T-wave oversensing precipitating cardiogenic shock in a pacer-dependent patient

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Introduction

T-wave oversensing (TWOS) is a well-known phenomenon that occurs secondary to sensing the T wave as an R wave, leading to inhibition of next pacing stimulus owing to double counting of the ventricular rate.[1] Several abnormalities, such as electrolyte imbalance, lead malposition, myocardial ischemia, prolonged QT syndromes, and even structural abnormalities like hypertrophic cardiomyopathy or any disease that can cause high-amplitude T waves, delayed T waves, or low-amplitude R waves, can increase the risk of TWOS.[2–5] TWOS can result in a variety of deleterious outcomes, with inappropriate implantable cardioverter-defibrillator (ICD) shocks and antitachycardia pacing and loss of biventricular pacing being the most common clinical scenarios. TWOS-induced inappropriate ventricular pacing (VP) inhibition that results in bradycardia is rarely reported in the literature.[6,7] The impact of this phenomenon becomes extremely critical in pacemaker-dependent patients. We report a unique case of TWOS in a pacemaker-dependent patient with acute decompensated heart failure that caused inappropriate VP and bradycardia that led to cardiogenic shock.

KEY TEACHING POINTS

- T-wave oversensing (TWOS) is a common electrophysiological phenomenon that can occur because of several derangements, such as electrolyte imbalance, lead malposition, or even myocardial ischemia.
- Inappropriate antitachycardia pacing and implantable cardioverter-defibrillator shocks are considered the most clinically encountered TWOS-induced complications.
- Inhibition bradycardia pacing caused by TWOS-induced inappropriate ventricular pacing inhibition is a potential complication; however, it is rarely encountered in clinical practice.
- TWOS-induced bradycardia in pacemaker-dependent, critically ill patients, such as patients with acute decompensated heart failure, can be hemodynamically significant and potentially precipitate cardiogenic shock.
- Multiple reprograming options to correct TWOS are available, including adjustment to ventricular sensitivity, lead sensing configuration, and postpacing blanking period.

Case report

An 80-year-old woman with a history of permanent atrial fibrillation (Afib), hypertension, hyperlipidemia, diabetes mellitus, and chronic obstructive pulmonary disease was transferred to our institution for a higher level of care. She had undergone atrioventricular node ablation with a Medtronic Azure XT SR MRI W1SR01 (Medtronic, Minneapolis, MN) single-chamber permanent pacemaker (PPM) placement at a different facility 2 years ago at the lower rate set at 70 beats per minute (BPM). She presented to the outside hospital with worsening NYHA class IV heart failure symptoms and signs of volume overload.

At our institution, her blood pressure was borderline with mean arterial pressure of 60 mm Hg, heart rate of 70 BPM, and oxygen saturation of 98% on noninvasive positive pressure ventilation. Twelve-lead electrocardiograms showed Afib with a ventricular paced (VP) rhythm at 70 BPM. Her laboratory investigations revealed no significant electrolyte abnormalities, creatinine of 1.9 mg/dL, elevated liver enzymes, B-type natriuretic peptide of 829 pg/mL (normal high reference is 100 pg/mL), and high-sensitivity troponin I of 644 pg/mL (normal high reference for females is $\leq$15 pg/mL) that trended down to 628 pg/mL. Subsequently, a transthoracic echocardiogram was performed and showed a left ventricular ejection fraction of 35% $\pm$ 5%. She was started on intravenous furosemide and heparin continuous

KEYWORDS T-wave oversensing; Bradycardia; Pacemaker; Inappropriate ventricular pacing; Cardiogenic shock
(Heart Rhythm Case Reports 2023;9:728–731)

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https://doi.org/10.1016/j.hrcr.2023.07.015

