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Original Article

Electrophysiological and anatomical background of the fusion configuration of diastolic and presystolic Purkinje potentials in patients with verapamil-sensitive idiopathic left ventricular tachycardia

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ABSTRACT

Background: It is unclear whether false tendons (FTs) are a substantial part of the reentry circuit of verapamil-sensitive idiopathic left ventricular tachycardia (ILVT). This study aimed to prove the association between FTs and the slow conduction zone by evaluating the electro-anatomical relationship between the so-called diastolic Purkinje (Pd) potentials and FTs using an electro-anatomical mapping (EAM) system (CARTO).
Methods: The 1st protocol evaluated the spatial distribution of Pd and presystolic Purkinje (Pp) potentials in 6 IVLT patients using a conventional CARTO system. In the remaining 2 patients (2nd protocol), the electro-anatomical relationship between the Pd–Pp fusion potential and the septal connection of the FT was evaluated using an EAM system incorporating an intra-cardiac echo (CARTO-Sound).
Results: Pd potentials were observed in the posterior–posteroseptal region of the LV and had a slow conduction property, whereas Pp potentials were widely distributed in the interventricular (IV) septum. At the intersection of the 2 regions, which was located in the mid-posteroseptal area, both Pd and Pp potentials were closely spaced and often had a fused configuration. In the latter 2 patients (2nd protocol), it was confirmed that the intra-cardiac points at which the Pd–Pp fusion potential was recorded were located in the vicinity of the attachment site of the FT to the IV septum. In all patients, ILVTs were successfully eliminated by the application of radiofrequency at those points.
Conclusion: FTs may at least partly contribute to the formation of the Pd potential, and thus form a critical part of the reentry circuit of ILVT.
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1. Introduction

Verapamil-sensitive idiopathic left ventricular tachycardia (ILVT) has been shown to be a clinical entity of left-sided idiopathic ventricular tachycardia (VT) [1,2]. In electrophysiological studies, two specific local potentials, the diastolic Purkinje (Pd) potential and presystolic Purkinje (Pp) potential, can be detected at the successful ablation site. The site is usually located in the left posterior septum, and these potentials are thought to be associated with the reentry circuit [3–5]. However, it remains uncertain whether these two potentials are critical for the induction and perpetuation of the tachycardia. Among these two potentials, the Pd potential is more likely to reflect a critical part of the reentry circuit (slow conduction zone, SCZ), and has been the target of catheter ablation with successful results [3–10]. The morphology of the Pd potential has been inconsistent in previous reports, showing a relatively spiky small potential [8,9], fragmented potential [10], or small slow potentials [3–7]. These might depend on individual electrophysiological properties of the Pd substrate, defined as an anatomical structure that produces a Pd potential by its electrical excitation. Entrainment pacing during tachycardia can sometimes selectively capture the Pd potential by local stimulation [5,7]. The Pd substrate is thus believed to be of a decent size, contain cardiac muscle tissue, and be insulated from the surrounding left ventricular myocardium. The tachycardia is successfully eliminated by radiofrequency (RF) deliveries targeting the Pd potential, which is commonly located in the left posterior or posteroseptal region [1–10]. Considering the anatomical features around this region, the most likely candidate for the Pd substrate might be a false tendon (FT), which has been shown to contain working myocardium or

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Table 1

Baseline data					Pd potential				Ablation site
Case no.	Age	TCL (ms)	QRS morphology	Mapping points	Size (mm)	Time (ms)	/TCL (%)	Calculated conduction velocity (m/s)	PD-QRS (ms)
1st protocol									
1.	32	283[a]	RBBB+LAD	139	34 × 13	169	59	0.38	54
2.	39	254[a]	RBBB+NW	204	35 × 14	139	54	0.25	47
3.	32	241[a]	RBBB+LAD	120	39 × 17	103	42	0.20	55
4.	20	420	RBBB+LAD	162	37 × 15	270	64	0.10	50
5.	67	270[a]	RBBB+LAD	106	20 × 15	150	55	0.19	63
6.	40	300[a]	RBBB+NW	143	23 × 18	172	57	0.12	56
2nd protocol									
7.	16	300	RBBB+NW	–	–	–	–	–	60
8.	56	330	RBBB+NW	–	–	–	–	–	55
Average	38	300		146	31 × 15	167	55	0.20	55
SD	17	56		35	8 × 2	56	7	0.10	5

Time indicates the interval between the earliest to latest Pd potential.
TCL=tachycardia cycle length.
[a] The tachycardia could be induced during intravenous infusion of isoproterenol at 0.01–0.02 μg/m/kg.

