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*Letter to the Editor* **· Open Access ·**

# Radiofrequency catheter ablation of ventricular tachycardia in a patient with dermatomyositis

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A 51-year old male who presented at our hospital for recurrent palpitation for several months was diagnosed dermatomyositis ten years ago and had interstitial lung disease since two years ago. Recently，he was admitted for atypical hepatitis, and received maintenance treatment of oral corticosteroids. The twelve-lead ECG during palpitation showed ventricular tachycardia (VT) with a superior axis, left bundle branch block morphology at a rate of 160 beats/min (Figure 1A). In addition, paroxysmal atrial fibrillation with bigeminy and trigeminy ventricular premature beats (Figure 1B) was also documented. Cardiac echocardiogram revealed dilated right ventricle (RV), left ventricle (LV) and both atria. Both LV and RV systolic function were subnormal (LV ejection fraction = 43%; RV fractional area change = 48%) with dyskinesia of the LV anteroseptal wall. Coronary angiography was normal. Cardiac MRI revealed areas of fatty tissue over the RV and LV apex, but there were no definite areas of fibrosis noted.

During electrophysiological studies, monomorphic VT (VT-1; cycle length: 390 ms; Figure 2A) was induced with isoproterenol instead of programed pacing protocols. The VT could not be entrained, but could be suppressed by rapid RV pacing (pacing cycle lengths within 340–370 ms) and by 12 mg adenosine intravenous bolus injection. Endocardial 3D-electroanatomic mapping showed a focal VT with the earliest activation site at the posteroinferior RV septum. Radiofrequency catheter ablation (RFCA) using a non-irrigated Navistar Carto ablation catheter (Biosense Webster, Diamond Bar, CA, USA) was applied at the posteroinferior septum to terminate the VT successfully (red dots; Figure 2C). However, a second VT (VT-2; cycle length: 410 ms; Figure 2B) with a slightly different QRS morphology occurred after VT-1 ablation. The earliest activation site of VT-2 was close to the first successful ablation site (blue dots; Figure 2C). As shown by voltage map (Figure 2D) and endocardial electrical recordings (Figure 2E), the earliest activation sites of VT-1 and VT-2 were located in low voltage zones. VT-2 was successfully eliminated by a second RFCA procedure. No VT was inducible thereafter and no recurrence of VT was documented by follow-up Holter and clinical visits.

Dermatomyositis rarely involves myocardial muscles but has been shown to be associated with arrhythmias, frequently manifesting as conduction disorders rather than rhythm disturbances.[1,2] Although ventricular ectopies are frequent, VT in patients with dermatomyositis is rare.[3,4] The underlying pathophysiology is either by direct lesions of the conduction system or by myositis with contraction band necrosis and/or focal myocardial fibrosis. Dermatomyositis-related ventricular tachyarrhythmia is usually bradycardia-dependent, which was not seen in our patient.[5,6]

Would patients with dermatomyositis presenting with VT benefit from steroids, antiarrhythmic drugs, RFCA or implantable cardioverter defibrillator (ICD) therapy? Generally, optimal therapy of ventricular arrhythmias associated with systemic inflammatory disorders like dermatomyositis is not well established. Although steroids improve overall mortality, there are reported cases of VT exacerbation with steroids due to attenuation of inflammatory responses with subsequent fibrosis.[7] In this patient who is already on maintenance corticosteroids, increasing the dose or adding an immunosuppressive agent is an option. However, if the arrhythmia mechanism is not due to disease activity, these drugs may be harmful rather than beneficial. VT in the setting of structural heart disease with a reduced LVEF is commonly treated by ICD implantation.[8] Retrospective trial

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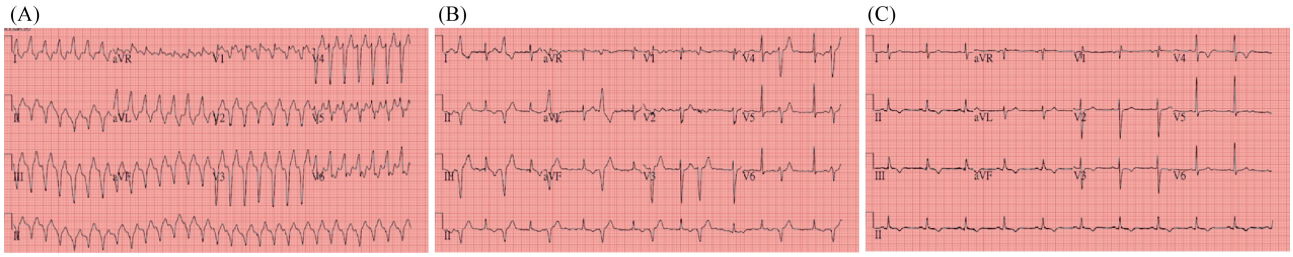


**Figure 1. Twelve-lead electrocardiograms.** (A): twelve-lead electrocardiogram during ventricular tachycardia; (B): ventricular premature beats and atrial fibrillation; (C): normal sinus rhythm. The morphologies of ventricular premature beat and ventricular tachycardia were quite similar.

