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The relationship between the S-wave in lead 1 and recurrence of RVOT PVC ablation☆

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ABSTRACT

Background: Radiofrequency catheter ablation (CA) is a common non-pharmacological treatment option for ventricular premature contractions (PVCs) originating from right ventricular outflow tract (RVOT). In this study, we aimed to investigate the relationship between recurrence after CA for RVOT-PVC and S-wave in lead 1 that was shown to be associated with RVOT depolarization.
Methodology: A total of 104 patients who were referred to our clinic for CA for idiopathic RVOT-PVC between 2012 and 2015 years were enrolled. All ECG parameters were measured before and after the ablation procedure.
Results: Ablation was successful in 100 patients (96,1%). These patients with successful ablation were followed for a mean duration of 1078 days. 13 patients (13%) had recurrence. Univariate logistic regression analysis revealed age (odds ratio: 1.916, p:0,012), presence of post-procedural S1 (odds ratio:1.040 p:0,028), post-procedural S1 area (oddsratio:1.023 p:0,041), ΔS1 area (odds ratio:1.242 p:0,004) as predictors for recurrence. Multivariate logistic regression analysis detected age (odds ratio:1.053 p:0,032) and ΔS1 area (odds ratio:0.701 p:0,009) as predictors for recurrence.
Conclusion: Radiofrequency CA for RVOT-PVC can be performed with high procedural success and low complication rates. Age and ΔS1 area might be helpful for prediction of recurrence after CA.

Introduction

Majority of idiopathic ventricular arrhytmias (VA) originate from right ventricular outflow tract (RVOT) [1]. Radiofrequency (RF) CA is a common non-pharmacological treatment option for RVOT-PVC [2–3]. Catheter ablation (CA) of RVOT-PVC is recommended by European Society of Cardiology guidelines with Class I indication [4].CA is highly effective for the treatment of these patients. However, there is around 10% recurrence rate after the ablation procedure [5]. Previous studies investigating the relationship between ECG and recurrence after catheter ablation for RVOT PVC reported a great variety of parameters as predictors for recurrence [6].

S-wave in lead 1 typically indicates activation of basal regions in both ventricles and is determinant for RVOT depolarization, hence is typically seen in conditions associated with right ventricular enlargement and fibrosis [7]. S-wave in lead 1 is reported to have prognostic significance in Brugada Syndrome and may reflect delayed activation of the RVOT [8]. It may be associated with increased rate of recurrence after RVOT-PVC ablation.

We aimed to investigate the relationship between recurrence after CA of RVOT-PVC and presence of S-wave in lead 1.

Methodology

Study population

A total of 104 patients who were referred to our clinic for idiopathic RVOT-PVC between 2012 and 2015 years were enrolled. PVCs had left bundle branch block (LBBB) morphology and inferior axis in all patients. Transthoracic echocardiography (TTE) and exercise test were performed in all patients. Further imaging methods such as cardiac magnetic resonance (MR) imaging and coronary angiography were done when necessary in order to rule out patients with structural heart disease. Patients with a history of ablation for RVOT-PVC, renal failure, electrolyte imbalance and patients who had structural heart disease were excluded. Patients with epsilon wave in V1–V3, Right bundle block (RBBB) and T wave inversion in V1–V3 were also excluded. The study

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was approved by the Ethics Committee of the İstanbul Medipol University Medicine Faculty.

ECG evaluation

Pre-procedural and post-procedural 12-lead electrocardiograms (ECGs) were performed by the conventional technique at a paper speed of 25 mm/s. Post-procedural 12-lead ECG was obtained as soon as the patient was transferred to his/her bed from the electrophysiology laboratory. All ECG parameters were measured by two experienced cardiologists who were blind to clinical data of the patients. ECG measurements were done by using a magnifying glass. The same magnifying glass was used by two cardiologists. Cardiologists were left free to use any magnification for accurate ECG measurements. The presence of an S-wave in leads 1 was examined. The amplitude (mV) from the isoelectric line to the nadir of the S-wave and the duration (ms) from the beginning to the end of the S-wave in leads 1 was measured with calipers. The area (mm^2) of the S-wave was calculated as the product of amplitude and duration (Fig. 1 – ECG evaluation). The QRS interval duration and QT intervals were measured. QT interval was defined as the interval between from the beginning of the QRS complex to the end of T-wave. The QT intervals were corrected according to the formula of Bazzett, where ($QTc = QT/\sqrt{RR}$). V1r and V6 s amplitudes were measured. TpTe interval in leads V2 and V6 was defined as the interval from the peak of the T wave to the end of T wave.

