Extreme Electrical Storm in a Patient with an Implantable Cardioverter Defibrillator

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The implantable cardioverter defibrillator is highly effective in primary and secondary prevention of life-threatening ventricular arrhythmias. About 40–70% of patients with an ICD will receive appropriate therapy following implantation [1]. Most of the time these therapies are limited to a small number of shocks or anti-tachycardia pacing. However, some patients receive multiple treatments over a short period. This situation has been defined as an "electrical storm" which consists of three or more distinct episodes of ventricular tachycardia and/or ventricular fibrillation within a 24 hour period. ES is a life-threatening situation that adversely affects the short and long-term prognosis and is a clinical and therapeutic challenge [2].

We report the case of a patient with ischemic cardiomyopathy and severe ES caused by repeated short coupled premature ventricular complexes that triggered polymorphic VT and VF, which were resistant to anti-arrhythmic drugs and complete percutaneous coronary revascularization. Successful treatment was finally achieved with rapid pacing and extensive catheter ablation.

ICD = implantable cardioverter defibrillator
ES = electrical storm
VT = ventricular tachycardia
VF = ventricular fibrillation

PATIENT DESCRIPTION

A 59 year old man with ischemic cardiomyopathy, status post-coronary artery bypass graft surgery and an ICD (Virtuoso® DR, Medtronic, Inc. USA) for primary prevention of sudden cardiac death 2 years earlier was admitted to the intensive cardiac care unit due to non-ST elevation myocardial infarction, acute heart failure and cardiogenic shock. Left ventricular ejection fraction was estimated to be 15%. He was intubated and urgently transferred to the cardiac catheterization lab where an intra-aortic balloon pump was inserted, followed by coronary angiography that demonstrated severe native three-vessel disease, a patent left internal mammary graft to the left anterior descending artery, and a patent vein graft to a marginal branch. Percutaneous coronary intervention to an occluded diagonal artery was performed.

On his seventh day of hospitalization he developed severe ES that started with rapid atrial fibrillation. Subsequent episodes usually began with short coupled PVC of right bundle branch morphology [Figure A] that triggered PMVT and were terminated by appropriate shocks from his ICD. Attempts to reduce the frequency of ventricular tachyarrhythmias with anti-arrhythmic drugs such as amiodarone and procainamide were unsuccessful. The only anti-arrhythmic drug that seemed to reduce the number of ventricular tachyarrhythmic events was lidocaine, which was administered as a continuous infusion. Overall, the patient received a total of 104 shocks from the device over a 4 day period until the battery of the device reached its end of service and electric shocks had to be administered by an external defibrillator. In an attempt to reduce the ischemic burden, the patient underwent a second successful percutaneous coronary intervention to the right coronary artery.

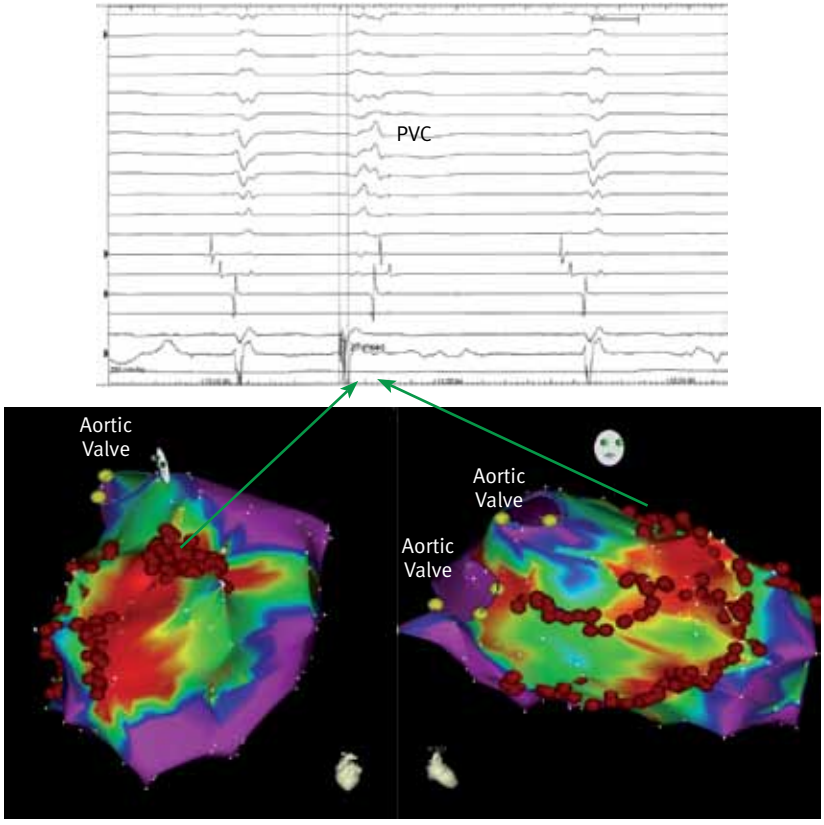
PVC = premature ventricular complexes
PMVT = polymorphic ventricular tachycardia

