

Non-invasive Management of High Defibrillation Threshold in Patient with Implantable Cardioverter-Defibrillator

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Case Report

We report a case of 81 year-old man with implantable cardioverter-defibrillator (ICD) admitted to our institution after repetitive DC-shocks of the device. The patient was followed by our Heart Failure Center because he had non-ischemic dilated cardiomyopathy (ejection fraction of the left ventricle was 30%, end diastolic volume was 176 ml). 13 months before, he was admitted to the Cardiology Department because he developed a complete atrioventricular block and he received in primary prevention a dual chamber ICD system (Medtronic Protecta DR; dual coil lead 6944 Sprint 4 Medtronic for right ventricle and 5076 Capsure fix Medtronic for the atrium). Defibrillation threshold (DFT) was tested at the implant session and the ICD successfully terminated induced ventricular fibrillation with a 21-Joules shock in 2 consecutive tests. The vector configuration of shock was distal coil (RV) as cathode vs. device can and proximal coil (SVC) as anode.

Thereafter, the patient developed relapsing pocket infection requiring multiple surgical revisions, leading to a rotation flap and to an "over breast" pocket which was performed with the help of a plastic surgeon (Figure 1, Panel A). He also developed severe renal function impairment (creatinine level 7.62 mg/dl), requiring weekly hemodialysis from a permanent central venous catheter positioned in the right jugular vein as it was not possible to perform an arterio-venous fistula (Figure 1, Panel A). For these reasons, despite the relapsing infections, it was impossible a new implant contralateral.

All of this went on for approximately 12 months, until after intense fatigue in the garden the patient had a syncope, which was promptly interrupted by several shocks from the ICD, resulting in an emergency alert. The ECG performed at patient's house (only 1 derivation) revealed repetitive episodes of ventricular fibrillation (VF), which continued causing several consecutive ICD's intervention. He was treated at home with 12 mg of midazolam iv and admitted to our cardiologic intensive care unit. His vital signs were: blood pressure 138/70 mmHg; oxygen saturation 98% on room air; heart rate 70 bpm. Physical examination was unremarkable; with no signs of cardiac failure. Acid-base and electrolytic status (in particular calcium and potassium) were normal. The ECG showed sinus rhythm and VDD stimulation with complete AV block. The ventricular complex had right bundle branch block morphology (Figure 1, Panel B) despite the echocardiogram showed the tip of the catheter in the distal apex of severely dilated right ventricle. Most likely the tip of the right ventricular lead projected to the left side due to the severe right ventricular dilatation.

The following ICD control revealed 72 DC shocks (47 ineffective)

delivered in a few hours by the device for 32 ventricular arrhythmic episodes interpreted by the device as VF. Nearly 50% of arrhythmic episodes were not visible for memory saturation of device. The analysis of latest episodes showed that the ICD correctly detected the arrhythmias but failed to interrupt it despite a high energy shock. The first non-committed shock failed to interrupt VF, instead it caused a cycle length with different morphology modification of the arrhythmia and his spontaneous termination mean time that the device was recharging for a second committed shock that, delivered during sinus rhythm induced the arrhythmia again (Figure 1, Panel C) with a sequence of repetitive shock. The other findings of the ICD were normal: battery voltage 2.7 V; atrial sensing 1 mV; ventricular sensing 10 mV; atrial threshold 0.5 V with 0.4 ms and ventricular threshold 0.5V with 0.4 ms, normal values of lead impedance and shock impedance.

ECG monitoring was started, as well as infusion of amiodarone and antero-posterior external defibrillator patches were applied to the chest as safety backups.

It is infrequent that high energy shocks for termination of ventricular arrhythmias are ineffective, unless ICD dysfunction. We proved the absence of any detectable dysfunction of our device, i.e. normal sensing and pacing parameters both atrial and ventricular, acceptable shock impedance. Several other factors may have played a role in increase DFT, however, many could be excluded such as clinical or metabolic derangements, pneumothorax or modification in intrathoracic impedance, and use of antiarrhythmic agents. The location of the generator and the shock vector can also affect the DFT, as uniform distribution of energy encompassing the entire left ventricle is crucial [1]. Shocks from an ICD are delivered from the coils of the lead that reach the generator by traversing through a critical portion of the myocardium enough to break the global wave of fibrillation. We believe that the shock's failure in the case described was caused by both poor can location (the can in supramammarian region was very close to RV coil) and shock vector (coil RV vs. can) involving insufficient mass of the left ventricle to stop the fibrillatory activation fronts.

Management of high DFT may require both non invasive and/or invasive strategies. Mainigi et al. [2] suggested a management algorithm for patients with a high defibrillation threshold and failure of the initial and maximal output shocks.

