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Case Report

Electrical storm in dilated cardiomyopathy treated using epicardial radiofrequency ablation as a first line therapy

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ABSTRACT

We report a patient with non-ischemic dilated cardiomyopathy and low left ventricular systolic function (28%) presenting with an electrical storm originated in epicardial scar and ablated by radiofrequency. This case report suggests that a strategy of epicardial catheter ablation is reasonable for the patient presenting with electrical storm related to structural disease with a low left ventricular ejection fraction.

1. Introduction

Patients with non-ischemic dilated cardiomyopathy (DCM) are at high risk for sudden cardiac death due to sustained ventricular tachycardia (VT) or ventricular fibrillation (VF).[1] Randomized trials have proven the benefit of an implantable cardioverter-defibrillator (ICD) as compared with anti-arrhythmic drug therapy, and current guidelines recommend ICD therapy in both primary and secondary prevention.[2–7] Although ICDs improve overall survival, they do not eliminate the arrythmogenic substrate. Despite the presence of an ICD, the occurrence of a ventricular arrhythmia storm (≥3 hemodynamically destabilizing VTs in a 24-h period or the detection of ≥3 VTs in a 24-h period in ICD patients) may still lead to elevated mortality.[8] According to the above-mentioned definition, the incidence of electrical storm (ES) ranges from 4%, over a 20.6-month period of follow-up in those studies in which ICD implantation was carried out for primary prevention, to 20% over a 31-month period of follow-up in secondary prevention ICD recipients.[8]

In patients experiencing ES, the key intervention is reduction of the sympathetic tone using beta-blockers, frequently combined with benzodiazepines; amiodarone intravenous infusion is also recommended while class I antiarrhythmic drugs are usually unsuccessful.[8] The non-pharmacological therapeutic option for the recurrence of VT or VF is transcatheter ablation of ventricular arrhythmogenic foci.[9–12]

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Although radiofrequency catheter ablation has an established role in the treatment of recurrent VT, its therapeutic role in patients with ES has been investigated only in selected populations with implanted ICDs. The current guidelines suggest that VT ablation intervention be used as an adjunctive therapy to ICD while radiofrequency ablation strategy alone has never been assessed in patients with structural heart disease and a low ejection fraction.

2. Case report

A 55-year-old man was admitted to the hospital due to a syncope episode preceded by palpitation. The patient had no cardiovascular risk factors, no familial history of sudden cardiac death, and no previous history of cardiovascular disorders, including syncope or palpitation episodes. During transport, the patient developed VT with hypotension and was defibrillated twice with 200 J from a biphasic defibrillator with standard anterior and lateral placement (one pad right parasternal and the second at the cardiac apex) obtaining temporary recovery of sinus rhythm with subsequent numerous subentrant episodes of VT. An IV infusion of amiodarone was immediately started and another 4 DC shocks were administered. While in the coronary care unit, by utilizing a conventional 12 lead surface electrocardiogram (ECG), sustained, wide complex tachycardia with a rate of 234 beats per minute with right bundle brunch and right-axis deviation morphology, and a pseudodelta wave in precordial leads were recorded. The tachycardia morphology was consistent with the diagnosis of VT originating from the left ventricle. The intravenous infusion of amiodarone, lidocaine, beta blockers, and magnesium sulfate was carried out with no resolution of the arrhythmia. After deep sedation and orotracheal intubation, sinus rhythm was restored by means of electrical cardioversion. Written informed consent for the cardiac catheterization and eventual electrophysiological study was obtained from the family of the patient. The patient underwent urgent cardiac catheterization, and coronary angiogram documented normal coronary vessels and diffuse left ventricular (LV) hypokinesia (left ventricular ejection fraction (LVEF) = 28%). The patient was referred for emergency electrophysiological study. Endocardial mapping did not identify any low-voltage areas suggestive of an arrhythmia focus. As the ECG-guided morphological analysis of the arrhythmia was coherent with an epicardial origin (pseudodelta wave) of the arrhythmia, a subxiphoid approach was undertaken to achieve pericardial access as described by Sosa et al.[13] Epicardial LV mapping during sinus rhythm revealed a 1.6 cm^2 scar at the posterolateral region of the basal segment of the LV epicardium with a voltage of 0.6 mV. After VT was induced (QRS morphology is identical to spontaneous ventricular arrhythmia), this isolated delayed potential became mid-diastolic and presystolic (180 ms before QRS complexes) (Fig. 1). One pulse radiofrequency current at this site interrupted VT after 4.5 s (maximum power, 45 W; maximum temperature, 42 °C). Several additional radiofrequency applications were placed in close proximity adjacent to this site on local abnormal ventricular activities (LAVA) potentials (Fig. 2). Coronary angiography was not performed before radiofrequency delivery because there was low possibility of damage of main coronary artery vessel.

