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Combination of a leadless pacemaker and subcutaneous defibrillator with nine effective shock treatments during follow-up of 18 months

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ABSTRACT

We present a case of combination of a leadless pacemaker (Micra) and a subcutaneous implantable cardioverter-defibrillator (S-ICD). The patient had a total of nine adequate shock treatments of ventricular fibrillation during 18 months of follow-up after the implantation. The shock treatments did not lead to any alteration in the Micra. All three sensing vectors of the S-ICD worked well. After 18 months, the functioning of both Micra and S-ICD continues to be uneventful. This case demonstrates that S-ICD combined with Micra may be a safe and feasible approach to provide pacing and ICD treatment without intracardiac leads.

Introduction

Subcutaneous implantable cardioverter-defibrillator (S-ICD) and leadless pacemaker are two novel technologies that have reduced lead-related complications. An important device-specific limitation of the S-ICD is its lack of pacing ability, which may be solved by combining the treatment with a leadless pacemaker. Thus, leadless pacemaker and S-ICD might be a useful combination for patients who are unsuitable for intracardiac electrode implantation, for example patients with tricuspid valve diseases and those lacking vascular access for transvenous lead insertion. Here we present a patient in whom the leadless pacemaker and S-ICD were used in combination, and nine effective shock treatments were delivered without any complications arising.

Case report

A 48-year-old woman with rheumatic heart valve disease and permanent atrial fibrillation was treated mechanical aortic and mitral heart valve prostheses, and a biological tricuspid valve prosthesis. She had been treated with Amiodarone for atrial fibrillation (Table 1), but the treatment was ended due to amiodarone-induced thyrotoxicosis three years before, she had episodes of syncope and subsequently suffered from out-of-hospital cardiac arrest (ventricular fibrillation (VF)), with successful defibrillation and resuscitation with 19 min to return of spontaneous circulation. Following the episode, the patient had a left ventricular ejection fraction (EF) of 40% with mild right ventricular dysfunction, QTc was 464 ms and heart rate was 40–50/min with repeated short non-sustained ventricular tachycardia (VT) episodes. Thyroid stimulating hormone (TSH) levels were normal, as were levels of T4 and T3. The cardiac arrest event was initially thought to be bradycardia-triggered, and the patient was therefore treated with a temporary transvenous pacemaker using the femoral approach. Due to recurrent short episodes of ventricular tachycardia (VT) during temporary pacing and the absence of bradycardia episodes, the cardiac arrest episode was primarily judged to be of a ventricular arrhythmia origin, although a bradycardia triggered arrhythmia could not be ruled out. It was therefore decided to implant a permanent ICD system on the indication of secondary prevention of sudden cardiac death (SCD) in a patient with prior VF.

During the ICD procedure it was initially attempted to place a lead into branch from the coronary sinus (CS). The CS lead procedure was selected in order to avoid placing a lead through the prosthetic valve in the tricuspid position because of risk for future valve degeneration. However, initial attempts to place a lead in the CS failed due to difficulties in locating the ostium of the CS. Finally, with the help of transesophageal echocardiography (TEE), the coronary sinus was identified, but the lead placement was prevented by an unfavorable angle in the coronary sinus. Eventually the ICD lead could be delivered into a posterolateral branch from the coronary sinus, but the amplitude of the R-wave was below 2 mV. A few different positions were tested in the vessel, but none were suitable. Thereafter, attempts to position the transvenous ICD lead via the tricuspid prosthesis to the right ventricle were unsuccessful due to a dilated right atrium and moderate tricuspid valve regurgitation. Thus, the placement of a functional transvenous ICD lead in the traditional way did not appear to be possible. It was therefore decided to perform a hybrid procedure using a leadless pacemaker in combination with a subcutaneous ICD. A Micra™ transcatheter pacing system device (Medtronic, MN, USA) was thus implanted three days

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Table 1
Timeline.

