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Electrocardiographic characteristics of idiopathic premature ventricular contractions originating from the junction of the right ventricular outflow tract and tricuspid annulus

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ABSTRACT

Aims: The right ventricular outflow tract (RVOT) and tricuspid annulus (TA) are common origins for idiopathic PVCs from the right ventricle. We sought to clarify the characteristics of a subgroup of idiopathic PVCs originating from the RVOT–TA junction.
Methods and results: Surface ECG and intra-cardiac electrophysiological characteristics were analyzed in 101 patients with frequent PVCs who underwent successful RFCA in the right ventricle. Pacing was performed in the right ventricle in another 5 control subjects. The origin of PVCs determined by the successful ablation site was at the RVOT, the TA and the RVOT–TA junction in 78, 11 and 12 patients, respectively. The PVCs originating from RVOT–TA junction showed a monophasic R wave in leads I, II, III and aVF and a flat QRS complex in lead aVL. A flat QRS complex (rsr′, qs, qr, rs or r pattern, mean r or qs amplitude, 0.3 ± 0.1 mV) in lead aVL distinguished the RVOT–TA junction origin from the RVOT (deep negative, -0.7 ± 0.4 mV) and the TA (tall positive, 0.8 ± 0.3 mV) origins. Activation mapping and pace mapping strategies were successfully applied to localize this specific origin of the PVCs. Pacing at the RVOT–TA junction in the control subjects validated a flat QRS complex in lead aVL.
Conclusion: We report for the first time that the RVOT–TA junction is a non-rare but distinct origin of right ventricular PVCs. The flat QRS complex in lead aVL distinguishes this origin from RVOT and TA. RFCA is highly effective for eliminating PVCs in this origin.

1. Introduction

The right ventricular outflow tract (RVOT) is the most common location of origin of idiopathic premature ventricular contractions (PVCs) [1]. The tricuspid annulus (TA) is also reported as another origin of idiopathic PVCs in the right ventricle [2]. Radiofrequency catheter ablation (RFCA) is effective in eliminating these PVCs [2–5]. Recently, we encountered a subgroup of challenging cases in which PVCs were located at the junction between RVOT and TA (RTJ). In this study, the distinct ECG and intra-cardiac electrophysiological characteristics of these PVCs originating from RTJ were clarified.

2. Methods

2.1. Study population

This retrospective study included 101 consecutive patients (46 men and 55 women; age 41 ± 16 years) with symptomatic, idiopathic PVCs who underwent a successful RFCA procedure in our hospital between January 2009 and December 2013. Only PVCs originating from the right ventricle (including TA, RVOT and RTJ), as determined by the successful ablation site, were enrolled and other origins, such as the aortic sinuses and the mitral annulus in the left ventricle, were excluded from this study. All patients had a normal ECG during sinus rhythm. Complete physical examination, echocardiography, exercise stress testing or coronary angiography demonstrated no evidence of structural heart diseases in any patient. Before RFCA, 12-lead ECGs were obtained, and 24-h ambulatory Holter monitoring was conducted at least once. Catheter ablation of PVC was performed in 1) patients with >10,000 PVCs/24 h; 2) patients who remained symptomatic despite conservative treatment; or 3) patients with a decline in left ventricular systolic function associated with PVC. Ethical approval was obtained from the hospital's ethics committee, and all patients gave written informed consent before participation.

Another 5 control subjects (3 women and 2 men; mean age 45 ± 10 years) were enrolled for the pacing study to confirm the ECG characteristics of RTJ PVCs. These 5 patients underwent RFCA for paroxysmal supraventricular tachycardia, including 4 cases of atrioventricular nodal reentrant tachycardia and 1 case of atrioventricular reentrant tachycardia. None had structural heart diseases.

2.2. Mapping and ablation

After withdrawal of anti-arrhythmic drugs for at least 5 half-lives, electrophysiological evaluation and RFCA were performed. The RFCA was performed when frequent PVCs were present. The probable origin of PVCs was postulated according to the widely accepted ECG clues reported by previous studies [6–9]. A 7-F ablation catheter with a 4-mm distal electrode and a deflectable tip (Biosense Webster, Diamond Bar, California) was used for mapping and ablation. Activation mapping and pace mapping were combined to identify the origin of PVCs. Pace mapping was performed at a pacing cycle length of 500 ms or 400 ms and a stimulus amplitude of 1 mA greater than the late diastolic threshold.

