Successful mapping and ablation of a pediatric-onset non-reentrant fascicular tachycardia

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July 20, 2022

Abstract

Non-reentrant fascicular tachycardia (NRFT) developed in a 6-year-old Japanese boy. Because of the drug-resistant recurrences, he received catheter mapping and ablation at age 10 years. An electrocardiogram exhibited a superior left axis deviation, a right bundle branch block-type configuration, and relatively narrow QRS with sharp R wave. It suggested verapamil-sensitive ventricular tachycardia (VT), but showed no sensitivity to verapamil or reentrant characteristics in the electrophysiological study. Detailed VT mapping determined the earliest presystolic Purkinje potential on the left posterior fascicle at the mid-ventricular septum. Radiofrequency current applications to the lesion led to his NRFT-free life without restriction for 16 months.

Introduction

The majority of idiopathic Purkinje-related ventricular tachycardias (VTs) are caused by reentrant mechanisms, and non-reentrant fascicular tachycardia (NRFT) is a rare form of idiopathic VTs in adults.[1-3)] It is crucial to distinguish NRFT from other idiopathic reentrant VTs for the accurate diagnosis as well as the effective treatment. However, there is no information about the electrophysiological studies and the treatment effect in the pediatric-onset NRFT. We herein report a pediatric case of refractory NRFT that was successfully treated by the catheter mapping and ablation.

Case

A 10-year-old Japanese boy, weighting 57 kg, was referred to our hospital because of syncope or dizziness during exertion. The symptoms started at the age of 6 years and the frequency came to increase after age 9, although he refrained from hard exertion. The 12-lead electrocardiogram (ECG) during sinus rhythm and the echocardiogram did not show any abnormalities (**Figure 1A**). VT was first recorded at the age of 10 (**Figure 1B**). An ECG exhibited a relatively narrow QRS regular tachycardia with a heart rate of 250 bpm, a superior left axis deviation, and a right bundle branch block-type configuration. The tachycardia was not suppressed after the administration of adenosine triphosphate 10 mg, or lidocaine 50 mg. After a slowly injection of verapamil 5 mg, the tachycardia was slightly decelerated on the sustained rate alone. The treatment response represented a low sensitivity to verapamil. Because tachycardia was not controlled by oral administration of propranolol 30 mg/day, radiofrequency (RF) catheter ablation was performed under the guidance of an electroanatomical mapping system (CARTO3, Biosense-Webster, Diamond Bar, CA).

Multipolar electrode catheters were inserted via the femoral vein and were placed in the coronary sinus, at the His bundle region, and at the right ventricular apex. In addition, a linear decapolar catheter (DECANAV, Biosense-Webster, Diamond Bar, CA) was inserted to the left ventricle via retrograde aortic approach. We first mapped the Purkinje potentials along the left anterior and posterior fascicle during sinus rhythm, and tagged the sites with white circles (**Figure 2A**). Then, we performed burst pacing repeatedly from several

sites of the ventricles, and the VT was induced intermittently by burst pacing from the inferoseptum of the left ventricle only after the administration of high dose of isoproterenol (2 μg). Although a presystolic Purkinje potential preceding the QRS onset by 27 ms was recorded during the VT on the left posterior fascicle at the mid-ventricular septum by mapping with the linear decapolar catheter, any diastolic potentials, a common ablation target of reentrant fascicular ventricular tachycardia, could not be recorded. We performed constant pacing from several sites of the right and left ventricles with variable cycle lengths, but did not identify any constant QRS fusion, indicating that the mechanism of VT was reentry (**Figure 3**). Based on the findings, the VT was determined to occur as NRFT. We inserted a multi-spline duodecapolar catheter (PENTARAY, Biosense-Webster, Diamond Bar, CA) via a retrograde aortic to map Purkinje potentials systematically along the fascicles, and tagged the sites with blue circles. The earliest presystolic Purkinje potential, preceding the QRS onset by 34 ms, was recorded on the left posterior fascicle in the vicinity of the earliest site with the linear catheter (**Figure 2**). We placed an irrigated-tip ablation catheter (SmartTouch, Biosense-Webster, Diamond Bar, CA) at the site of the earliest presystolic Purkinje potential with the duodecapolar catheter via a transseptal approach to obtain better catheter contact, and completed RF current applications (30-50 W) at around the site (**Figure 2A**). After ablation, the NRFT came to be non-inducible under high-dose isoproterenol and ventricular burst pacing. Thereafter, tachyarrhythmia has not recurred for the following 16 months without antiarrhythmic drugs and exercise restriction.

