



Case Report

His-Purkinje system-related incessant ventricular tachycardia arising from the left coronary cusp



Eiji Sato, MD^{a,b}, Tetsuo Yagi, MD, PhD^{a,*}, Akio Namekawa, MD^a, Akihiko Ishida, MD^a, Yoshiaki Mibiki, MD, PhD^a, Yoshihiro Yamashina, MD, PhD^a, Hirokazu Sato, MD, PhD^a, Takashi Nakagawa, MD^a, Manjirou Sakuramoto, MD^a, Jyuri Komatsu, MD^a, Tomoyuki Yambe, MD, PhD^b

^a Division of Cardiology, Sendai City Hospital, Wakabayashi-ku, Shimizukouji3-1, Sendai, Miyagi 984-8501, Japan

^b Department of Medical Engineering and Cardiology, Institute of Development, Aging and Cancer, Tohoku University, Sendai, Japan

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ABSTRACT

We describe the case of a 23-year-old woman who had His-Purkinje system-related incessant ventricular tachycardia with a narrow QRS configuration. The ventricular tachycardia was ablated successfully in the left coronary cusp where the earliest endocardial activation had been recorded. We hypothesize that a remnant of the subaortic conducting tissue was the source of the ventricular arrhythmias.

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1. Introduction

Idiopathic ventricular tachycardia (VT) can occur in patients with structurally normal hearts. Idiopathic VT is classified into several subtypes according to its mechanism, QRS morphology, and site of origin [1]. The His-Purkinje system (HPS) has been found to be responsible for some forms of idiopathic VT. One form, verapamil-sensitive idiopathic left VT, arises close to or in the left posterior fascicle. It is the most common form of HPS-related VT and is considered a re-entrant mechanism. Another rare form of HPS-related VT is focal Purkinje VT, which is due to abnormal automaticity or triggered activity in the ventricular conducting system [2]. Focal Purkinje VT arises from both the left bundle branch (LBB) and right bundle branch (RBB) and may be abolished by radiofrequency (RF) catheter ablation at the site of the earliest Purkinje potential during VT [3,4]. In the present report, we describe a case of HPS-related VT, with a narrow QRS configuration, that was successfully ablated in the left coronary cusp (LCC).

2. Case report

A 23-year-old woman, with a 5-year history of repetitive palpitation and an otherwise unremarkable medical history, was admitted to our hospital. Clinical examination and echocardiography findings ruled out structural heart disease. A 12-lead electrocardiography (ECG) during tachycardia revealed frequent and incessant narrow QRS tachycardia, with only short-duration, intervening episodes of junctional rhythm (Fig. 1A). Ambulatory monitoring of the patient showed frequent episodes of narrow QRS tachycardia, with junctional rhythm accounting for approximately 65% of the total heartbeats per day.

After written informed consent was obtained, an electrophysiological study was performed. The patient's basic rhythm was junctional rhythm. Repetitive, short-duration, narrow QRS tachycardia appeared during junctional rhythm. The R–R interval of the tachycardia was irregular (Fig. 1B). Two quadripolar 5-Fr catheters were positioned in the His-bundle region and in the right ventricular apex, and an octapolar 5-Fr catheter was placed in the coronary sinus. An intracardiac ECG during tachycardia showed ventriculoatrial (VA) dissociations and His deflections preceding each QRS complex (Fig. 1B). During pacing from the atrium, the HV interval – the time from the initial deflection of the His bundle (H) potential and the onset of ventricular activity (V) – was 43 ms,

* Corresponding author. Tel.: +81 22 266 7111; fax: +81 22 211 8972.

E-mail address: tetsuo.yagi@nifty.com (T. Yagi).

