Extreme Electrical Storm in a Patient with an Implantable Cardioverter Defibrillator

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**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 cardic 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 tachycardias with anti-arrhythmic drugs such as amiodarone and procainamide were unsuccessful. The only anti-arrhythmic drug that seemed to reduce the number of ventricular tachyarrrhythmias 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.

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...
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 was gradually weaned from the ventilator and discharged to an inpatient rehabilitation facility. 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.

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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References

Capsule
Parasitic co-infections challenge the immune system – different pathogens often require different flavors of immune responses for their elimination. Two teams studied what happens when parasitic worms and viruses infect mice at the same time. Reese et al. (Science 2014; 345: 73) found that parasite co-infection woke up a dormant virus. Osborne et al. (Science p. 517) found that mice already infected with parasitic worms were worse at fighting off viruses. In both cases, worms skewed the immune response so that the immune cells and the molecules they secreted created an environment favorable for the worm at the expense of antiviral immunity.
Eitan Israeli

Capsule
The long and short of hair growth
The length of your eyelashes probably differs from the length of the hair on your head – and unlike your hair, your eyelashes can never reach your shoulders. What controls how long hair can get? To find out, Higgins et al. studied people with a rare disorder called familial trichomegaly, who have very long eyelashes and longer hair on the arms. They found that these people had a mutation in the gene that encodes fibroblast growth factor 5 (FGF5). When human hair follicles produce FGF5, they stop growing hair. Targeting FGF5 could potentially control the growth and rest phases of hair follicles, preventing unwanted hair from sprouting or growing longer lashes and locks.
Proc Natl Acad Sci USA 2014;10.1073/pnas.1402862111
Eitan Israeli

Capsule
Mycobacterium make not-so-painful ulcers
Buruli ulcer disease causes extensive skin lesions and can be deadly, but the lesions themselves don’t hurt, which can stop patients from seeking the appropriate care. The pathogen Mycobacterium ulcerans causes Buruli ulcers and also alleviates the pain. Although many scientists studying this disease thought the pathogen caused nerve damage that blocked the pain, Marion et al. show that the mycobacteria produce the mycolactone toxin, which causes analgesia by blocking the function of pain-responsive nerves. The findings could potentially help researchers develop a whole new class of painkillers.
Cell 2014;157: 1565
Eitan Israeli

Capsule
A neuropeptide kills patient’s motivation
Chronic pain is not only extremely disturbing and unpleasant, it can also make people depressed and demotivated. What causes these effects? Schwartz and co-researchers discovered that chronic pain causes changes in the way a neuropeptide called galanin affects certain neurons in a brain region called the nucleus accumbens. Galanin influences a variety of behaviors, including feeding and certain aspects of pain. In this case, it depresses synaptic transmission at specific excitatory synapses. It does so, in part, by changing the ratio of subunits of an important receptor protein.
Science 2014; 345: 535
Eitan Israeli