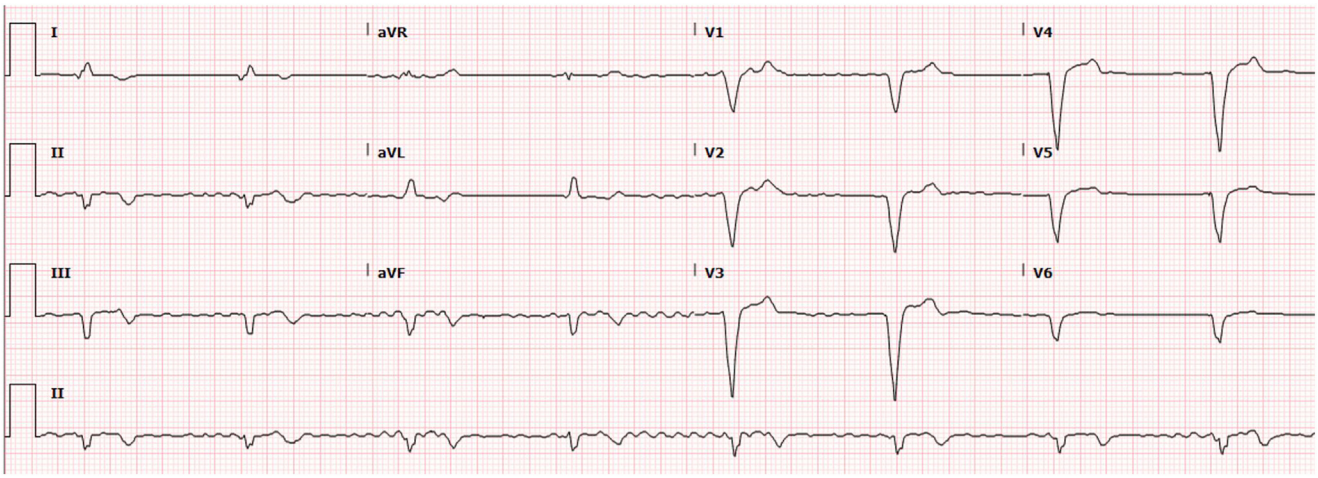

Figure 1 Twelve-lead electrocardiogram showing atrial fibrillation and ventricular pacing at a rate of 48 beats/min.

infusion and was admitted with diagnoses of acute decompensated heart failure and non-ST-elevation myocardial infarction.

On the next day, she became bradycardic with a heart rate of 40 BPM, her hemodynamic status started to deteriorate, and her kidney function worsened. A repeat electrocardiogram showed Afib with VP rhythm at 48 BPM with no evidence of any failure to capture (Figure 1). Chest radiography showed appropriate PPM lead position. Cardiogenic shock was suspected; thus, a pulmonary artery catheter was placed and revealed severely reduced cardiac output and cardiac index (CI) as well as elevated systemic vascular resistance and pulmonary capillary wedge pressure, confirming the diagnosis of cardiogenic shock. Afterward, the patient was started on intravenous milrinone infusion.

PPM device interrogation revealed a VVI programming mode with a lower limit of 70 BPM, acceptable right ventricular lead impedance, programmed sensitivity of 0.9 mV, and 99.9% VP requirement. Further analysis of the bradycardic episode showed TWOS that caused an inappropriate delay of the VP and resulted in bradycardia (Figure 2). Subsequently, we increased the right ventricular lead sensitivity to 2 mV to avoid further TWOS. That programming adjustment resulted in restoring the appropriate VP at 70 BPM. The patient's CI rapidly improved from 1.68 to 2.36 L/min/m^2 after resolution of the bradycardia (Figure 3). Her kidney function normalized, and she was successfully weaned off the milrinone infusion within the next 12 hours. She underwent coronary angiography, which ruled out ischemia and showed only mild-to-moderate nonobstructive coronary

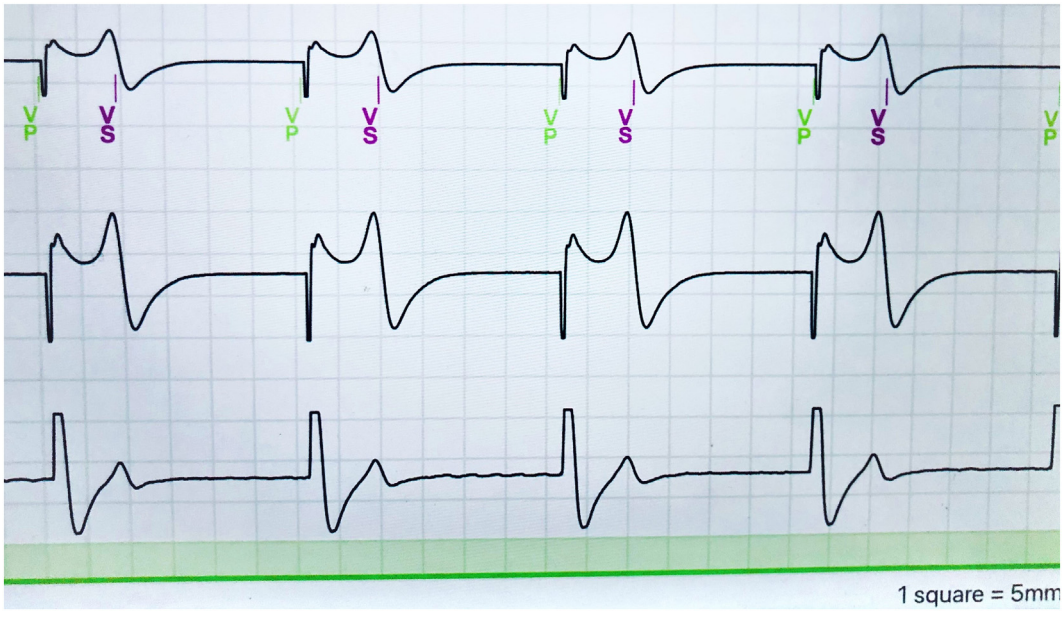

Figure 2 Permanent pacemaker interrogation showing inappropriate ventricular sensing at the T waves, confirming T-wave oversensing that caused a delay in ventricular pacing.

