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Successful Ablation of Single Reentrant Ventricular Tachycardia Arising from Peri-Aortic Scar in a Patient with an Apparently Normal Heart

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Abstract

Peri-aortic region is one of the arrhythmogenic foci associated not only with idiopathic ventricular tachycardia (VT), but also scar-related VT in patients with an apparently normal heart.¹⁻³ A recent study reported that the patients with scar-related VT were significantly older, had a frequent history of hypertension, and inducibility of multiple monomorphic VTs compared to the patients with idiopathic VT.² However, whether these clinical features are the causes of the peri-aortic scar or innocent by-standers, remain uncertain. Here, we present a relatively young normotensive patient with a peri-aortic scar and emphasize the importance of cardiac MRI to detect latent arrhthmogenic substrates.

Case Report

A 58-year old man with a 3-month history of palpitations had a documented sustained VT with a cycle length (CL) 350ms on the 24-hour Holter ECG. A diagnosis of idiopathic VT was made based on the normal findings of the twelve-lead ECG, and no evidence of structural heart disease with coronary angiography and echocardiography. The VT was refractory to the beta-blocker and he was referred to our hospital for further treatment. Cardiac MRI was scheduled for an intensive diagnostic work-up prior to the radiofrequency catheter ablation (RFCA). Cardiac MRI revealed normal right and left ventricular chamber sizes without any abnormalities of the wall motion, which were consistent with the echocardiographic findings. However, contrast enhancement MRI revealed a scar involvement in the peri-aortic region (Figure 1). An electrophysiological study was scheduled after all antiarrhythmic

Key Words:

Ablation, Ventricular Tachycardia, Scar Detection, MRI, Structural Normal Heart.

Disclosures:

Corresponding Author: Hiro Yamasaki, MD Department of Electrophysiology, Heart Center, University of Leipzig, Struempellstr. 39, Leipzig, Sachsen, 04289, Germany. drugs had been discontinued for at least five half-lives. Programmed simulation from the right ventricular apex (RVA) repeatedly induced a hemodynamic tolerable monomorphic VT (CL 350ms) with a right bundle branch block and inferior axis morphology (Figure 1), which was compatible with the scar location and considered as the clinical VT from the tachycardia CL and clinical symptoms. Based on the MRI findings and the reentrant electrophysiological properties, we decided to perform detailed mapping of the outflow tract region in both ventricles using an electroanatomical mapping system (CARTO 3, Biosense Webster Inc, Diamond Bar, CA). Initially, activation mapping in the right ventricle (RV) was performed during the VT using a 4-mm distal tip open-irrigated catheter (NaviStar ThermoCool, Biosense Webster), which revealed the earliest activation site on the RV septum with a local ventricular activation 30ms earlier than the onset of the QRS complex (Figure 2), but with preserved bi-polar voltage signals (> 1.5mV). After terminating the VT with high-rate pacing from the RVA, further voltage mapping of the left ventricle (LV), mainly in the outflow tract, was performed via a transseptal approach based on the previous MRI findings. A total of 57 points were collected to clarify the border of the low voltage area (<1.5mV). The low voltage area with local abnormal ventricular activity was confined in the limited area which was proximal to the His recording site and distributed from the septal to the anterior portion of the peri-aortic region (Figure 1). The QRS morphology during pace mapping in the low voltage area was showed only minor differences in the QRS morphology to the clinical VT-QRS morphology with a stimulus-QRS interval of 73ms (Figure 1). Furthermore, the local ventricular activation at that site during the VT exhibited a fragmented potential preceding the onset of the

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outflow tract; RA=right atrium; RV=right ventricle

QRS complex by 68ms and was considered as the presumptive exit of the VT (Figures 2 and 3).⁴ Radiofrequency energy was delivered at a power of 40W and the VT was terminated within 6 seconds (Figure 4). After the termination of the VT, energy applications were further delivered at the border zone of the low voltage area for substrate modification. The clinical VT could no longer be induced thereafter under extrastimulation with 3 extrastimuli scanned to a minimum coupling interval of 180ms from the RVA. After the successful RFCA of the single monomorphic VT, an event recorder was implanted. The patient was discharged and after 3 months of



follow-up, no recurrence has been observed.

Discussion

We described a patient with an apparently normal heart, but with a single monomorphic scar-related VT from peri-aortic scar. The peri-aortic region is a common arrhythmogenic focus not only for idiopathic VT, but also VT in dilated cardiomyopathy (DCM) patients.⁵⁻⁶ However, VT without any abnormalities in a routine diagnostic work-up including a 12-lead-ECG, transthoracic echocardiography, and coronary angiography, is categorized as idiopathic VT,7 and further evaluation using cardiac MRI is left to the physician's decision.8 Therefore, the clinical features of patients with reentrant VTs from peri-aortic scar have only been evaluated in a small study. Nagashima et al. described the clinical features of these patients and mentioned that those patients with scar-related VT were significantly older and had a frequent history of hypertension, and raised the possibility that cardiac remodeling with aging and hypertension might be the cause of the peri-aortic scar.² However, our patient characteristics were not consistent with the previous findings and suggested that the peri-aortic scar was more likely to be an early presentation of a cardiomyopathic process. In general, the diagnosis of DCM is masked before the impairment of the LV function or an electrocardiographic abnormality including an emergence of VT. Therefore, cardiac MRI should always be included in the routine diagnostic work-up of VT regardless of the clinical presentation.

Recently, the feasibility of RFCA as the primary management of hemodynamically tolerated monomorphic VT in patients with an LV function >30% has been described.⁹ However, data on the clinical course regarding the LV function and/or VT recurrence after the RFCA in patients with peri-aortic scar is scarce. Moreover, a high recurrence rate of the VT after the ablation in DCM patients with anterior septal scar has previously been reported.⁵⁻⁶ Therefore, our decision was to follow the patient with an event recorder system





32 Journal of Atrial Fibrillation



which enabled us to understand the clinical course of the patient after the successful RFCA of the scar-related VT arising from periaortic scar.

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