

T-wave oversensing and symptomatic bradycardia in a pacemaker-dependent heart failure patient due to drug-induced hyperkalemia

Samuray Zekeriyeyev¹
ORCID: 0000-0001-7458-3956

Ugur Canpolat¹
ORCID: 0000-0002-4250-1706

Kudret Aytemir¹
ORCID: 0000-0001-9279-8424

¹ Hacettepe University Faculty of Medicine, Department of Cardiology, Ankara, Türkiye.

Corresponding Author: Ugur Canpolat
E-mail: dru_canpolat@yahoo.com

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ABSTRACT

Hyperkalemia is a common clinical condition among heart failure patients especially if there is coexisting older age, potassium-sparing medication, and renal insufficiency. Close follow-up and titration of renin-angiotensin-aldosterone system inhibitors are essential to prevent the development of hyperkalemia. It can also cause significant abnormalities in patients with cardiac implantable electronic devices (CIED) including pacemakers and implantable cardioverter defibrillators (ICD) due to its impact on cardiac electrophysiology. Herein, we presented a pacemaker-dependent heart failure patient with single-chamber ICD in whom bradyarrhythmia signs and symptoms were observed due to T-wave oversensing and managed appropriately.

Keywords: heart failure, hyperkalemia, t-wave oversensing, bradycardia.

INTRODUCTION

In clinical practice, hyperkalemia is a common electrolyte abnormality particularly among elderly heart failure patients (with potassium-sparing medications) if there is coexistent acute/chronic renal failure 1. In patients with a cardiac implantable electronic device (CIED), hyperkalemia causes significant changes including widening of the paced QRS complex, increased atrial and ventricular pacing thresholds causing failure to capture, increased latency (usually with ventricular pacing) presented by a delay of the interval from the pacemaker stimulus to the onset of QRS, and oversensing of paced or spontaneous tall T waves causing inappropriate implantable cardioverter-defibrillator (ICD) shocks 2. Herein, we presented a pacemaker-dependent heart failure patient who was admitted with dizziness, weakness, and bradycardia despite the basal rate of pacing being set at 60 bpm in whom hyperkalemia induced T wave oversensing and delay in ventricular pacing were diagnosed and managed appropriately.

CASE

A 79-year-old male patient with a history of cardiac resynchronization therapy defibrillator (CRT-D) implantation after an atrioventricular (AV) node ablation due to ischemic cardiomyopathy and drug-resistant AF with high ventricular rates was admitted to our clinic due to CIED infection. After complete extraction of the infected left-sided device including all leads and battery, the patient was followed up with a temporary transvenous pacemaker for 3 weeks under i.v. antibacterial therapy. After 3 weeks, the patient underwent right-sided VVI-ICD implantation. The basal rate of the VVI-ICD was set to 60 bpm. During in-hospital follow-up, the patient was closely monitored and the heart rate was observed as 60 bpm at the electrocardiography (ECG). He has been discharged uneventfully with optimal medical therapy for heart failure and atrial fibrillation. His final medical therapy included rivaroxaban (15 mg o.d), carvedilol (12,5 mg b.i.d), candesartan (8 mg o.d), furosemide (40 mg o.d), and spironolactone (25 mg o.d). At the

time of discharge, serum potassium level was 3.66 mEq/L, and serum creatinine level was 1.13 mg/dL.

During the first week's follow-up visit, he complained of intermittent dizziness and the laboratory tests showed a serum potassium level of 5.42 mEq/L, and a serum creatinine level of 1.85 mg/dL. The 12-lead ECG revealed a ventricular paced rhythm with a rate of 60 bpm (Figure 1). Pacemaker interrogation revealed no abnormality at baseline parameters, pacing, and sensing measures. Thus, candesartan 8 mg o.d and spironolactone 25 mg o.d was stopped due to impaired renal functions and increased potassium level and a 24-hour Holter monitorization was planned for his intermittent dizziness. However, his dizziness frequency was increased and weakness in the upper and lower extremities was developed during follow-up. 24-hour Holter monitorization showed intermittent bradycardia episodes (ventricular rate of 40 bpm) during his symptoms (Figure 2A). Therefore, he was hospitalized for further follow-up. It was learned

that the patient continued to use candesartan 8 mg o.d and spironolactone 25 mg o.d. The 12-lead ECG showed an intermittent bradycardia episode with a ventricular pace rate of 40 bpm despite the pacemaker basal rate setting being 60 bpm (Figure 2B). No abnormal pacing spikes or capture failure was observed. Laboratory tests showed a serum potassium level of 6.96 mEq/L, and a serum creatinine level of 2.08 mg/dL. After ICD interrogation, intermittent T-wave oversensing was observed causing a ventricular rate of 40 bpm and it was eliminated by changing the sensing polarity. Intravenous insulin, inhaled beta-agonists, and intravenous calcium gluconate were administered for hyperkalemia management. All nephrotoxic medications were stopped during the hospitalization period because of acute renal failure and hyperkalemia. His symptoms, ECG recording, and T-wave oversensing were improved after normalization of serum potassium and creatinine levels and discharged uneventfully.