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Three-dimensional electromagnetic mapping (CARTO, Biosense Webster, Diamond Bar, CA, USA) was performed in some patients. Radiofrequency energy was applied at the site where the earliest ventricular activation was recorded, and perfect or near-perfect pace mapping was obtained. Radiofrequency energy was delivered using a maximum power of 35 to 50 W and a maximum electrode-tissue interface temperature of 50 °C–55 °C. The application time of the RF energy was 60–90 s. Successful catheter ablation was determined by the absence of spontaneous PVCs immediately after the start of RF and the absence of PVCs with the same morphology after 24 h of Holter monitoring in the absence of anti-arrhythmic drugs.

2.3. Definition of location of PVC origin

The location of PVC origin was identified as the successful ablation site according to the fluoroscopic views in right anterior oblique (RAO) and left anterior oblique (LAO) projection. The RVOT origin was easily identified in the superior portion of the right ventricle. The TA origin was identified by the fluoroscopic views and further confirmed by the recording with clear atrial and ventricular electrograms at the successful ablation site. The ratio of the atrial to ventricular electrograms at the ablation site was <1. The RTJ was distinguished by the fluoroscopic views and local electrograms and further confirmed by angiography, echocardiography or three-dimensional electromagnetic anatomy. The location of this area was significantly lower compared with RVOT in the fluoroscopic views. The local recording at this area did not show atrial activation, indicating distance from the TA.

2.4. Pacing study

A pacing study was performed in 5 control subjects who underwent RFCA for paroxysmal supraventricular tachycardia. After successful ablation of their original tachycardia, pacing at a cycle length of 500 ms or 400 ms was performed from the posterior RVOT, the anterior TA and the RTJ. The pacing sites were confirmed by fluoroscopy and local recordings. The paced QRS was compared among these sites.

2.5. Statistical analysis

All continuous variables were expressed as the mean ± standard deviation (SD). Comparisons among groups were performed using one-way analysis of variance (ANOVA) followed by Bonferroni's test. All statistical analysis was accomplished using SPSS 16.0 for Windows (SPSS Inc., Chicago, IL, USA). Statistical significance was defined as $P < 0.05$.

3. Results

3.1. PVC origins

All the patients had more than 10,000 PVCs in 24-h Holter monitoring (Table 1). In these 101 patients, the origin determined by the successful ablation site was at the RVOT in 78 patients, the TA in 11 patients and the RTJ in 12 patients. In addition to fluoroscopy, RVOT origin was confirmed by electroanatomical mapping in 21 out of 78 patients. There was no atrial electrogram in local recording at RVOT. The TA origin was also confirmed by fluoroscopy and electroanatomical mapping and further confirmed by clear atrial and ventricular electrogram recordings at the successful ablation site. Electroanatomical mapping was used in 3 cases. The RTJ origin was confirmed by fluoroscopy (N = 12), electroanatomical mapping (N = 3) or angiography of the right ventricle (N = 3). In the latter 6 cases, only fluoroscopy was used to determine the location because the ECG indicated a typical RTJ origin. The local electrogram at the successful ablation site did not show an atrial electrogram, suggesting remoteness from the TA.