Discussion

This is the first pediatric report of intractable NRFT that was successfully controlled by catheter mapping and ablation. NRFT reportedly occurs only in 2.8% of idiopathic VT cases in adults.[3)] The mechanism of NRFT was not precisely determined, but was assumed to originate from abnormal automaticity from Purkinje fibers. NRFT is defined in adult patients by the following criteria[3,4)]: (1) Normal ECG and intracardiac conduction interval, (2) QRS morphology with a relatively narrow right bundle branch block waveform, (3) Inducibility by intravenous isoproterenol infusion and burst ventricular stimulation, (4) The absence of criteria of reentrant fascicular VT, such as transient entrainment and verapamil sensitivity, and (5) Successful ablation to the site of the earliest presystolic Purkinje potential. These findings are essential to differentiate NRFT from the reentrant fascicular VT. Because the present case fulfilled all of these criteria, the tachyarrhythmia was finally determined to occur as NRFT.

NRFT is challenging to diagnose as well as to control even after ablation therapy. The long-term success rate of the first ablation therapy was reportedly 74% (11 out of 15) with high recurrence rate[3)] The reasons for recurrences are thought as follows: First, it is hard to induce NRFT reproducibly and stably and to map the accurate focus of VT. Second, pacemap-guided approach does not always provide the site of the origin of NRFT. Even when identical QRS morphology is obtained by pacemapping at the exit site to myocardium, the VT origin from Purkinje fiber may be escaped from the RF current applied.[2)] Third, it is not easy to contact the ablation catheter with the site of focal VT origin sufficiently and stably because of the network structure of the Purkinje fibers. In the present case, we performed burst pacing repeatedly from several sites of the ventricles under high doses of isoproterenol to induce NRFT. After the careful diagnostic pacing, we drew VT map using the multi-electrode catheters and identified to record the earliest preceding Purkinje potential. We also performed a transseptal approach before RF application to obtain a better catheter contact with the focal VT origin site and to avoid any injury to the proximal part of the fascicle with the ablation catheter. Thus, we were able to diagnose the VT as NRFT and to provide an effective and safe catheter ablation therapy. Further studies and long-term follow up are needed for the pediatric-onset NRFT.

Conclusion

We successfully diagnosed and ablated NRFT originating from the left posterior fascicle in a child. NRFT is a controllable VT not to be over\souttlooked when a 12-lead electrocardiogram shows relatively narrow QRS tachycardia with a right bundle branch block-type configuration even in the pediatric setting.

Author contribution: Y.N. and S.T. drafted the main manuscript text and prepared figures. K.S., H.T. and S.O. gave conceptual advice and supervised the study process. K.Y. and H.N. critically reviewed the

manuscript. All authors reviewed the manuscript and approved the final manuscript.

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Figure Legends

Figure 1 : A . A 12-lead electrocardiogram (ECG) during sinus rhythm. **B** . A 12-lead ECG during tachycardia with a cycle length of 240 ms. The ECG shows a relatively narrow QRS with sharp R wave, a superior left axis deviation and a right bundle branch block-type configuration. VA dissociation is also identified.

Figure 2: A . Right anterior oblique (RAO) and superior (SUP) view of a ventricular muscle activation map during the non-reentrant fascicular tachycardia (NRFT) , showing centrifugal activation from the earliest site at the ventricular septum (highlighted in red). The white circles indicate the sites where Purkinje potentials are recorded during sinus rhythm along the left anterior and posterior fascicles. The blue circles indicate the sites where preceding Purkinje potentials are recorded during the NRFT. The larger blue circle indicates the site of the earliest Purkinje potential recorded with the multi-spline duodecapolar catheter. The location of the breakout site to the ventricular muscle is the apex side of the earliest Purkinje potential site. We delivered radiofrequency current to the sites with red circle.**B** . Intracardiac electrogram during the NRFT mapping. The earliest Purkinje potential, preceding the QRS onset by 34 ms, is recorded by the electrodes 15-16 (blue asterisk) of the multi-spline duodecapolar catheter. HBE; his bundle electrogram, CS; coronary sinus, PEN; PENTARAY catheter.

Figure 3: Twelve-lead electrograms **A** . during ventricular tachycardia (VT), and **B** . during pacing from right ventricular septum and **C** . during pacing from left ventricular inferoseptum. We performed constant pacing from several sites of the ventricles with variable cycle lengths during the VT, however, only pacing waveforms were obtained and constant QRS fusion, indicating that the mechanism of the VT was reentry, could not be confirmed.