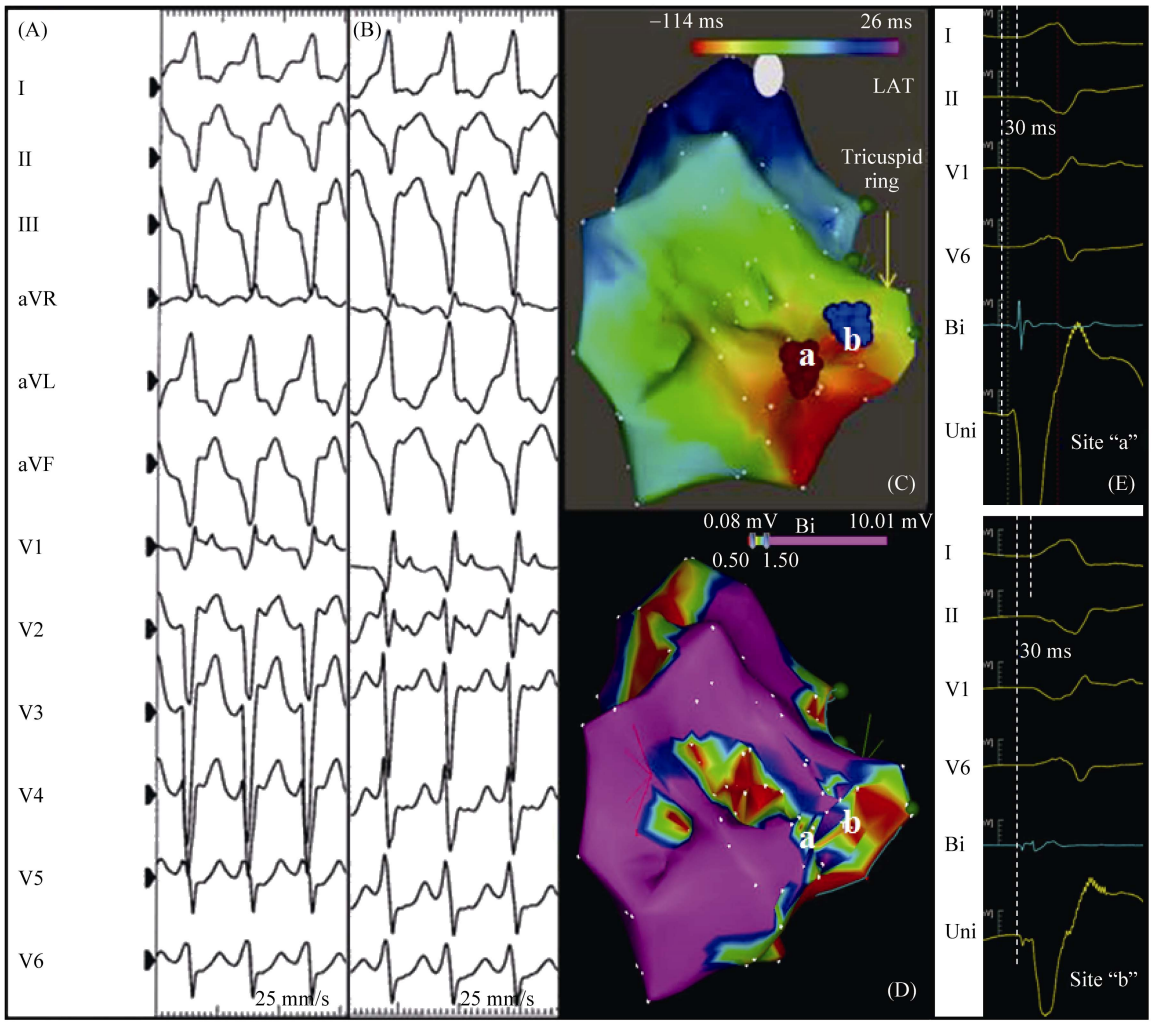


**Figure 2. Endocardial 3D-electroanatomic mapping and ablation.** (A): electrocardiogram of VT-1; (B): electrocardiogram of VT-2; (C): activation map during VT-1. The earliest activation site is located at the posteroinferior right ventricular septum where successful ablation was applied (red dots). The blue dots indicate the successful ablation sites of VT-2 ablation. Note that the blue dots are close to the red dots. Yellow arrow indicates tricuspid ring; (D): voltage map of the RV showing areas of low voltage areas involving the earliest activation sites of VT-1 and VT-2; (E): the earliest endocardial electrical signals at sites "a" and "b" (labeled in panel C) during VT-1 and VT-2, respectively, which preceded QRS by 30 ms. Bi: bipolar; LAT: local activation time; Uni: unipolar; VT: ventricular tachycardia.

however shows RFCA could be a primary effective management for VT in such patients without subsequent implantation of ICD.[9] The VTs in our case were isoproterenol-triggered, adenosine-suppressed, and focally originating from the posteroinferior RV septal area with the most likely underlying mechanism of abnormal automaticity. Therefore, it was

expected that elimination of the triggers can cure the VT. Our patient received a successful 3D-electroanatomical mapping-guided RFCA at the earliest activation sites of the focal VTs with no recurrence, which highlights the role of electrophysiological study and RFCA in patients with dermatomyositis presenting with VT.

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