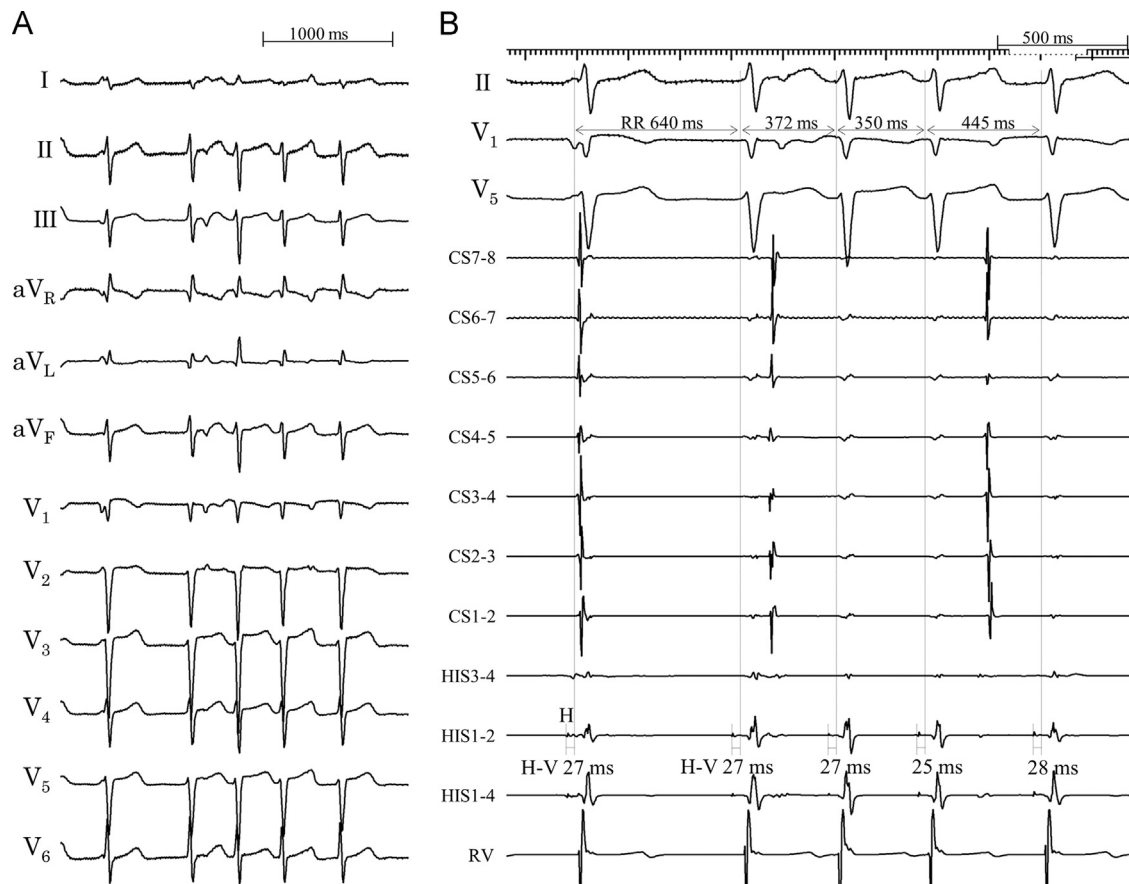


Fig. 1. (A) Twelve-lead electrocardiograms of narrow QRS tachycardia. Ventriculoatrial (VA) dissociation is apparent in lead V1. (B) Intracardiac electrogram during tachycardia. An irregular R-R interval and VA dissociation are observed during tachycardia. His bundle deflection precedes each QRS complex during tachycardia. The HV interval is 27 ms, which is shorter than that observed during atrial pacing. CS=coronary sinus; H=His bundle deflection; HIS=His bundle; and RV=right ventricle.

whereas during tachycardia, the HV interval was 27 ms. The tachycardia could not be induced or terminated by programmed ventricular stimulation. The Purkinje potentials of the RBB, recorded in the right ventricle, showed antegrade conduction of RBB signals; none of these Purkinje potentials were preceded by His deflections. Consequently, left ventricle (LV) mapping was performed, which showed antegrade conduction of the Purkinje potentials of the left anterior fascicle (LAF) during tachycardia. A tiny single potential (TSP) was recorded in the LCC (Figs. 2A, B, and 3A) and preceded the His deflection by 15 ms during tachycardia. During pacing from the atrium, the activation sequence of the TSP and His deflection was reversed (Fig. 3B). Furthermore, the TSP–His deflection interval was unchanged during the different pacing cycle lengths. These findings indicated that the TSP was connected with the HPS.

After positioning the ablation catheter in the LCC and confirming its positioning by coronary angiography, RF energy was delivered to the site where the TSP was first recorded. Following the application of RF, the tachycardia was terminated, junctional rhythm was preserved, and AV conduction remained intact while a His deflection preceded a TSP recording in the LCC. A year after the procedure, this patient was completely asymptomatic, with no evidence of tachycardia on Holter monitoring.