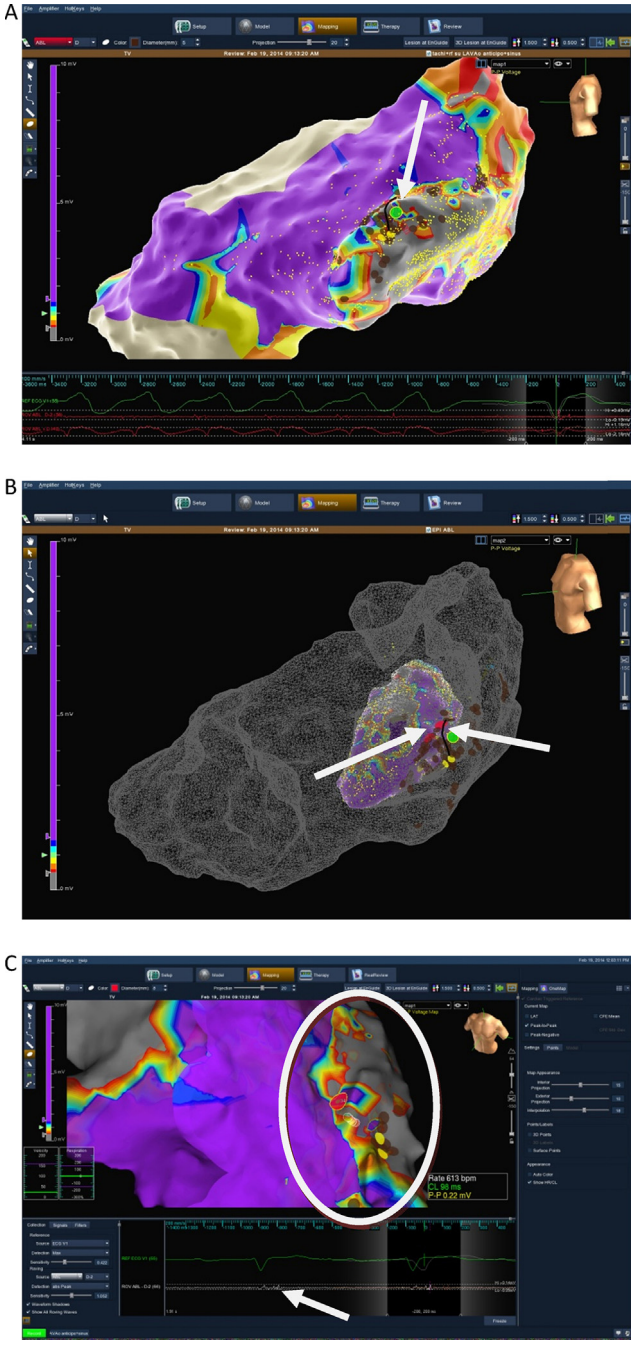

Fig. 1 – Electroanatomical mapping. Left ventricular posterior views of epicardial mapping (A) and endocardial/epicardial map (B). The earliest epicardial activation site during ventricular tachycardia (A, marked by a white arrow) shows the site where VT has been interrupted, and brown tags (A and C) indicate the ablation site on the LAVA potentials. A white circle (C) indicates voltage mapping during sinus rhythm (C) with isolated delayed potential around the ablation area.

After the ablations were completed, VT could no longer be provoked.

