Changes in the Reentrant Pathway in Verapamil-Sensitive Fascicular Reentrant Ventricular Tachycardia During Ablation

Patrick Tchou, MD*, Khaldoun Tarakji, MD, MPH, Mohamed Kanj, MD

KEYWORDS

• Verapamil • Reentrant ventricular tachycardia • Ablation • Case study
• Fascicular activation patterns

KEY POINTS

- This case demonstrates that fascicular reentrant tachycardias can generate different QRS morphologies depending on the path of breakout into the myocardium.
- Ablation of 1 exit path may results in propagation over other fascicular paths.
- Changes in fascicular activation patterns causing changes in cycle lengths demonstrates the reentrant nature of this tachycardia.

CLINICAL HISTORY

A 58-year-old woman with a previously noted structurally normal heart presents to the emergency department with a sustained tachycardia. Fig. 1 shows the 12-lead electrocardiogram (ECG) of the tachycardia. Intravenous adenosine, lidocaine, and amiodarone infused in the emergency department failed to affect the tachycardia. Cardioversion of the tachycardia resulted in sinus rhythm for a few beats followed by recurrence of the tachycardia. The patient was then admitted to the intensive care unit. The ECG findings were noted to be typical of a verapamil-sensitive ventricular tachycardia (VT). Transient termination of the tachycardia could be achieved with boluses of verapamil. However, even at maximally tolerated infusions of verapamil, the tachycardia would not terminate. The patient was thus taken to the electrophysiology laboratory for mapping and ablation.

ELECTROPHYSIOLOGY STUDIES AND ABLATIONS

The tachycardia was incessant and can only be slowed with large doses of verapamil, which also caused it to become irregular in rate. Mapping of the tachycardia identified early activation near the inferior septal portion of the left ventricle where a fascicular potential preceded ventricular activation (Fig. 2). Ablation at this site terminated the tachycardia. A 12-lead of sinus rhythm after ablation is shown in Fig. 3.

In sinus rhythm, ventricular function was noted to be markedly depressed. This was attributed to the prolonged and incessant nature of the presenting tachycardia. Verapamil was discontinued and the patient was observed clinically. Within 24 hours, the patient had recurrence of a similar tachycardia. However, now the tachycardia had an inferior axis. The patient was then again taken urgently to the electrophysiology laboratory for

Department of Cardiovascular Medicine, Cleveland Clinic Main Campus, Mail Code J2-2, 9500 Euclid Avenue, Cleveland, OH 44195, USA
* Corresponding author.
E-mail address: TCHOUP@ccf.org

Card Electrophysiol Clin 8 (2016) 773–777
http://dx.doi.org/10.1016/j.ccep.2016.08.001

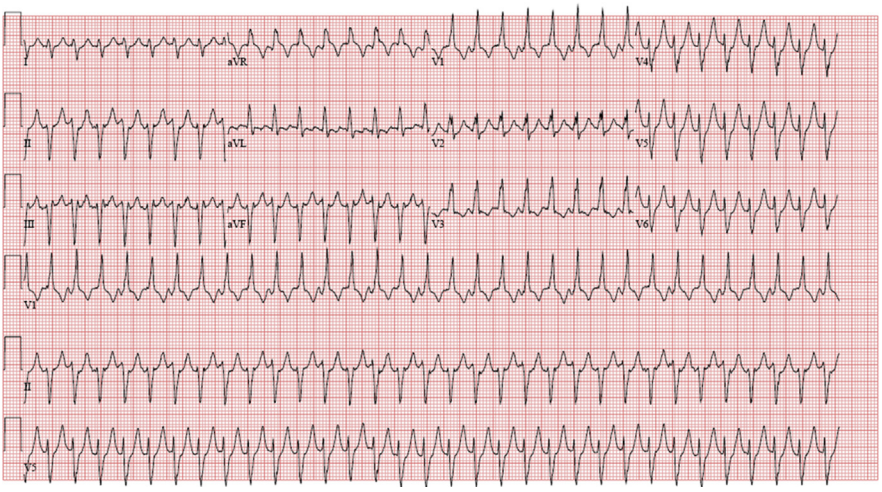

Fig. 1. The presenting 12-lead electrocardiogram with a 3-lead rhythm strip. This shows a tachycardia having a cycle length of 310 ms. The QRS has a right bundle branch block morphology with a superior axis typical of a verapamil sensitive ventricular tachycardia. Ventriculoatrial dissociation is noted in lead V1.

another mapping and ablation procedure. A 12-lead ECG comparison of the baseline tachycardia with the recurrent one is shown in **Fig. 4**. Although the QRS of the recurrent VT maintained a right bundle branch block pattern in V1, its duration was distinctly shorter and its axis had shifted inferiorly.

Mapping in the left ventricle now showed early activation in the anterior wall and an early fascicular potential was identified in the anterior septal region, which activated 45 ms before earliest ventricular activation. The effects of ablation at this site is shown in **Fig. 5**. During application of radiofrequency energy, there was a transition of the QRS morphology over 3 beats from the right bundle branch block QRS to a left bundle branch block pattern having a much longer QRS duration. The cycle length of the tachycardia slowed by 20 ms with this transition and there was prolongation of the fascicular potential to local ventricular activation interval noted on the ablation tip electrode.

Further mapping more inferiorly along the proximal septum identified earlier fascicular potentials and ablation at this site terminated the tachycardia. No further recurrence of the tachycardia occurred. Her 12-lead ECG then showed a conduction defect with a QRS duration of 114 ms, **Fig. 6**. The HV interval was 50 ms in sinus rhythm.

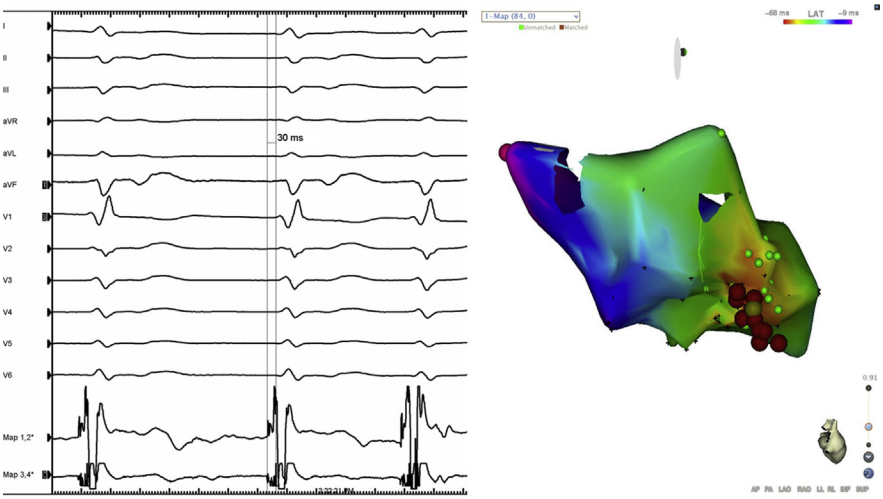

Fig. 2. (*Left*) Intracardiac electrocardiograms at the site of earliest ventricular activation in tachycardia where radiofrequency energy delivery terminated the tachycardia. (*Right*) Right lateral view of CARTO map indicating site of ablation lesions delivery.