Anatomical Ablation Strategy for Noninducible Fascicular Tachycardia

Ahmed Karim Talib, MD, PhD, Akihiko Nogami, MD, PhD*

KEYWORDS

• Fascicular tachycardia • Verapamil-sensitive ventricular tachycardia • Catheter ablation
• Anatomical approach

KEY POINTS

- The presence of structural heart disease does not exclude fascicular ventricular tachycardia (VT), especially if the VT is verapamil sensitive.
- An empirical anatomical approach is effective when fascicular VT is noninducible or if diastolic Purkinje potential (P1) cannot be recorded during VT mapping.
- Pace mapping at the successful ablation site is usually not effective because selective pacing of P1 is difficult and there is an antidromic activation of the proximal P1 potential.

CLINICAL PRESENTATION

A 22-year-old man presented to the emergency department with palpitation associated with wide QRS complex tachycardia (212 beats per minute), which was slowed and terminated by intravenous Verapamil (5 mg) injection (**Fig. 1**). The patient was kept on oral verapamil; however, he was still symptomatic and was referred to the authors' hospital for catheter ablation. The patient has a known history of Kawasaki disease and high-grade stenosis in the proximal segment of the left anterior descending artery associated with rich collaterals from both the left circumflex and right coronary arteries. During admission, coronary angiogram revealed no significant changes (ie, left anterior descending artery stenosis with rich collaterals) and the left ventriculography revealed normal ventricular wall motion.

Question 1: What Is the Most Likely Type of This Tachycardia?

Verapamil-sensitive fascicular tachycardia is characterized by right bundle branch block (RBBB) configuration and left axis deviation.[1] It is typically idiopathic but can occur in patients with organic heart disease.[2] Although the patient had coronary lesion, left ventricular function and wall motion were normal. Moreover, 3-dimensional electroanatomical mapping revealed normal endocardial voltage.

VENTRICULAR TACHYCARDIA INDUCTION

Burst and programmed atrial and ventricular stimulation failed to induce clinical ventricular tachycardia (VT). Intravenous atropine administration followed by programmed atrial pacing resulted in wide QRS complexes (**Fig. 2**).

Question 2: What Are These Wide QRS Complexes and What Is the Role of Atropine?

During atrial pacing, His bundle was activated and His potential (H) was recorded in the distal His bipole. However, the wide QRS complexes were not preceded by His bundle activation; hence, aberrant conduction was ruled out. The wide QRS

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Cardiovascular Division, Faculty of Medicine, University of Tsukuba, Tsukuba, Japan
* Corresponding author. Cardiovascular Division, Faculty of Medicine, University of Tsukuba, 1-1-1 Tennodai, Tsukuba, Ibaraki 305-8575, Japan.
E-mail address: akihiko-ind@umin.ac.jp

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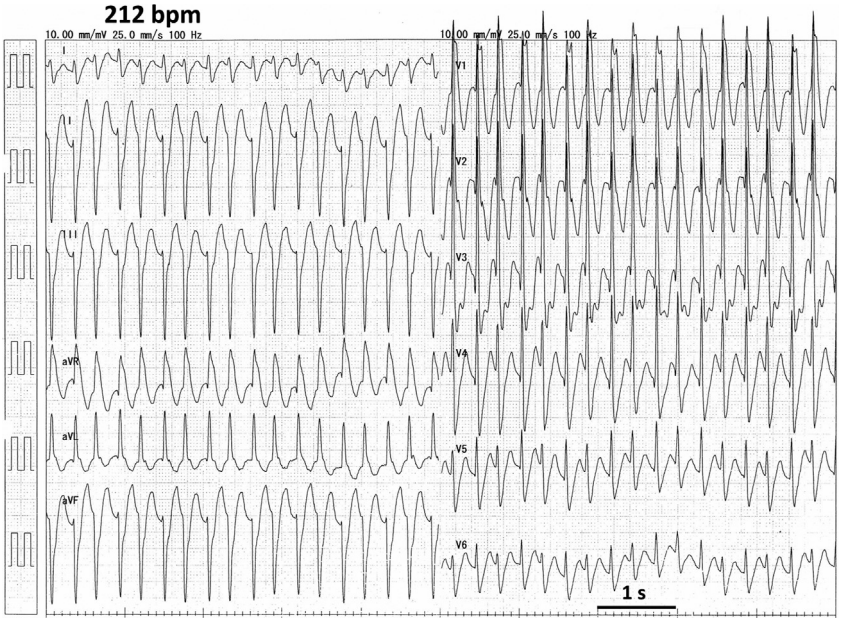