The findings are consistent with a small, epicardial arrhythmogenetic scar area, possibly related to idiopathic

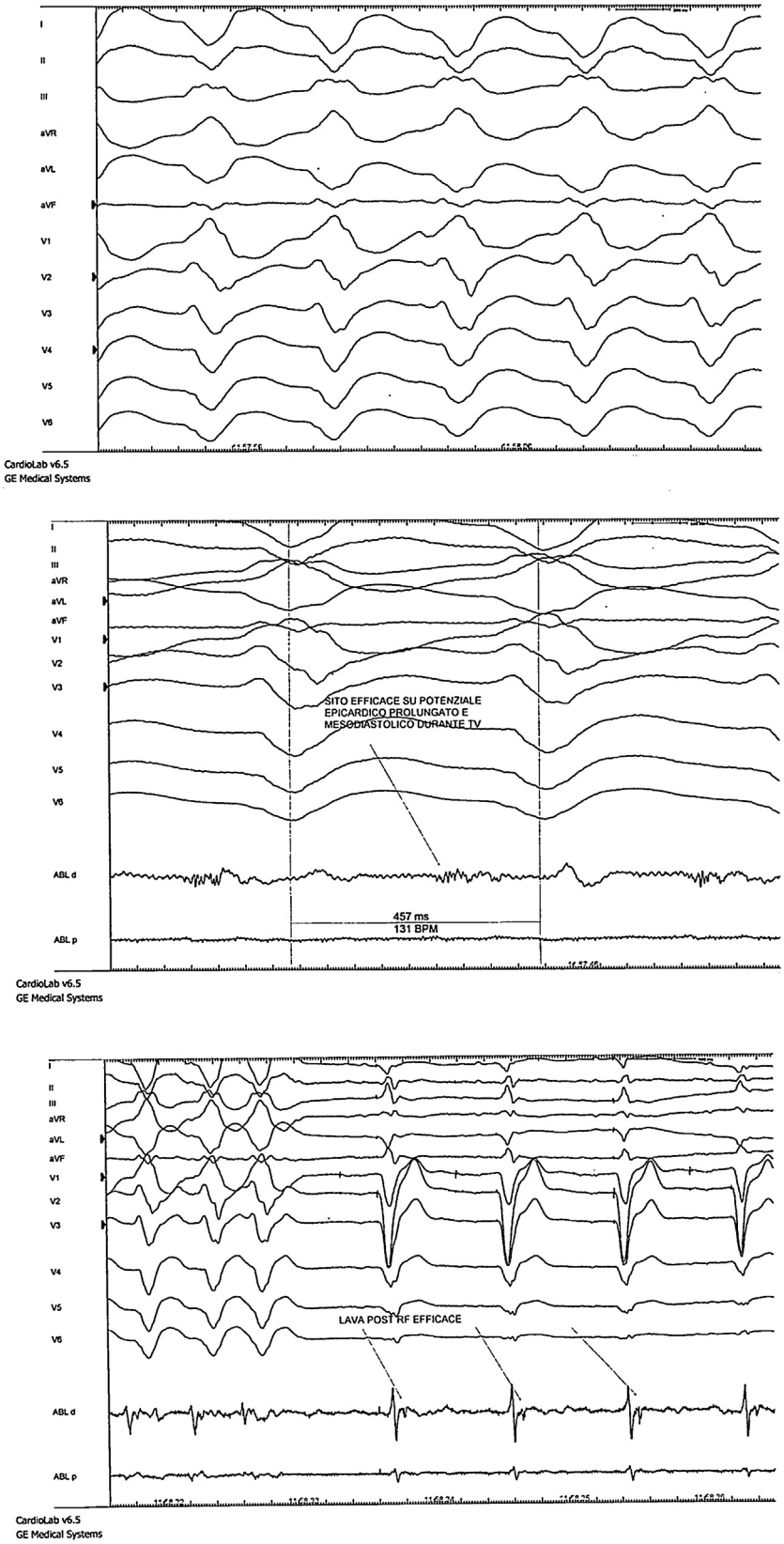

Fig. 2 – A twelve-lead ECG during ventricular tachycardia. Note the right bundle-branch morphology of the QRS complex (A). Findings during epicardial mapping during ventricular tachycardia show the presence of presystolic or mid-diastolic fragmentation (B). Panel (C) shows that the isolated potential in the epicardium occurs approximately 180 ms before the QRS complex during VT. Ablation at the epicardial site terminates ventricular tachycardia. In this area, during sinus rhythm it is possible to observe local abnormal ventricular activities (LAVA) potentials (white arrows). LAVA was defined as electrograms