3.2. ECG characteristics of PVCs originating from RTJ, RVOT and TA

The PVCs originating from the RTJ showed a positive QRS polarity in leads I, II, III and aVF in all patients (Fig. 1). The duration of the QRS complex of the PVCs was 160.2 ± 13.5 ms (range 128–205 ms). The R wave amplitude in lead I was 0.8 ± 0.3 mV, similar to the R wave amplitude of PVCs arising from the TA (Table 1). The amplitude of the R wave in leads II, III and aVF for all 12 patients with PVCs originating from the RTJ was 1.4 ± 0.2 mV, 0.9 ± 0.4 mV, and 1.1 ± 0.3 mV, respectively. A flat QRS (rsr′, qs, qr or r pattern, Fig. 1) was recorded in lead aVL with a mean QRS amplitude (r or qs amplitude) of 0.3 ± 0.1 mV, significantly lower than in PVCs arising from RVOT or TA (Table 1). Notching of the R wave was recorded in 8 patients in lead III. A deep negative wave was found in lead aVR in all 12 patients. The precordial lead transition was at V2–V3 in 4 patients, V3 in 2 patients, V3–V4 in 5 patients and V4 in 1 patient, respectively. In the 4 patients with transition at V2–V3, an early transition was also noticed at the sinus beats.

The PVCs originating from RVOT manifested left bundle branch block morphology and inferior frontal plane axis (monophasic R waves in leads II, III, and aVF, Fig. 2). The duration of the QRS complex of the PVCs was 164.6 ± 15.4 ms (range 122–195 ms). The amplitude of the R wave in leads II, III and aVF for all 78 patients was 1.5 ± 0.4 mV, 1.4 ± 0.3 mV, and 1.4 ± 0.4 mV, respectively. The precordial lead transition occurred at or later than lead V3. The QRS wave polarity in lead aVL was deep negative in 78 patients (mean QRS amplitude −0.7 ± 0.4 mV). Twenty-four patients in whom PVCs originated from the posterior RVOT showed a positive QRS wave polarity in lead I (mean QRS amplitude 0.6 ± 0.2 mV).

The PVCs originating from TA also presented left bundle branch block morphology with the precordial lead transition at or later than lead V3 (Fig. 2). However, PVCs originating from the septal TA exhibited the precordial lead transition between lead V2 and V3 (n = 4).

Table 1
Clinical, electrocardiographic, and electrophysiologic characteristics.

	RTJ (n = 12)	RVOT (n = 78)	TA (n = 11)	P value
Age (years)	46 ± 15	40 ± 16	43 ± 18	NS
Gender, n (M/F)	7/5	32/46	7/4	<0.05
PVC count (no./24 h)	21,518 ± 6432	20,018 ± 5120	19,560 ± 6020	NS
QRS				
Duration (ms)	160.2 ± 13.5	164.6 ± 15.4	155.2 ± 14.8	NS
R amplitude (mV) in lead I	0.8 ± 0.3	0.6 ± 0.2[a]	0.7 ± 0.4	NS
R amplitude (mV) in inferior leads	1.1 ± 0.4	1.4 ± 0.6	1.2 ± 0.3[b]	NS
R amplitude (mV) in Lead aVL	0.3 ± 0.1[c]	-0.7 ± 0.4	0.8 ± 0.3	<0.05
Precordial transition zone score[d]	3.1 ± 0.5	3.3 ± 0.6	3.5 ± 0.6	NS
V-QRS (ms) at ablation site	30 ± 12	34 ± 11	28 ± 10	NS
RF duration (s)	110 ± 12	96 ± 9	108 ± 11	<0.05
Fluoroscopy time (min)	16 ± 8	10 ± 6	14 ± 7	<0.05
No. of RF lesions	4.1 ± 0.9	2.0 ± 1.0	3.5 ± 1.1	<0.05

[a] Calculated from 24 patients with PVCs originating from posterior RVOT and showing a positive QRS wave polarity in lead I.
[b] Calculated from 6 patients with PVCs originating from anterior, anteroseptum and antero-lateral portion of TV and showing a positive QRS wave polarity in all inferior leads. R amplitude was calculated as an averaged voltage of all inferior leads (II, III, aVF).
[c] The R amplitude was calculated as r or qs amplitude.
[d] The transition zone was defined as the position of the precordial leads in which the amplitudes of the R and S waves were equal. According to the site of the transition zone in the precordial leads, transition zone score was graded in 0.5-point increments [10]. When the site of R-wave transition locates in V1, the transition zone score is 1. When the site of R-wave transition locates between V1 and V2, the transition zone score is 1.5. When the site of R-wave transition locates in V6, the transition zone score is 6.