Normal beats not the PVCs were checked for the presence of S-wave in lead 1. Similarly, other ECG parametres (QRS duration, QT, QTc, V1r, V6s, TpTe) were measured from normal beats.

Structural analysis

Echocardiography

All patients underwent transthoracic echocardiography prior to the study. The focus on the right ventricle included function, as well as a description of localized right ventricular aneurysms, segmental dilatation or regional wall motion abnormalities.

Magnetic resonance imaging

MRI of the heart was performed according to a standardized technique. Each study was examined for abnormalities in the morphology of the right and left ventricle. Measurements of right ventricular free wall thickness were made with focal thinning defined as a wall thickness of <2 mm. Fatty deposition was defined as high intensity intramyocardial lesions on T1-weighted images; the presence and extent of fatty deposition was recorded. Cine studies evaluated wall motion with areas of hypokinesis, dyskinesis or aneurysm formation. MRI abnormalities included right ventricular dilatation, wall motion abnormalities, diffuse right ventricular wall thinning and diffuse fatty infiltration.

RF catheter ablation

All procedures were performed by single operator. Standardized electophysiological study was performed in a fasting state. No sedation was given to the patients in order not to suppress spontaneous PVCs. Antiarrhythmic drugs were discontinued for a minimum of five half-lives before RF CA. Blood pressure, heart rate, rhythm and oxygen saturation was continuously monitored during the procedure. Two femoral vein punctures were done. A decapolar diagnostic catheter was advanced from 6F sheath to the coronary sinus. An ablation catheter (Marinr®; Medtronic Inc., Minneapolis, MN, USA) was advanced from 7F sheath to the right ventricle. When there was spontaneous PVC, activation mapping was done by ablation catheter. A 3D mapping system (En Site Nav XTM, St Jude Inc., St Paul, MN, USA) was used for localization of arrhythmogenic foci. Ablation catheter is also used for right ventricular pacing when necessary. In the absence of spontaneous PVC, rapid ventricular pacing and programmed stimulation up to three extrastimuli were performed with a catheter placed at the right ventricular apex and RVOT sequentially. If PVC was still not inducible, intravenous isoprenaline 1–5 mic g/min was infused. If spontaneous PVCs were

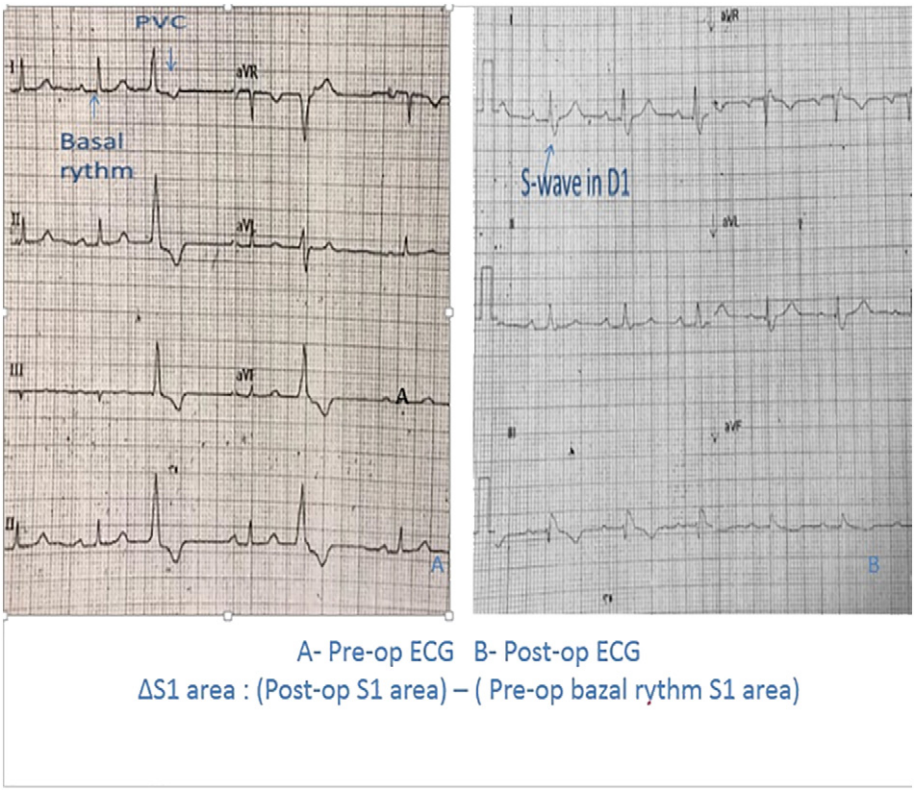

Fig. 1. Preprocedural and post procedural ECG.

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