Time	Events
Year-14	Mechanical aortic and mitral prostheses implantation
Year-6	Amiodarone treatment start for atrial fibrillation
Year-3	Biological tricuspid valve prosthesis implantation
Year-3	Thyrotoxicosis due to amiodarone. Amiodarone out.
Month-4	Reveal LINQ ICM (Medtronic) implantation due to syncope ×2
Day-23	Out of hospital cardiac arrest (ventricular fibrillation)
Day-14	Attempts to position the ICD lead in the CS or RV failed
Day-11	A Micra™ Transcatheter Pacing System device was placed
Day 0	A subcutaneous ICD (S-ICD) was implanted
Month 3/Day 14	First appropriate detection and shock treatment of VF
Month 10/Day 25	Shock treatment of VF
Month 11/Day 10	Shock treatment of VF
Month 11/Day 12	Shock treatment of VF
Month 11/Day 16	Mexiletine in
Month11/Day 17	Mexiletine out due to side effects
Month 12/Day 29	Start radioiodine treatment
Month 13/Day 26	Shock treatment of VF
Month 14/Day 6	Shock treatment of VF
Month 14/Day 10	Untreated VF episode
Month14/Day 12	Sotalol in and Metoprolol out
Month 17/Day 0	Shock treatment of VF
Month 17/Day 21	Shock treatment of VF
Month 18/Day 4	Shock treatment of VF
Month 18/Day 11	Start amiodarone treatment. Sotalol out
Month 24	No VF

later (Fig. 1). At the beginning, the leadless pacemaker was placed at the RV apex, but with high Threshold (2 V at 0.4 ms). The leadless pacemaker was implanted again high up on the septum between the tricuspid valve and the apex of RV with good measurements (threshold 0.5 V at 0.24 ms, R-wave 7.6 mV, and lead impedance 670 Ω). Eleven days later, the patient was received a subcutaneous ICD (S-ICD) (Emblem MRI; Boston Scientific, MA) under general anesthesia, with the can placed in a left lateral position over the fifth and sixth intercostal space between the anterior surface of the serratus anterior muscle and the posterior surface of the latissimus dorsi muscle. The defibrillation lead was tunneled towards the xiphoid process and then cranially to the left of the sternum. Defibrillation test was performed by the end of the implantation and a 65 J shock effectively restored rhythm. Shock treatment did not result in mode reversion, shutdown, or dislodgment of the leadless pacemaker. The procedure was well tolerated without any complications.

Exercise test was performed one month after the implantation. The heart rhythm during the stress test was atrial fibrillation. Working capacity was 48% of the expected, and maximum pulse during work was approximately 130 beats per minute (approximately 75% of the calculated max pulse). Work was limited by general fatigue and shortness of breath, while there was no chest pain. The S-ICD control did not show any misinterpretation of pacing artifacts or T-wave over-sensing during the exercise test. All three sensing vectors of the ICD worked well.

During follow-up, the patient has had nine episodes of syncope. Subsequent device interrogation confirmed appropriate detection and adequate shock treatment of ventricular fibrillation in all episodes (Fig. 2) without any adverse effect on the leadless pacemaker. Some episodes started as polymorphic VT, some as torsade de pointes VT, all in the VF zone (>250 beats/min). At one year follow up after S-ICD implantation, left ventricular EF was 30–35% with moderate right ventricular dysfunction.

After the fourth episode of syncope (and correctly delivered shock for VF) during the follow up period it was decided to start amiodarone treatment. But, before initiation of amiodarone it was decided to treat patient with radioiodine to prevent possible amiodarone-induced thyrotoxicosis. Following Radioiodine treatment at 12 months post ICD, but before initiation of amiodarone treatment (initiated at 18 months post ICD) there were 5 additional episodes of VF. However following initiation of amiodarone at 18 months there were no more registered episodes of VF up to last follow up at 24 months (Table 1).

At 18 months of follow-up, the characteristics of both the leadless pacemaker and the S-ICD continue to be normal, with 88.9% ventricular pacing: RV threshold 0.75 V at 0.24 ms, R-wave 15.9 mV, and RV lead impedance 430 Ω (Micra measurements). At 24 months of follow-up, both the leadless pacemaker and the S-ICD still worked well. No oversensing or double counting was detected by the S-ICD during pacemaker tests at follow-up interrogations.