cardiomyopathy. Endomyocardial biopsy had excluded inflammatory and non-inflammatory causes of idiopathic cardiomyopathy. Since we did not perform cardiac magnetic resonance, we cannot exclude a priori areas of myocardial inflammation or fibrosis near the site of epicardial ablation. During follow-up, no further ventricular arrhythmias were registered over a period of 30 days after ablation. Echocardiography showed severe LV systolic dysfunction with a LV ejection fraction of 28%. A dual chamber ICD was implanted for secondary prevention. At a 24-month follow-up, no further episodes of VT were recorded. No antiarrhythmic therapy was administered following the ablation.

3. Discussion

This case report suggests that a strategy of epicardial catheter ablation is reasonable for a patient presenting with ES related to structural disease having a low LVEF (LVEF = 28%).

Randomized controlled trials support the use of ICDs for the secondary prevention of sudden cardiac death. In patients with a history of cardiac arrest, an ICD is associated with clinically and statistically significant reductions in sudden death and total mortality as compared with antiarrhythmic drug therapy.[1]

Catheter ablation (CA) has a well-established role in managing recurrent VT in order to reduce shocks in patients with ICDs. Many studies have demonstrated that, after CA, the frequency of VT was reduced by 50–67% at a 6-month follow-up. Della Bella et al. have demonstrated that the elevated success rate of CA is associated with a lower arrhythmia recurrence rate, also due to the systematic implementation of an ablation strategy, which focused on endocardial-epicardial substrate modification, independent of VT inducibility and tolerance. CA also improved the survival rate by 18–25% in a 1-year follow-up period.[12]

Transthoracic epicardial radiofrequency has been demonstrated to be a safe, feasible, and effective alternative in patients undergoing unsuccessful endocardial ablation.[13] Generally, an epicardial approach was performed in 17–19% of VT ablation procedures ranging from 6% in normal hearts to 16% for ischemic cardiomyopathy, 35% for DCM, and 41% for arrhythmogenic right ventricular cardiomyopathy.[14] However, there are no reports in the literature regarding this procedure in patients before ICD implantation. Recently, Maury et al. reported that CA with a deferral of ICD implantation is reasonable for a patient presenting with well-tolerated sustained monomorphic VT related to structural disease having an LVEF $>$ 30%.[15] However, this ablation strategy has not been reported in patients with hemodynamically non-tolerated VTs and with severe altered left ventricular function as in our case report. We decided to implant an ICD after CA because a poor left ventricular ejection fraction and ES are independent predictors of VT recurrence, even if our patient had no arrhythmias in a 24-month follow up. Patients with VT ablation are more likely to remain at risk of sudden death and an ICD is usually warranted before hospital discharge.[1]

4. Conclusion

To our knowledge, this is the first case reporting the efficacy of urgent VT ablation in a patient presenting with hemodynamically non-tolerated ES. Furthermore, we considered multiple factors in making our decision regarding this epicardial approach; this included ECG analysis of the clinical VT for features suggesting an epicardial source, history of previous failed endocardial ablation, and the presence of ES.

Conflicts of interest

The authors declare that they have no conflicts of interest.

Ethical standards

This is a case report.

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from poorly coupled surviving myocardial fibers with the following features: (1) sharp, high-frequency ventricular potentials distinct from the far-field ventricular electrogram; (2) occurring anytime during or, more frequently, after the far-field ventricular electrogram during sinus rhythm; and (3) sometimes displaying double or multiple high-frequency signals separated by very low-amplitude signals or an isoelectric interval. The complete elimination of LAVA would lead to a reduction in arrhythmia-free survival.

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