Fig. 1. Twelve-lead electrocardiogram (ECG) of clinically documented tachycardia. ECG exhibited right bundle branch block configuration and left axis deviation. QRS alternans was observed during ventricular tachycardia (VT). The VT was terminated by intravenous verapamil administration. bpm, beats per minute.

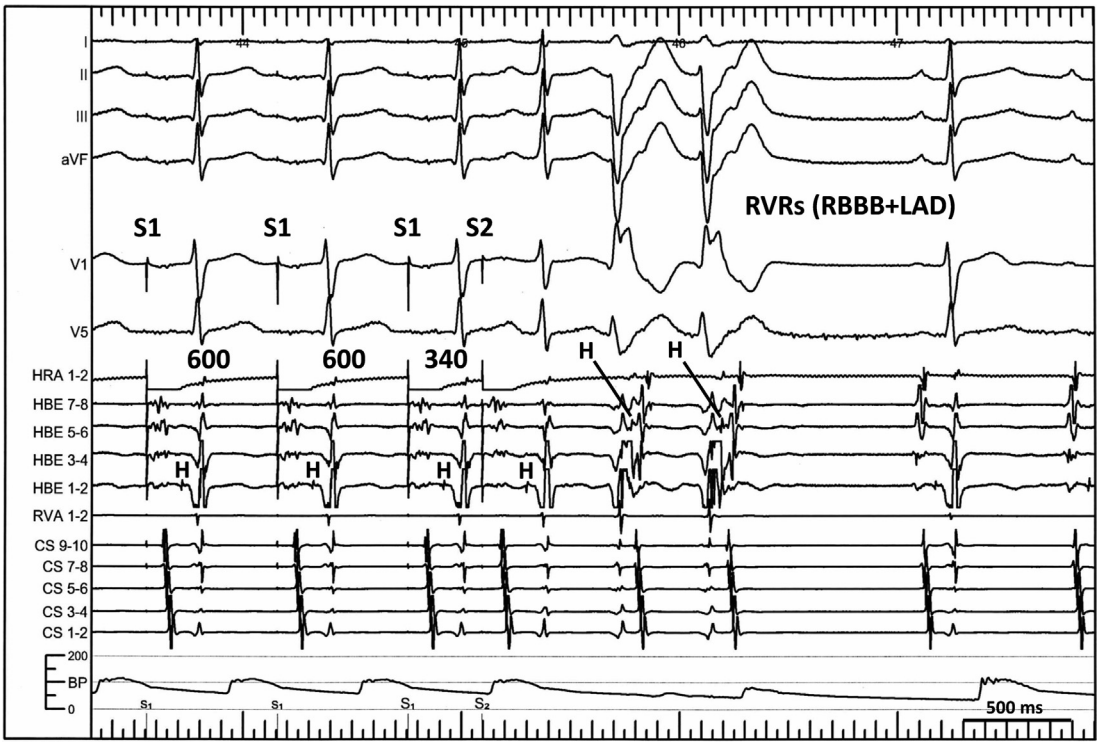

Fig. 2. Programmed atrial stimulation after intravenous atropine injection. Programmed atrial stimulation after intravenous atropine injection induced wide QRS complexes with RBBB configuration and left axis deviation, which is similar to the clinical VT. Negative His potential (H)-V interval was observed during these complexes. H-V interval during fascicular VT depends on the site of the upper turnaround. If the site of turnaround is high (or close to His bundle), H-V interval will be positive (but shorter than that during sinus rhythm) and vice versa (ie, if the lower turnaround site is more distal, the H-V interval will be negative). 1 to 2, distal bipole; 9 to 10, proximal bipole; CS, coronary sinus; HBE, His-bundle electrogram; HRA, high right atrium; LAD, left axis deviation; RBBB, right bundle branch block; RVA, right ventricular apex.