Discussion

A few previous case reports have described S-ICD systems in patients with a leadless pacemaker [1,2]. To our knowledge, this is the first case report involving repeated, clinically correct effective shock treatments in a patient with a leadless pacemaker implanted in combination with an S-ICD. No dysfunction of the leadless pacemaker after delivery of S-ICD shocks was found in this case. The leadless pacemaker works well directly after shock treatment. Moreover, no misinterpretation of pacing artifacts or T-wave oversensing was perceived by the S-ICD. On the other hand, nine treated VF episodes and an untreated self-limiting VF episode were all correctly detected. This case demonstrates that S-ICD treatment combined with a leadless pacemaker may be a safe and effective approach to providing pacing and ICD functions without intracardiac leads.

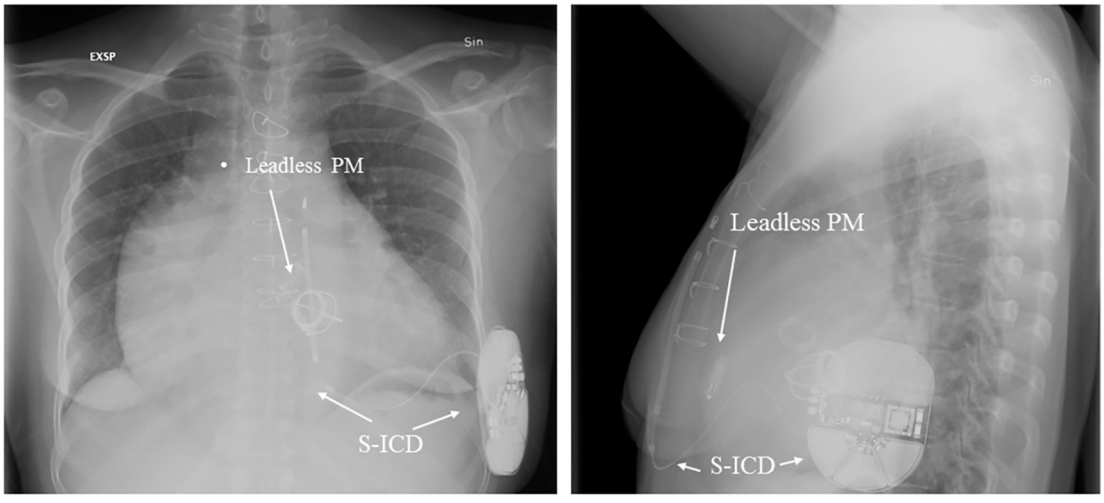

Fig. 1. Chest X-ray after implantation of the subcutaneous implantable cardioverter-defibrillator (S-ICD) and the leadless pacemaker.

