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CASE REPORT

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Subcutaneous ICD implantation in a patient with hypertrophic cardiomyopathy after transvenous ICD failure: A case report

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Abstract

We describe the case of a patient with hypertrophic cardiomyopathy who experienced the failure of a transvenous implantable cardioverter defibrillator (T-ICD) lead and the following inability of a second T-ICD to convert a ventricular fibrillation. A subcutaneous ICD (S-ICD) was finally implanted and was effective at defibrillation test.

KEYWORDS
complications, defibrillation threshold test, hypertrophic cardiomyopathy, ICD replacement, subcutaneous defibrillator

1 | INTRODUCTION

In hypertrophic cardiomyopathy (HCM) patients, increased myocardial mass may make it difficult to interrupt ventricular arrhythmias by implantable cardioverter defibrillator (ICD). We describe the case of a patient with HCM who experienced the failure of a transvenous ICD (T-ICD) lead and the following inability of a second T-ICD to convert a ventricular fibrillation. A subcutaneous ICD (S-ICD) was finally implanted and was effective at defibrillation test (DFT).

2 | CASE REPORT

A 20-year-old male patient was admitted to the emergency room of our center after ICD shock delivery. His past medical history included symptomatic HCM, a previous septal myectomy performed at the age of 15 and a previous episode of resuscitated ventricular fibrillation at the age of 18. He was therefore treated in another hospital with the implantation of a single-chamber T-ICD in the right pectoral side, for secondary prevention. The right side was preferred due to tortuosity of the left subclavian vein. DFT was not performed according to local clinical practice. Cardiac magnetic resonance was not performed at the time of implantation.

Present echocardiographic findings were left ventricular ejection fraction 36%, end-diastolic diameter 36 mm, end-systolic diameter 23 mm, and ventricular septal thickness 39 mm with pseudonormal filling pattern.

At the time of admission, the patient was on bisoprolol therapy (2.5 mg/die) and no antiarrhythmic therapy. The evaluation of the electrogram stored at the time of the shock episode revealed that the therapy was inappropriate and was due to the presence of electrical artifacts, caused by a probable lead fracture. The decision was made to attempt the removal and replacement of the lead. During the procedure, adherences along the course of the lead and venous occlusion were noticed in the right subclavian vein. Thus, it was decided to leave the failed lead in situ. A new single-coil lead was advanced through the left subclavian vein to the right ventricular apex and connected to the T-ICD in a new pocket on the left pectoral side. Despite optimal electrical parameters (pacing threshold 0.4V, impedance 650 Ohm, sensed R-wave amplitude 30 mV), during the defibrillation test, the T-ICD failed to convert a ventricular fibrillation with two shocks at 30J and 40J, and the arrhythmia was interrupted with an external shock. The lead was then repositioned in a septal position, and a second ventricular fibrillation was induced. The system failed again to convert the arrhythmia with two 40J shocks, with both standard and reverse polarity, and an external shock was

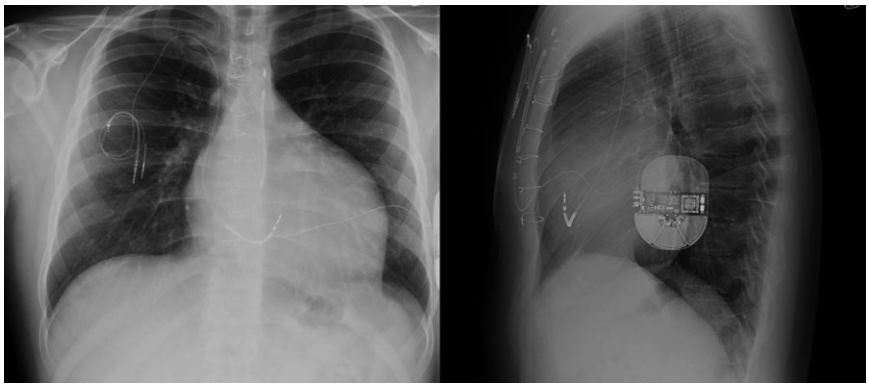

FIGURE 1 Final positioning of the S-ICD and previous transvenous lead left in situ

required to restore sinus rhythm. The T-ICD was removed, and the procedure was interrupted. It was therefore decided to attempt the implantation of an S-ICD (Boston Scientific, Natick, MA, USA). The preimplant surface electrocardiogram screening was successful, and all sensing vectors were considered acceptable. The S-ICD was implanted with an intermuscular approach for the pocket between the anterior surface of the serratus anterior and the posterior surface of the latissimus dorsi. The lead was vertically positioned in the subcutaneous tissue of the chest, 2 cm to the left of the sternal midline (Figure 1). Ventricular fibrillation was induced using 50-Hz transthoracic stimulation and successfully interrupted with a 70J direct-polarity S-ICD shock after 13 seconds (Figure 2). Shock