3. Discussion

In the present report, we describe a case of a rare type of HPS-related VT that was caused by focal activation of the conducting tissue connected to the His bundle. To our knowledge, there have

been no previous reports demonstrating HPS-related VT successfully ablated in the aortic sinus of Valsalva (ASV).

The site of the successful ablation was the earliest site where a TSP was recorded during tachycardia. The TSP preceded each His bundle deflection by 15 ms during tachycardia, but was inverted during atrial pacing. Moreover, the TSP–His deflection interval remained unchanged at different pacing cycle lengths, suggesting that the TSP was derived from the conducting system. In addition, both the RBB and LBB were activated in a proximal-to-distal direction during tachycardia. Activation of the HPS started at the distal portion of the His bundle; the His bundle was activated in a retrograde manner, whereas both the RBB and LBB were activated in an antegrade manner. Because the LCC where the TSP was recorded was not connected to either the His bundle or the LAF, the tissue conducting the TSP was presumed to be directly connected to the distal portion of the His bundle (Fig. 4A). The clinical tachycardia was associated with a narrow QRS configuration, since the activation pattern of the ventricle was the same as the junctional rhythm or atrial pacing rhythm. The HV shortening can be explained by the activation pattern of the HPS, with the timing of the antegrade conduction of the ventricular conduction system being offset by the retrograde conduction of the His bundle.

We hypothesize that the source of the TSP could be a conducting tissue such as a Purkinje fiber, in the subaortic region. This hypothesis is supported by the findings of Kurosawa et al. [5] who reported that a “dead-end-tract” – a remnant of the AV non-branching conducting system – was observed beneath the aortic root. Under normal circumstances, this branch disappears at maturity; however, it may persist as a remnant of the conducting

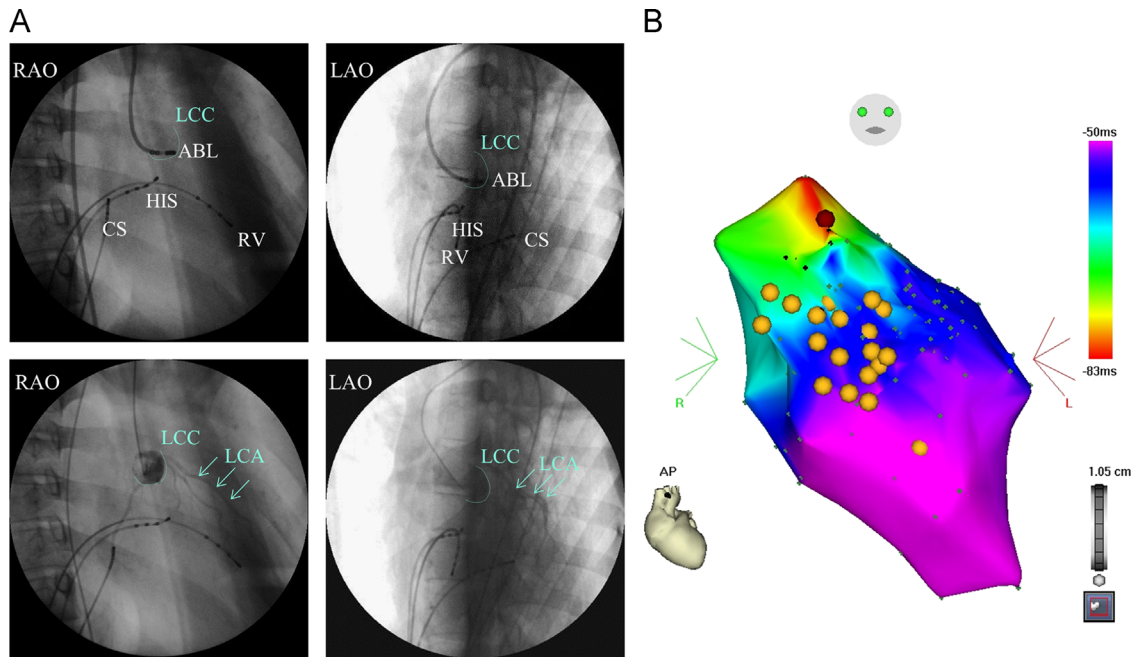


Fig. 2. (A) Upper panel: Location of the mapping catheter recording the tiny single potential (TSP) in the left coronary cusp (LCC). Lower panel: The LCC was identified by angiography of the left coronary artery. (B) Activation mapping of the left ventricle during ventricular tachycardia shows the earliest activation site in the LCC. The red dot indicates the site of successful ablation. The yellow dots indicate the site where the Purkinje potential of the left anterior fascicle was recorded. ABL=ablation catheter; CS=coronary sinus catheter; HIS=His bundle catheter; LCA= left coronary artery; LCC=left coronary cusp; and RV=right ventricle.

