



Hyperglycemia-Induced T-Wave Oversensing as a Cause of Cardiac Resynchronization Therapy (CRT) Failure

Mohammad Assadian Rad, MD*, Zahra Emkanjoo, MD, Hassan Moladoust, PhD

Heshmat Cardiovascular Medical Center, Guilan University of Medical Sciences, Rasht, Iran.

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Abstract

T-wave oversensing occurs when the counter starts giving dual beeps for every cardiac cycle instead of one. This usually happens when the monitoring lead displays a tall T wave, which is also sharp. R wave sensing algorithms of the devices do not sense T wave because the slow rate of the T wave is much less than that of the R wave. But the slow rate of T waves may change with time and also because of parameters like potassium levels and hyperglycemia. We present a 67-year-old female who underwent the implantation of cardiac resynchronization therapy (cardiac resynchronization and implantable cardioverter defibrillator [CRT-D]) because of severe left ventricular systolic dysfunction and ventricular dyssynchrony experienced recurrent inappropriate implantable cardioverter-defibrillator (ICD) shocks and CRT failure. Device analysis showed that the CRT failure was in consequence of T-wave oversensing due to hyperglycemia. Elimination of the T-wave oversensing after hyperglycemia control conferred good biventricular pacing and good response to CRT during a 6-month follow-up period.

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Introduction

Loss of cardiac resynchronization therapy (CRT) is a frequent problem that limits the potential benefits associated with this form of non-pharmacological treatment for patients with heart failure.¹ It usually occurs in consequence of loss of left ventricular (LV) and right ventricle (RV) capture, loss of atrial sensing, or loss of atrial tracking due to atrial rates greater than the maximum tracking rate; however, its presentation at rates lower than the programmed maximum tracking rate is also possible. In this case report, we present a 67-year-old woman with hyperglycemia-induced T-wave oversensing as a cause of CRT failure.

Case report

A 67-year-old female with a history of diabetes mellitus and renal failure as well as a diagnosis of dilated cardiomyopathy and New York Heart Association Functional Classification III, referred for cardiac resynchronization defibrillator therapy. A CRT-D device is an implantable cardioverter-defibrillator (ICD) enabled with CRT implantation. Echocardiography showed a left ventricular ejection fraction (LVEF) of 20% and left ventricular end-diastolic dimension (LVEDD) of 60 mm. Basal electrocardiogram (ECG) showed a left bundle branch block (LBBB) pattern and sinus rhythm. Additionally, the patient had serum creatinine (Cr) of 1.8 mg/dL, blood

*Corresponding Author: Mohammad Assadian Rad, Department of Pacemaker and Electrophysiology, Guilan University of Medical Sciences, Heshmat Cardiovascular Medical Center, Rasht, Iran. 4193955588. Tel: +98 9123367496. Fax: +98 131 6669064. E-mail: Dr_a_assadian@yahoo.com.



urea nitrogen (BUN) of 55 mg/dL, and serum potassium (K^+) of 5.4 mm/dL.

The patient underwent implantation of a dual-chamber InSync Sentry DR 7298 ICD (Medtronic Inc., USA). The ICD was programmed to DDD, 60-120 beat/min, with a single shocking zone and a rate cut-off of 188 beat/min. She was discharged in good condition. During the next three months, ICD interrogation revealed good parameters and no identified tachyarrhythmias. In addition, the patient's dyspnea improved post ICD-CRT implantation. Three months later, she returned to the emergency ward due to frequent ICD shocks and deterioration of dyspnea.

The results of the admission blood test were as follows: K^+ = 5.2 mg/dL, blood sugar = 560 mg/dL, serum creatinine = 2.6 mg/dL, and BUN = 65 mg/dL. Also, the serial cardiac markers' levels were within the normal limits, and arterial blood gas measurement did not show acidosis or alkalosis. Chest radiography showed normal position of the implanted leads. The CRT-D parameters were checked in supine position. The stored electrograms at supine position revealed a ventricular sensing threshold of 6 mV and T-wave oversensing during sinus rhythm (ventricular sensitivity = 0.5 mV). Moreover, atrial and ventricular pacing, sensing thresholds, and leads impedances were unchanged in comparison with previous measurements.