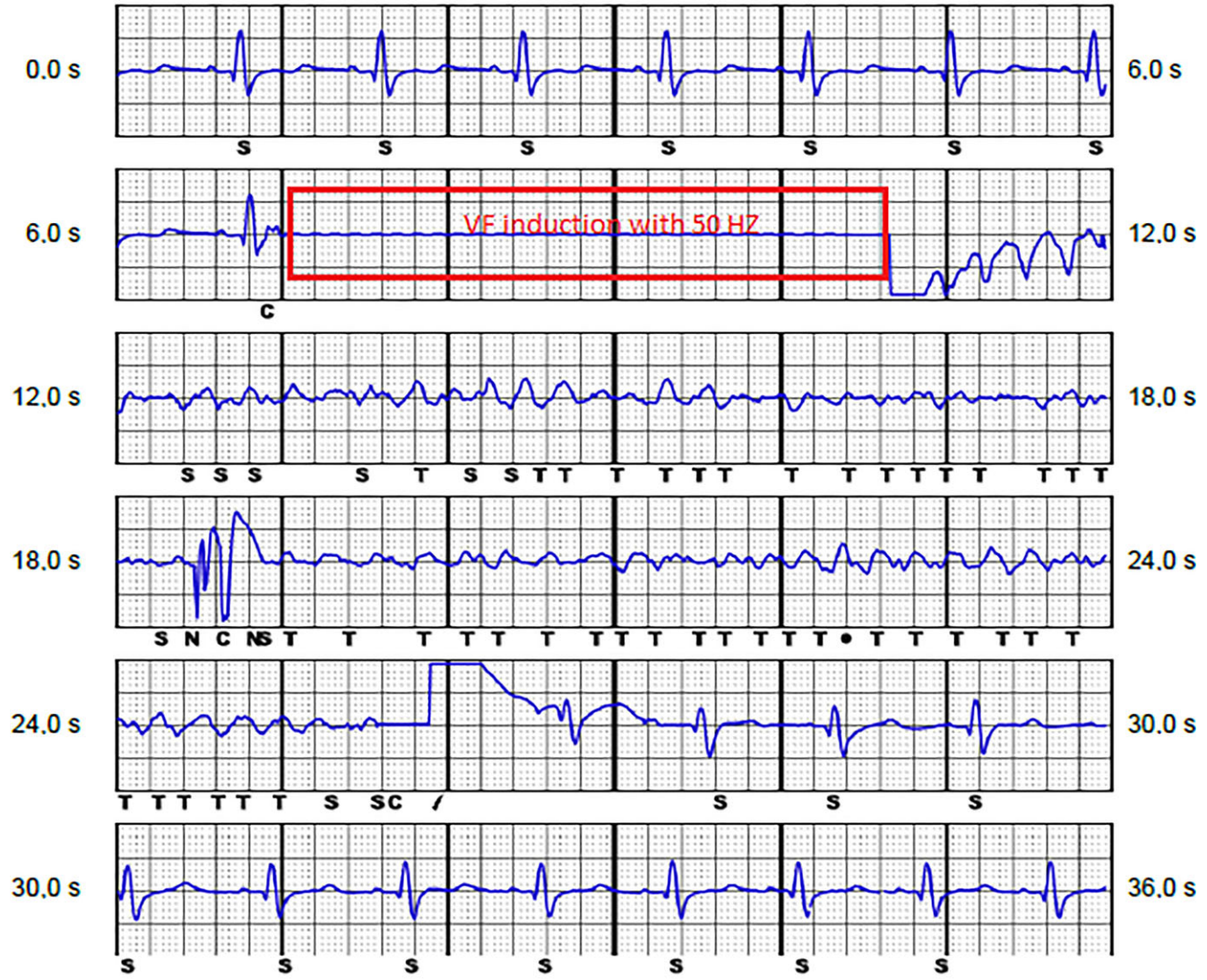

FIGURE 2 Induction of ventricular fibrillation and successful termination with a 70J S-ICD shock after 13 s

impedance was optimal (65 ohm), despite patient's moderate overweight (BMI = 26). The secondary vector was used for sensing during the test and for chronic programming. The patient had uncomplicated postoperative course and was discharged 3 days after the procedure.

3 | DISCUSSION

The S-ICD is an alternative for patients with an ICD indication who do not have a pacing indication or ventricular tachycardia for which antitachycardia pacing may be required.[1]

In HCM patients, increased myocardial mass may make it difficult to interrupt ventricular arrhythmias by ICDs. For this reason, the DFT test is still generally recommended for this category of patients. In this case, the advanced stage of the disease and the presence of a previously abandoned catheter may have contributed to the failure of DFT testing despite multiple shocks configurations.

Patients with HCM are known to have higher DFTs than patients implanted with T-ICDs for other indications, and the DFT is known to increase with increasing left ventricle wall thickness.[2] By contrast, it has been recently shown that in HCM patients, S-ICD was effective at recognizing and terminating ventricular fibrillation at implant with a wide safety margin,[3] and that extreme left ventricular hypertrophy did not affect the performance of the device.[4] The result of the present case is in agreement with these findings. The possible reason is that the S-ICD is capable to deliver greater shock energies (up to 80 J) compared to T-ICD. Moreover, the T-ICD lead is placed inside the right ventricle, in the anterior portion of the heart. Although shock vectors originating from right ventricle are adequate to convert ventricular arrhythmias in most of the cases, in HCM patients, the majority of the pathological myocardial mass is positioned in the posterior part of the heart[5] and may be at least partially not involved by a shock delivered from the coil to a prepectoral pocket or to a superior vena cava coil. By contrast, the posterolateral positioning of S-ICD generator can produce an optimal shock vector involving a bigger portion of left ventricular mass.

In addition, although both T-ICD and S-ICD have been shown to be effective in HCM patients, another key advantage of the S-ICD is the avoidance of lead complications because many HCM recipients are young patients and they are at risk of the long-term morbidities associated with intravascular leads, as the present case shows.

In conclusion, the S-ICD proved to be effective in this patient with HCM, previous failure of transvenous ICD lead, and inability of a T-ICD to convert a ventricular fibrillation. Therefore, in young HCM patients who do not require pacing, S-ICD can be considered a first-line therapy for the prevention of sudden cardiac death. Nonetheless, either with transvenous ICD or with S-ICD the defibrillation testing should not be abandoned in this population.

CONFLICT OF INTEREST

Authors declare no Conflict of Interests for this article.

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