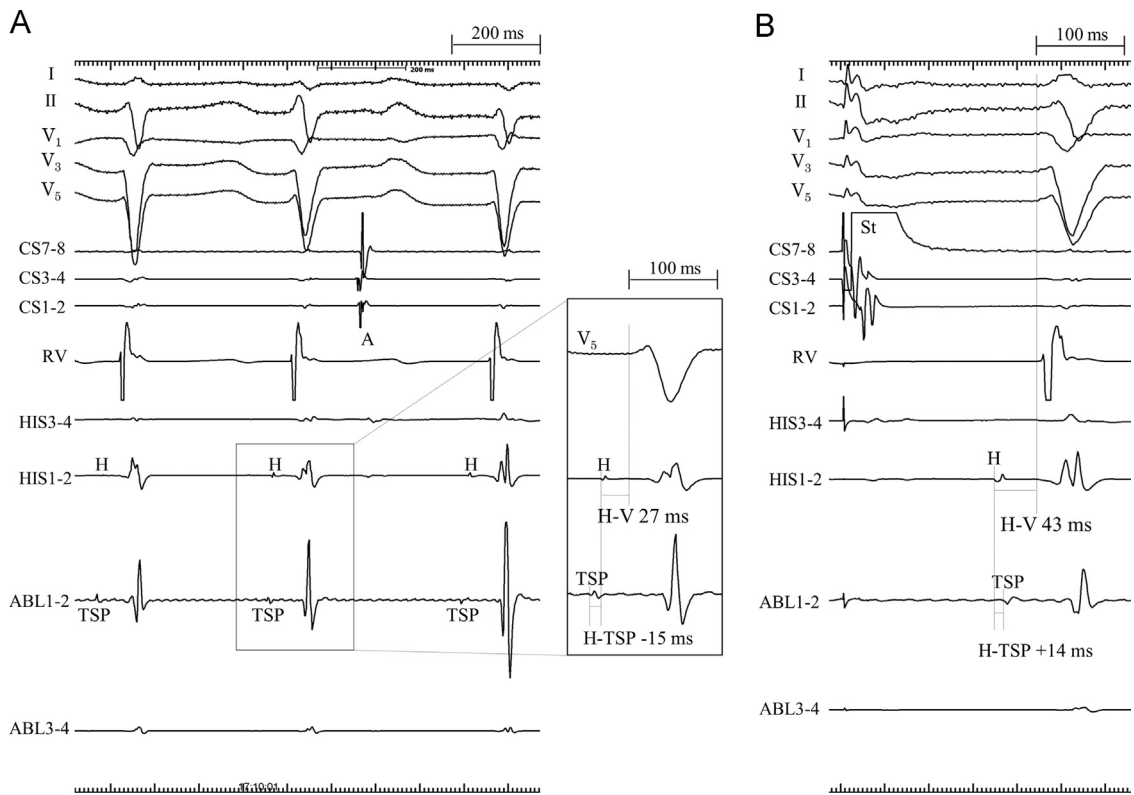


Fig. 3. (A) Intracardiac electrograms of the tiny single potential (TSP) recorded in the LCC during tachycardia. The TSP preceded the His bundle deflection by 15 ms (square panel). (B) During atrial pacing, the activation sequence of the TSP and the His bundle deflection were reversed. The HV interval during atrial pacing was longer than that during tachycardia. A=atrial potential; ABL=ablation catheter; CS=coronary sinus; H=His bundle deflection; HIS=His bundle catheter; LCC=left coronary cusp; RV=right ventricle; St=stimulus; and TSP=tiny single potential.

tissue extending from the distal portion of the His bundle. Two studies [6,7] have described this remnant tract as an “aortic ring.” If this remnant conducting tissue is present in the extended myocardium of the left coronary cusp, it could become an

arrhythmogenic focus. Anatomically, at the level of the ASV, the myocardium of the LV comes in direct contact with the aorta, thus facilitating the ablation of ventricular arrhythmias in the ASV. A focal activation from this remnant of the subaortic conducting