specialized conduction tissue such as Purkinje fibers [11,12]. However, the possibility that an FT forms a substantial part of the reentry circuit is controversial [13,14]. Therefore, we evaluated the spatial distribution and activation sequence of the Pd potential using a conventional 3-dimensional (3-D) electro-anatomical mapping (EAM) system. We then targeted the latest appearance of the Pd potential, which was usually fused with the Pp potential (spiky bundle potential), in the left ventricular posteroseptal region. From the theoretical point of view, such a potential should be recorded from the exit site of the SCZ (FT-septal connection). Finally, the aim of this study was to prove the electro-anatomical relationship between the Pd–Pp fusion potential and the septal connection of the FT using a new EAM system incorporating an intra-cardiac ultrasound system.

2. Material and methods

2.1. Study population

Our study population comprised 8 patients (7 male patients, mean age: 38 ± 17 years) (Table 1) with verapamil-sensitive ILVT. None of the patients had evidence of structural heart disease on transthoracic echocardiography (TTE). The mean cycle length of the spontaneous VT was 270 ± 73 ms. The QRS morphology was a right bundle branch block (RBBB) pattern with left axis deviation in 4 patients and an RBBB pattern with a north–west axis in the remaining 4 patients, indicating a superior axis in all patients. All VTs were successfully terminated by an intravenous infusion of verapamil (3–5 mg).

2.2. Methods

Our study protocol was two-fold. *The 1st protocol* evaluated the spatial distribution of the Pd and Pp potentials and the conduction property and activation sequence of Pd potentials in 6 patients (Pts. 1–6 in Table 1). These were mapped during an induced clinical VT using the conventional EAM system. The electrophysiological study was performed after obtaining written informed consent. All anti-arrhythmic agents were discontinued for at least 3 half-lives before the study. Three multipolar electrode catheters were positioned in the right atrial appendage, right ventricular apex (RVA), and His bundle region. A 7F steerable catheter with a 4-mm electrode tip (Navi Star, Biosense Webster) was inserted via the left femoral artery and retrogradely advanced into the LV for endocardial mapping and catheter ablation. Programmed ventricular stimulation including up to triple extrastimuli at 2 basic cycle lengths (600 ms and 400 ms) and burst pacing was applied from both the RVA and RV outflow tract to induce VT. If a sustained VT could not be induced, the programmed pacing was repeated under an intravenous infusion of isoproterenol (0.05–0.1 μg/kg/min).

2.2.1. Left ventricular endocardial mapping during VT

When the induced VT was sustained, the LV was mapped using a conventional EAM system (CARTO, Biosense Webster Inc., Diamond Bar, CA, USA). The local bipolar electrogram was recorded simultaneously either with the EAM system and an EP-WorkMate (EP MedSystems Inc., Mt. Arlington, New Jersey, USA) or with a LabSystem PRO EP Recording System (Bard Electrophysiology, Lowell, Massachusetts, USA) at a filter setting of 30–500 Hz. A Pd potential was defined as a dull potential observed in the diastolic phase during VT that preceded the onset of the QRS complex by more than 40 ms (Fig. 1). On the CARTO mapping image, Pd potentials were marked with yellow dots. The conduction velocity of the SCZ was simply calculated by dividing the length between the earliest Pd site and the latest Pd site by the conduction time between these sites. The Pp potential was defined as a spiky potential observed during the presystolic phase that preceded the onset of the QRS complex by more than 0 ms (Fig. 1). Pp potentials were marked with white dots. The points at which Pd and Pp potentials were observed in proximity to one another in the same recording were marked with blue dots.

2.2.2. The 2nd protocol

An additional protocol was carried out to confirm that the anatomical structure responsible for the Pd potential (Pd substrate) was the FT. EAM utilizing a new CARTO system incorporating an intracardiac echocardiogram (CARTO-Sound, Biosense Webster Inc.) was performed in the 2 most recent patients. An FT bridging from the left posterior papillary muscle to the left ventricular septum was clearly seen in each patient. We examined the local potential at the tendon–septal junction demonstrating a Pd–Pp fusion potential to clarify whether this indicated the successive activation of the false tendon and the connecting Purkinje fibers.

2.2.3. Catheter ablation procedures

The target site for catheter ablation was determined according to specific local electrogram features. During endocardial mapping of the VT, we identified points at which the Pd and Pp potentials occurred in proximity to one another in the same recording

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