Although the ICD reached its end of service and the device could no longer deliver shocks, the pacing mechanism of the device was still functioning and reprogrammable. The managed ventricular pacing (MVP®, Medtronic) mode was turned off to eliminate the possibility for long-short episodes and the device was programmed to DDD mode 90/110 beats per minute lower/upper rate, respectively, with a long atrioventricular delay to prevent short coupled PVCs and reduce the chance of right ventricular pacing. The combination of rapid atrial pacing with the administration of intravenous lidocaine seemed to reduce, but did not eliminate, the episodes of ventricular tachyarrhythmias.

As a final recourse, the patient was taken to the electrophysiology lab for electrophysiology study and catheter ablation. Administration of lidocaine was stopped and the rate of pacing was slowed. Since we could not map the ventricular tachycardia, the strategy of the electrophysiology study was to create a substrate mapping of the left ventricle using 3-D electroanatomic voltage mapping (CARTO 3TM, Biosense Webster, Diamond Bar, CA, USA). Scar areas were defined by local voltage < 0.48 mV. Healthy tissue was defined as local voltage > 1.5 mV.

Mapping the site where the PVC originates was facilitated by an early electrocardiogram that preceded the QRS onset during PVC. We also created lines of ablation at the border of the scars and through

[A] Voltage map of the left ventricle using the CARTO 3 system, in left lateral and right anterior oblique views. The red colored areas represent the scar zone, the yellow-green the borderline zone, and purple indicates healthy tissue. The red dots show ablation applications. The endocardial map illustrates a large anterior-lateral-septal infarct. The arrows indicate the origin of the initiating PVC corresponding to the early electrocardiogram that preceded the QRS onset by 37 msec during the PVC. We also created lines of ablation at the border of the scars and through areas that could create re-entry tachycardia



areas that could create re-entry tachycardia [3-5] [Figure A].

At the end of the ablation procedure we recorded stable sinus rhythm, without PVCs or ventricular arrhythmias. We performed a modest programmed extra-stimulation from the right ventricular apex without triggering any ventricular tachyarrhythmias and stopped the procedure at that point. The ICD was reprogrammed back to high pacing rate and the patient was placed on mexiletine 200 mg orally four times a day and metoprolol. Following the catheter ablation, the patient underwent prolonged intensive rehabilitation without any ventricular arrhythmic events. After a few weeks, his ICD was replaced, gradually lowering the lower pacing rate to 70 beats per minute, and mexiletine and metoprolol were continued.

After 2 months of hospitalization the patient was gradually weaned from the ventilator and discharged to an inpatient rehabilitation facility. At 2 months follow-up after the implantation of his new ICD, there was no evidence of recurrent ventricular arrhythmias. Transthoracic echocardiography showed some improvement in left ventricular function.

COMMENT

We report the case of a severe unrelenting electrical storm in a patient with ischemic cardiomyopathy. Recurrent episodes of PMVT and VF were initiated by short coupled PVCs that seemed to be monomorphic.

Our comprehensive approach to this difficult situation included administering anti-arrhythmic drugs, maximal achievable revascularization, rapid pacing to reduce the possibility of early PVCs and, finally, extensive catheter ablation as a rescue procedure. The outcome was remarkable.

The catheter ablation was based on substrate-voltage mapping since we were unable to map during VT. We defined dense scar areas with bipolar potentials < 0.48 mV, drawing lines of ablation on the border of the dense scar tissue and through areas that could create re-entry tachycardia. We also looked for areas with early potentials during PVC and pace mapping compared to the 12 lead electrocardiogram morphology of the PVC that initiated the PMVT. We believe that the success of this ablation procedure was mainly due to targeting the origin of this PVC.

The scar area shown in the voltage map does not explain the patient's low cardiac function. A possible explanation is the deleterious effect of 104 consecutive shocks that could further impair the contractile properties of the left ventricle. Hibernating or stunned myocardium post-myocardial infarction is another possibility. Repeat echocardiography did show some improvement in ventricular function.

In conclusion, this case supports the use of catheter ablation as a life-saving rescue procedure in an extreme situation of refractory VT or VF, effectively abolishing the electrical storm.

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