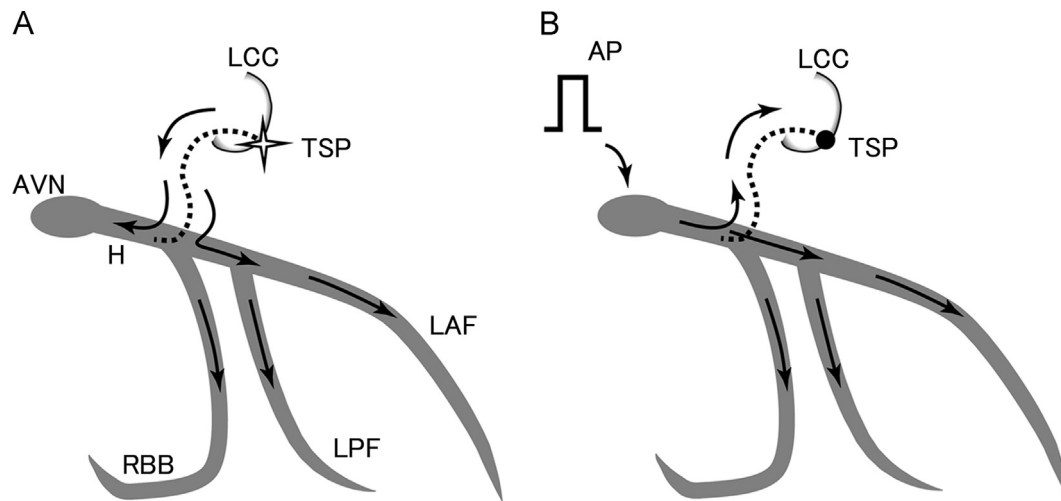


Fig. 4. Proposed mechanism for the patient's tachycardia. (A) During tachycardia, the focal activation arising from the remnant conducting system – the dotted line – beneath the LCC activates the distal portion of the His bundle, facilitating retrograde His bundle activation and antegrade activation of the ventricular conducting system. (B) During atrial pacing, His bundle activation precedes the activation of the TSP. AP=atrial pacing; AVN=atrioventricular node; H=His bundle; LAF=left anterior fascicle; LCC=left coronary cusp; LPF=left posterior fascicle; RBB=right bundle branch; and TSP=tiny single potential.

tissue could activate the distal portion of the His bundle, resulting in retrograde activation of the His bundle and antegrade activation of the ventricular conducting system (Fig. 4A).

Some cases of focal Purkinje VT, a less common form of HPS-related VT, have been reported [2–4]. Focal Purkinje VT is considered to be due to abnormal automaticity or triggered activity in the damaged HPS, arising from both the LBB and RBB. RF catheter ablation is normally attempted at the earliest site during tachycardia; however if, the VT originates in a proximal portion of the conduction system, a bundle branch block or AV block may occur. In the present case, the tachycardia was treated by RF ablation at the earliest site. As the clinical tachycardia originated in the LCC, anatomically separated from the specialized conducting system, the tachycardia could be eliminated without impairing the conducting system.

The tachycardia observed in the present case can be distinguished from junctional tachycardia (JT) in 3 ways. First, JT is an automatic tachycardia that arises from the AV node or the His bundle. However, the tachycardia observed in the present case was thought to arise from the remnant of the conducting tissue that was directly connected to the distal portion of the His bundle. The activation pattern of the HPS was associated with a narrow QRS configuration, since the activation pattern of the ventricle was the same as that of the JT. Second, the LCC in which the TSP was recorded was not close to either the AV node or the His bundle. The tachycardia observed in the present case is thought to have arisen from the remnant of the conducting tissue within the subaortic ventricular myocardium. Hence, the tachycardia we treated originated from the ventricle. Third, JT is an automatic tachycardia that arises from the AV node or the His bundle.

Therefore, RF ablation is associated with a high risk of AV block. The tachycardia observed in the present case was successfully ablated without impairing AV conduction, since it originated in neither the AV node nor the His bundle.

In conclusion, this report describes a rare type of HPS-related VT that might be due to the focal activation in the remnant of the subaortic conducting tissue connected to the distal portion of the His bundle. The VT was successfully treated by RF ablation in the LCC where the TSP was recorded.

Conflict of interest

None to declare.

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