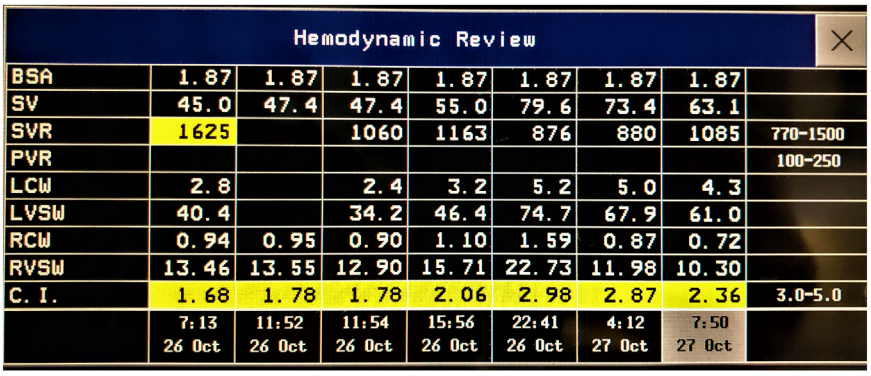

Figure 3 Hemodynamics review chart showing the improvement of cardiac index (C.I.) from 1.68 to 2.36 L/min/m^2 after abolition of the T-wave oversensing and restoration of an appropriate ventricular pacing.

artery disease, and cardiac magnetic resonance imaging, which showed improvement of left ventricular function with an ejection fraction of 61% and no evidence of any infiltrative disease. Eventually, she was discharged home in stable condition.

Discussion

Several deleterious outcomes can occur secondary to TWOS. Inappropriate antitachycardia pacing and ICD shocks are well-known complications of this phenomenon and are frequently reported in the literature.[2–5] TWOS is scarcely reported in pacemakers and is usually thought of by clinicians and presented in textbooks as ICD phenomenon. Suboptimal biventricular pacing and loss of cardiac synchronization therapy secondary to TWOS have also been described.[8] Although inappropriate VP inhibition owing to TWOS is a potential cause of significant bradycardia, we usually do not encounter these situations in our day-to-day practice. To the best of our knowledge, there is a paucity of reported cases describing clinically significant bradycardia caused by TWOS. There are few case reports that described inappropriate VP inhibition and bradycardia during postimplantation device interrogation that was found to be due to TWOS; however, no serious consequences were reported.[6,7] Nathan and colleagues[9] reported a case of TWOS that caused inappropriate VP and symptomatic bradycardia that manifested with persistent dyspnea on exertion and exercise intolerance. Punjabi and colleagues[10] reported another case of TWOS-induced intermittent symptomatic bradycardia that manifested as intermittent exertional lightheadedness. However, both cases remained hemodynamically stable.

We present a unique case of serious TWOS consequences. Our patient was unfortunate not only because she was pacemaker dependent, but also because she experienced TWOS during an episode of acute decompensated heart failure. The TWOS-induced bradycardia was clinically significant enough to cause an acute drop in her cardiac output and CI and eventually precipitated cardiogenic shock. Her CI normalized after abolition of the TWOS and restoration of an appropriate VP.

Treating patients with TWOS is usually not complicated; however, it can be challenging in some cases, especially when the diagnosis is missed. Generally, addressing any reversible cause of TWOS, such as correcting electrolyte imbalance, myocardial ischemia, or lead dislodgement, should be considered. Several device programming adjustments can be performed to eliminate TWOS, such as increasing the ventricular lead sensitivity, increasing the post–ventricular pacing blanking period, or changing the lead sensing configuration from true bipolar to integrated bipolar or vice versa. In some challenging cases, refractory TWOS was abolished only by changing the generator for a new one that uses a different sensing algorithm.[9,10] In our case, we were able to eliminate TWOS by increasing the programmed lead sensitivity, which resulted in appropriate VP. She tolerated this adjustment without further inhibition of ventricular pacing.

Conclusion

TWOS can result in a variety of deleterious outcomes, including inappropriate VP inhibition and resultant bradycardia. This phenomenon can result in serious outcomes, especially in high-risk patients with heart failure who are pacemaker dependent. We reported a case of TWOS-induced bradycardia that was significant enough to precipitate cardiogenic shock. Abolishing TWOS resulted in a dramatic improvement of the patient's hemodynamics.

Funding Sources: This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Disclosures: No conflict of interest for all authors.

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