Correspondence

Non-response to Implantable Cardioverter Defibrillator in a Post-Infarction Patient with Recurrent Ventricular Tachycardia After Catheter Ablation

Xiang-Fei Feng, Jian Sun, Jun Wang, Yi-Gang Li
Department of Cardiology, Xinhua Hospital, Shanghai Jiaotong University School of Medicine, Shanghai 200092, China

To the Editor: A 73-year-old man presented to Department of Cardiology at Xinhua Hospital, Shanghai Jiaotong University School of Medicine with a history of anterior wall myocardial infarction (MI) and multimorphic ventricular tachycardia (VT) in approximately September 2013. His VT burden became progressive increase despite treatment with percutaneous coronary intervention (PCI) and multiple antiarrhythmic medications such as beta blockers, amiodarone, magnesium sulfate, and so on.

One month after PCI, the patient received an implantable cardioverter defibrillator (ICD) (Marquis, Medtronic, USA) implantation due to complex ventricular arrhythmias and depressed left ventricular ejection fraction (36%). The detection and therapy programming of the device included 2 zones: ventricular fibrillation (VF) zone (rate > 188 beats/min) and VT zone (rate 150–188 beats/min to VF zone). Later, the patient started to experience palpitations and shocks during physical activity. One month after the ICD implanted, the patient had to have the first radiofrequency catheter ablation (RFCA). Electroanatomic substrate mapping demonstrated a large area (53 cm²) of low amplitude electrograms (<0.5 mv) consistent with scar extending forward from the septum over to the lateral aspect of the heart [Figure 1]. Programmed stimulation induced two types of VT, VT1 (cycle-length (CL) 380 ms) “nonclinical” and the remaining VT2 (CL 430 ms) “clinical” as determined predominantly from the intracardiac electrogram morphology and CL analysis. A limited activation map demonstrated early activation at the apex and the lateral aspect of the scar, with a local electrogram preceding the onset of the QRS by approximately 70 ms. Entrainment mapping near these locations demonstrated concealed entrainment with a postingspacing interval minus tachycardia CL of 15 ms. Ablation at these sites resulted in immediate VT termination.

Two weeks after the first RFCA, incessant, tolerated, slow (120–150 betas/min) VTs were detected by electrocardiogram monitoring, and these VTs were neither identified by ICD nor responded to multiple anti-arrhythmic medications. The ICD device was reprogrammed and new antitachycardia pacing (ATP) algorithms (VT zone: rate 130–188 beats/min) were performed, but this adjustment was failed and ended with shock therapy. To reduce the risk of receiving an inappropriate shock, the patient had a second RFCA treatment 1-month later. The area of second scarring had a larger scar (69 cm²) as assessed by electroanatomic mapping compared with the first, and the VT3 (CL 500 ms) critical isthmus was limited to the new region, which was “adjacent” to prior ablation lesions [Figure 1]. Radiofrequency lesions

Figure 1: The electroanatomic map (right anterior oblique view) and ventricular tachycardia morphology. The top panel: electroanatomic map of the initial ablation procedure in a patient with recurrent ventricular tachycardia (VT). Brown tags indicated sites with capture showing distinct VT1/VT2 morphology. Blue tags showed the locations of line ablation lesions. The bottom panel: electroanatomic map obtained during the repeat ablation procedure. Shown were the locations of new low-voltage area (black line coil) which was “adjacent” to prior ablation lesions, capture sites showing distinct VT 3 morphology (brown tags), and line ablation lesions (blue tags).

Access this article online
Quick Response Code: www.cmj.org
DOI: 10.4103/0366-6999.150122

Address for correspondence: Dr. Yi-Gang Li, Department of Cardiology, Xinhua Hospital, Shanghai Jiaotong University School of Medicine, Shanghai 200092, China
E-Mail: drlyigang@outlook.com
were placed at the new scar site and extended to the sites of late potentials in order to consolidate the lesions. Following ablation, VTs could no longer be induced. The procedure went well and there were no evidences of recurrent VTs and shock during 6 months postoperative follow-up.

Several factors influence the termination of VT by ATP. The distance between the pacing site and the VT circuit may critically affect the ATP effectiveness. Small changes in the VT CL were useful in predicting the ATP success. In the present case, although an episode of VT had initiated, as the R-R intervals fluctuated beyond the detection threshold of ATP therapy, the device did not perceive these VT events, consequently ATP therapy was withheld.

Little is known about the reason for recurrent VT after acutely “successful” catheter ablation. Progressive fibrosis and remodeling may lead to the development of VT after the index event. Continued infarct remodeling may lead to the formation of new VT circuits that were not present during the initial ablation session.

In this case, the present patient had recurrent VTs that were resistant to treatment with antiarrhythmic drugs and ICD. The coexistence of three conditions is rare, and it is a challenge to treat.

Some VTs failed to ATP therapy can lead to a delay in appropriate therapy, even contribute to a negative consequence. Thus, RFCA has become an important tool for the successful management of these VTs. It could result in the prevention of future VTs by ablating regions of slow conduction and all late potentials. Hence, it is possible and effective for us to finish a linear pattern of lesions through sites of late potentials and good pace-maps.

References
1. Byrd IA, Kay MW, Pollard AE. Interactions between paced wavefronts and monomorphic ventricular tachycardia: Implications for antitachycardia pacing. J Cardiovasc Electrophysiol 2006;17:1129-39.
2. Jiménez-Candil J, Hernández J, Martín A, Moríñigo J, López R, Ledesma C, et al. Influence of cycle length variations on antitachycardia pacing effectiveness among ICD patients. Heart Rhythm 2013;10:207-13.
3. Gerstenfeld EP. Recurrent ventricular tachycardia after catheter ablation in post-infarct cardiomyopathy: “Failure” of ablation or progression of the substrate? J Am Coll Cardiol 2013;61:74-6.
4. Yokokawa M, Desjardins B, Crawford T, Good E, Morady F, Bogun F. Reasons for recurrent ventricular tachycardia after catheter ablation of post-infarction ventricular tachycardia. J Am Coll Cardiol 2013;61:66-73.
5. Jais P, Maury P, Khairy P, Sacher F, Nault I, Komatsu Y, et al. Elimination of local abnormal ventricular activities: A new end point for substrate modification in patients with scar-related ventricular tachycardia. Circulation 2012;125:2184-96.

Received: 16-06-2014 Edited by: De Wang
How to cite this article: Feng XF, Sun J, Wang J, Li YG. Non-response to Implantable Cardioverter Defibrillator in a Post-Infarction Patient with Recurrent Ventricular Tachycardia After Catheter Ablation. Chin Med J 2015;128:415-6.

Source of Support: Nil. Conflict of Interest: None declared.