In our patient, the venography demonstrated left venous access patency, still but we decided a conservative approach considered all the concomitant comorbidity active. There was a chance to avoid re-intervention: altering the shock vector. Accordingly, we programmed

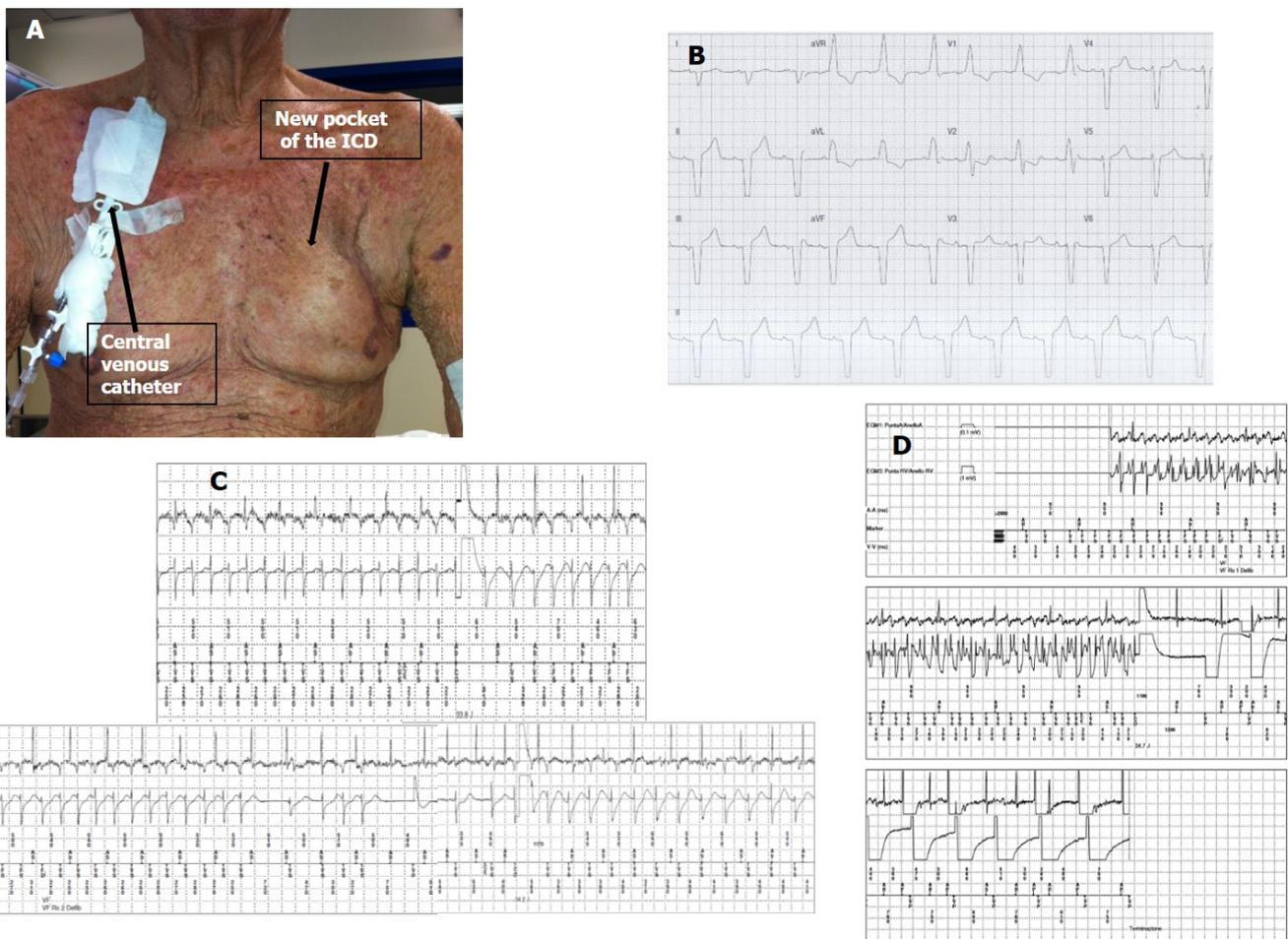


Figure 1

Panel A: An “over breast” ICD pocket is visible in the right side. A permanent central venous catheter positioned in the right jugular vein is visible in the left side.

Panel B: 12 lead ECG of VDD pacing with RBBB morphology.

Panel C: In the upper part the first non-committed shock failed to interrupt VF. In the lower part cycle length and morphology modification of arrhythmia and his spontaneous termination whether the device was charging capacitors for a second committed shock that, delivered during sinus rhythm conditioned the arrhythmia’s restart.

Panel D: During DFT-ventricular fibrillation (VF) induced with erogation of direct current: the ICD successfully terminated induced VF with a 25-Joules shock.

shocking circuit with current pathway from the RV coil to the slightly more proximal coil (SVC coil) excluding can from the circuit. The patient was then taken to the electrophysiologic laboratory and, previous sedation with midazolam and fentanyl, we induced a VF by delivering direct continuous current. The ICD successfully terminated induced ventricular fibrillation with a 25-Joules shock, in 2 consecutive tests (Figure 1, Panel D).

At the discharge the patient was asymptomatic, and remained on amiodarone per os. 10 months later, the patient had another episode of ventricular fibrillation during sleep correctly detected and terminated with 35-Joules DC-shock form ICD.

Conclusion

In conclusion, inefficient shock from ICD is the result of a complex interplay between molecular, electrical, mechanical, anatomical, and pharmacological factors. Although there have been reports suggesting that DFT testing does not predict survival or improve clinical outcomes in ICD recipients, there is no clear consensus about steering away from

this convention [3]. In ICD recipients who undergo device’s reposition in alternative and non conventional sites DFT should be taken into consideration as delivered shock could not involve critical mass and therefore could be ineffective in solving ventricular arrhythmias. Changing shock vector may safely and non-invasively help to manage this problem.

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