IMPLANTABLE DEFIBRILLATION THERAPY

COMPLEX CASE STUDY

Ineffective Shock Therapy in a Subcutaneous Implantable Cardioverter-Defibrillator Patient with Hypertrophic Cardiomyopathy

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ABSTRACT. We report the case of an 18-year-old hypertrophic cardiomyopathy female with a subcutaneous implantable cardioverter-defibrillator (S-ICD) for primary prevention who presented with monomorphic ventricular tachycardia and five ineffective 80-J shocks. We have examined the patient case notes, imaging and electrophysiology study data, and traces obtained from S-ICD device interrogation. This is a rare example of device failure in an otherwise safe and effective system as illustrated by EFFORTLESS and IDE data.

KEYWORDS. hypertrophic cardiomyopathy, subcutaneous implantable cardioverter-defibrillator, sudden cardiac death.

Introduction

The subcutaneous implantable cardioverter-defibrillator (S-ICD) is a safe and effective option for the primary and secondary prevention of sudden cardiac death (SCD). Follow-up data from the EFFORTLESS trial and IDE registry support this, highlighting equivalent first and multiple shock efficacies when compared with transvenous (TV)-ICDs. Data from patients with hypertrophic cardiomyopathy (HCM) are lacking, although a subgroup analysis of the aforementioned studies has demonstrated effectiveness in the HCM population.1

Case report

We report the case of an 18-year-old female with HCM diagnosed on familial screening and an S-ICD for primary prevention. This device was chosen after discussion of the relative risks and benefits between the S-ICD and TV-ICD. It was implanted 1 year prior to admission after an exercise test-induced episode of non-sustained polymorphic ventricular tachycardia (VT). Her on-table defibrillation threshold (DFT) testing at implant was effective at 65 J with normal polarity. Her only medication was bisoprolol 1.25 mg once daily.

She presented with an episode of non-hemodynamically compromising monomorphic VT during a brisk walk and received five ineffective 80-J shocks. This VT fell within the ventricular fibrillation (VF) zone of her device programmed at a rate of 230, with a conditional VT-1 zone set at a rate of 200. Her device was interrogated and showed appropriate arrhythmia detection and shock delivery (Figure 1) with equivalent impedances to implant. The rhythm spontaneously converted shortly after the fifth shock.

Serum electrolytes were normal and chest radiography confirmed that the device position was unchanged from implant (Figure 2). Echocardiography showed eccentric mid-apical left ventricular hypertrophy and some features suggestive of non-compaction. No significant intraventricular gradient was noted both at rest and during the Valsalva maneuver. Electrophysiological study demonstrated dual atrioventricular node physiology, and no aberration or inducible arrhythmias including VT up to Stage XII of the Wellens protocol.

It was felt, given the ineffectiveness of the S-ICD in terminating this arrhythmia episode despite appropriate
device function, that it should be replaced with a TV-ICD in order to offer the option of ATP to terminate further VT as well as deliver shock therapies. This was undertaken without complication.

The above account describes a rare and unusual outcome for an otherwise safe and effective system in the prevention of SCD. Although regarded as a “device failure” in this context, it is important to note that studies looking at shock efficacy focus on shock energy and number, and not the temporal relationship between shock delivery and cardioversion. In fact, the only study we could find looking at this was in cardiac arrest patients with VF, quoting a definition of success as defibrillation in less than 5 s from shock delivery.2 From the pooled data analysis of EFFORTLESS and IDE, 60 episodes of monomorphic VT occurred in 44 patients, of which 55 were converted with the first shock and all converted with up to five shocks, proving excellent efficacy in this cohort.3 The only failure was in a patient with polymorphic VT who had spontaneous arrhythmia termination following five consecutive S-ICD shocks. In both cases, there was appropriate arrhythmia detection and therapy delivery, the main difference being the ability of the transvenous system to perform antitachycardia pacing (ATP) that terminated the arrhythmia in the latter. Indeed, a recent study identified a higher failure rate for TV-ICD shocks in patients with sustained monomorphic VT and also HCM cases.5 Although ATP provides a useful and less painful adjunct in the treatment of monomorphic VT, it does expose patients to the inherent long-term lead complications associated with a TV-ICD. The risk–benefit ratio of both needs to be weighed carefully and those patients with a high burden of monomorphic VT may be better suited to a transvenous system delivering ATP, although this is yet to be tested in a randomized controlled trial with matched sustained rate duration and VT rate thresholds.

References