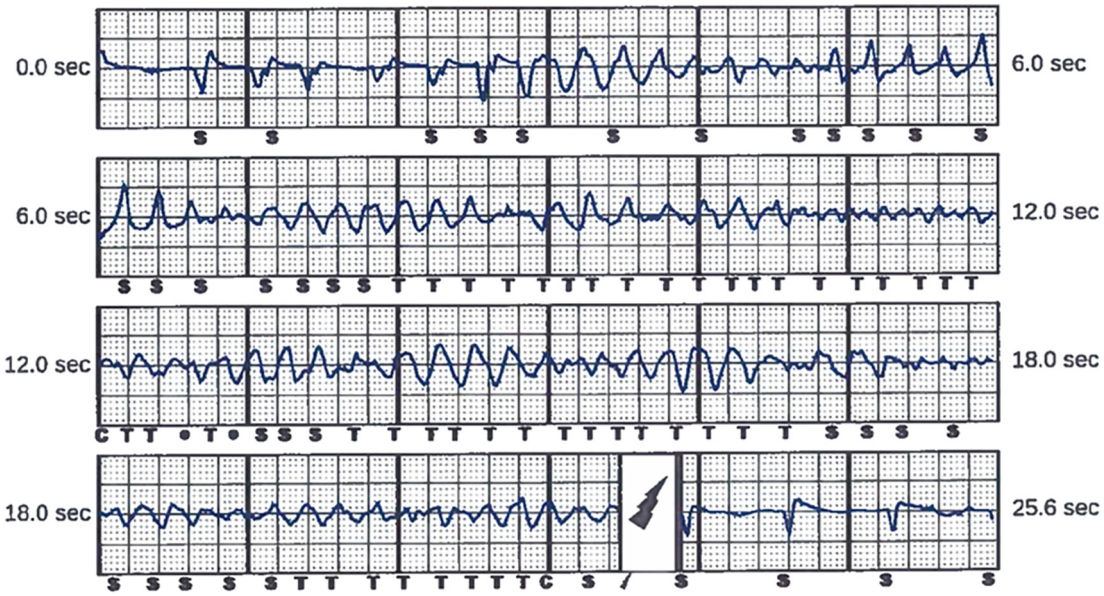

Fig. 2. Shock treatment of ventricular fibrillation.

The main cause of the recurrent VF episodes after implant may be due to the slight worsening of the patient's left ventricular EF (40% pre implant vs. 30% post implant) and/or possibly due to the small reduction in right ventricular function seen post procedure. Less likely as the cause of the ventricular arrhythmia episodes is late of effects of thyrotoxicosis although it cannot be completely be ruled out. The thyrotoxicosis event occurred three years before out-of-hospital cardiac arrest and the patient hade subsequent blood tests with TSH, T3 and T4 in the normal range. Furthermore, it has to be considered that there may be a proarrhythmic effect of the pacing of a leadless pacemaker and thus a possible cause of the post procedure episodes of VF [9]. Lastly, it has to be noted that high rate RV pacing can also impair cardiac function which could possibly then also contribute to ventricular arrhythmias.

Epicardial ICD could be a solution for patients without intracardiac leads; this was first used for life-threatening ventricular arrhythmias in 1980 [3]. Epicardial ICD systems consisted of contoured epicardial patches to provide adequate defibrillation; these needed a thoracotomy for implantation and they were associated with a high rate of lead malfunction [4], including dense fibrosis around the patches, accumulation of fluid beneath the patches, constrictive pericarditis, and difficult surgical removal due to fibrosis and calcification. For these reasons, epicardial patches have been abandoned [4]. ICDs using epicardial pacing/sensing and pleural shock leads [5] would be another possible approach for patients not suitable for intracardiac leads, but the lead-related complications continue to be the most challenging issue. An alternative approach to this also involves a transvenous ICD lead being inserted directly into the left atrium (transatrial approach) and then positioned into the left ventricle [6]. This may increase the risk of thromboembolic complications and should be evaluated further.

Transvenous leads are a known source of iatrogenic tricuspid regurgitation [7] and the patients with a tricuspid valve prosthesis may develop valvular degeneration necessitating a need for a new valve in the tricuspid position in due time. For those patients who need VVI pacing and ICD treatment but in whom standard transvenous approaches are not feasible or desirable, the combination of a leadless ICD and an S-ICD might therefore be a useful strategy in such clinical settings. An alternative approach to this also includes implantation of an ICD lead into a side branch of the coronary sinus instead of a surgically placed epicardial lead, to avoid the risk of repeated surgery and the unreliable and limited longevity of epicardial leads [8]. Attempts to follow this approach unfortunately failed in our case because of difficulties in coronary sinus cannulation, with resulting the low R-wave amplitudes and exit block.

The recent introduction of an entirely subcutaneous ICD is an important step forward in defibrillation technology towards a less invasive approach and a leadless pacemaker gives effective pacing without intracardiac leads. A leadless pacemaker implanted in combination with an S-ICD in patients who are unsuitable for placement of an intracardiac electrode is a safe and feasible approach to providing pacing and ICD functions without intracardiac leads. Follow-up over 18 months in this case has shown the effectiveness and safety of S-ICD combined with a leadless pacemaker despite delivery of multiple shocks.

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Declaration of Competing Interest

The authors report no conflicts of interest.

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