertion of the false tendon. **D:** Intracardiac electrograms during sinus rhythm and atypical premature ventricular contraction originating from the false tendon. The His-ventricle interval was shorter during ectopy (0 ms) than during sinus rhythm (35 ms).

during tachycardia or ectopy (0 ms) than during sinus rhythm (35 ms) (Figure 1). These findings suggested a possible arrhythmia origin inferior to the His bundle (VT, fascicular tachycardia). Therefore, further mapping was done in the right and left ventricles (using a retrograde transaortic approach). There was no evidence of dual atrioventricular nodal physiology or atrioventricular accessory pathway.

Electroanatomic mapping

A detailed 3-dimensional electroanatomic map of the right and left ventricular (LV) endocardial activation during clinical ectopy was carried out (Carto3, Biosense Webster, Johnson & Johnson, Diamond Bar, CA). When the local myocardial activation was mapped, multiple sites of early activation were noted on the LV septum. Moreover, activation of the LV lateral wall was earlier than expected (Figure 1). Several LV sites also showed a prepotential consistent with fascicular signals, all preceding the surface QRS by 20–28 ms. The activation of the right bundle branch and moderator band was significantly later than the LV fascicular sites. The His bundle activation was approximately 10 ms after the proximal left bundle activation, which