The patient was admitted to the coronary care unit (CCU) and treated with insulin to control her diabetes. After the patient's blood sugar was reduced to 187 mg/dL, another ICD interrogation was carried out, which demonstrated no T-wave oversensing at a maximum sensitivity of 0.18 mV. With the patient's permission, the serum glucose was allowed to increase through a temporary discontinuation of insulin therapy. Two days later at a capillary glucose of 531

mg/dL, intermittent T-wave oversensing was detected at a sensitivity level of 0.5 mV.

After a two-month follow-up period, the patient's serum glucose was well controlled with insulin therapy and she had no further ICD shocks and T-wave oversensing, which conferred good biventricular pacing and good response to CRT.

Discussion

T-wave oversensing is the major cause of inappropriate ICD shocks (Figure 1).¹ Different mechanisms having been implicated in the double counting of R and T waves include drugs, hyperkalemia, hyperglycemia variations in sympathetic tones, and alterations in the shape of the intracardiac electrogram.^{2,3}

Elimination of T-wave oversensing is possible through the correction of reversible causes and device programming, and the repositioning of the ventricular electrode to find a greater R-wave amplitude makes oversensing less likely.³⁻⁵ T-wave oversensing was avoided by considering the following options in the present study: (1) reprogramming the ICD to lower sensitivity; (2) implanting the LV epicardial lead for sensing and pacing; (3) changing the detection counts; (4) administering heart rate slowing drugs; (5) repositioning the right ventricular lead to obtain ventricular electrograms with higher amplitudes; (6) implanting another endocardial sense-pace lead; and (7) prolonging postsens refractory periods.

Decreasing the sensitivity or programming longer refractory periods can sometimes treat T-wave oversensing, but these measures may interfere with the ability of the ICD to correctly detect tachyarrhythmias. The best option for the



Figure 1. Stored intracardiac electrogram (EGM) of the patient in emergency room at first visit during intracardiac device interrogation

Line 1: Atrial EGM (A. EGM)

Line 2: Tip to ring configuration (V. EGM)

Line 3: Marker channel. Recorded electrogram of this patient's cardiac resynchronization therapy reveals T-wave oversensing at 0.5 mV during sinus rhythm at a rate of 83 beats/min leading to loss of biventricular pacing. Oversensing of the T wave places the subsequent P wave in the ventricular refractory period, resulting in a lack of tracking and loss of biventricular pacing.

AR, Atrial refractory; AS, Atrial sense; TF, Tachy-fibrillation; BV, Biventricular pace; VS, Ventricular sense

treatment of T-wave oversensing due to a reversible cause is the treatment of the underlying cause. The sensitivity and refractory periods of the device were, therefore, not changed in the present study.

In this type of Medtronic ICD, postsense refractory periods are not programmable. Another option for the prevention of inappropriate shocks due to T-wave oversensing is changing the detection counts; i.e., increasing the number of successive counts before the ICD is triggered to call the arrhythmic ventricular fibrillation (VF). Appropriate identification and correction of the underlying cause of T-wave oversensing is an essential management strategy. In the case presented herein, the elimination of T-wave oversensing through a successful control of the patient's blood sugar led to the tracking of the sensed or paced atrial impulses and biventricular pacing.

CRT, a technology that simultaneously paces both the left and right ventricles, has emerged as an important treatment tool for heart failure patients with reduced LV function and ventricular dyssynchrony. There are several possible reasons why as many as 30% of treated patients remain unresponsive to CRT. When a non-responder is identified, the first thing that should be determined is whether the patient is actually receiving CRT. There are two reasons for this problem: first, possible loss of function of the lead in the coronary sinus; and second, the rapid or ectopic intrinsic heart rhythm (e.g., atrial fibrillation or frequent premature ventricular complexes). One should also take into account inappropriate programming or oversensing as the other possible causes of biventricular pacing failure leading to CRT failure.

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