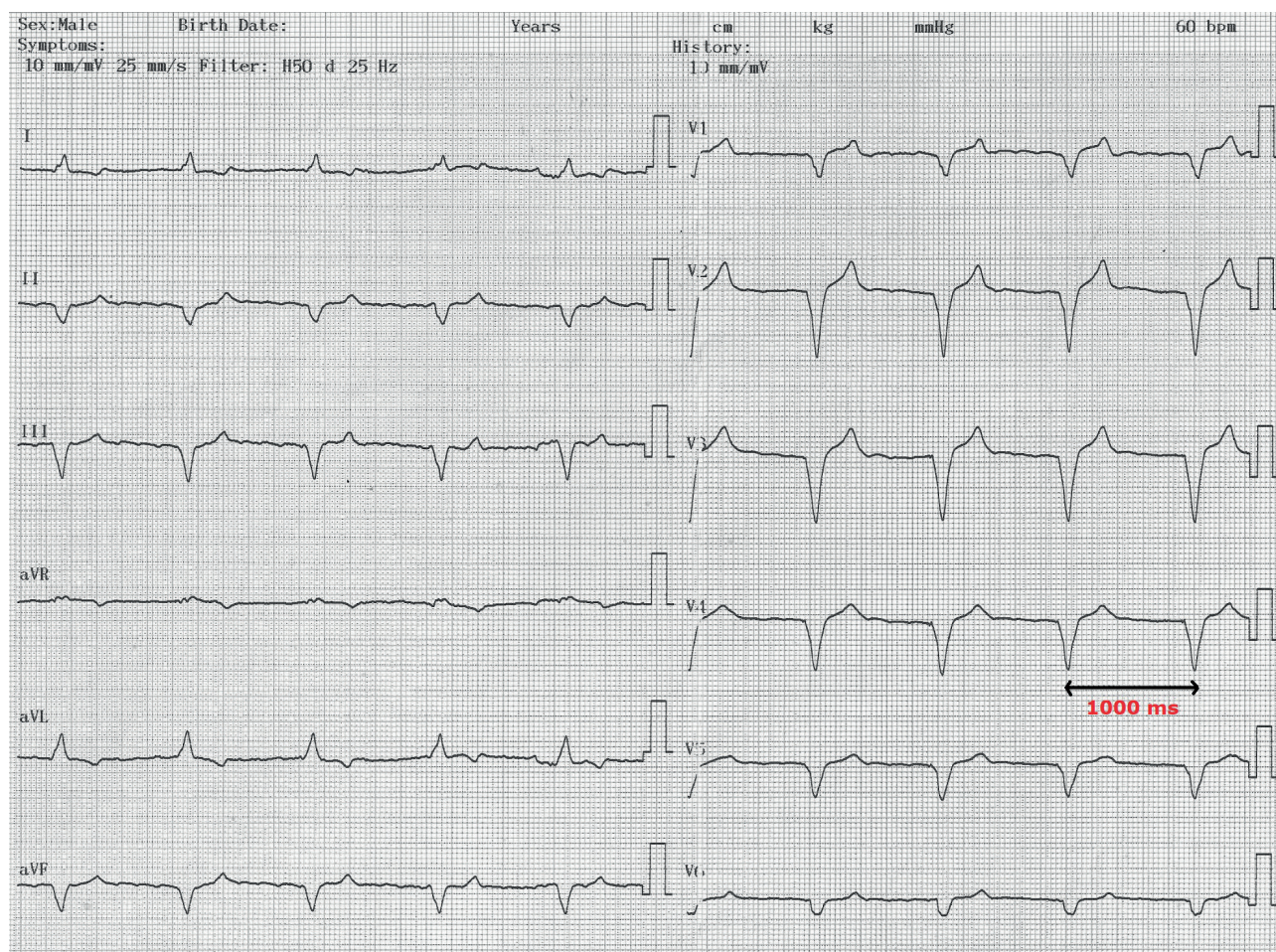


Figure 1. The 12-lead ECG revealed a ventricular-paced rhythm with a rate of 60 bpm

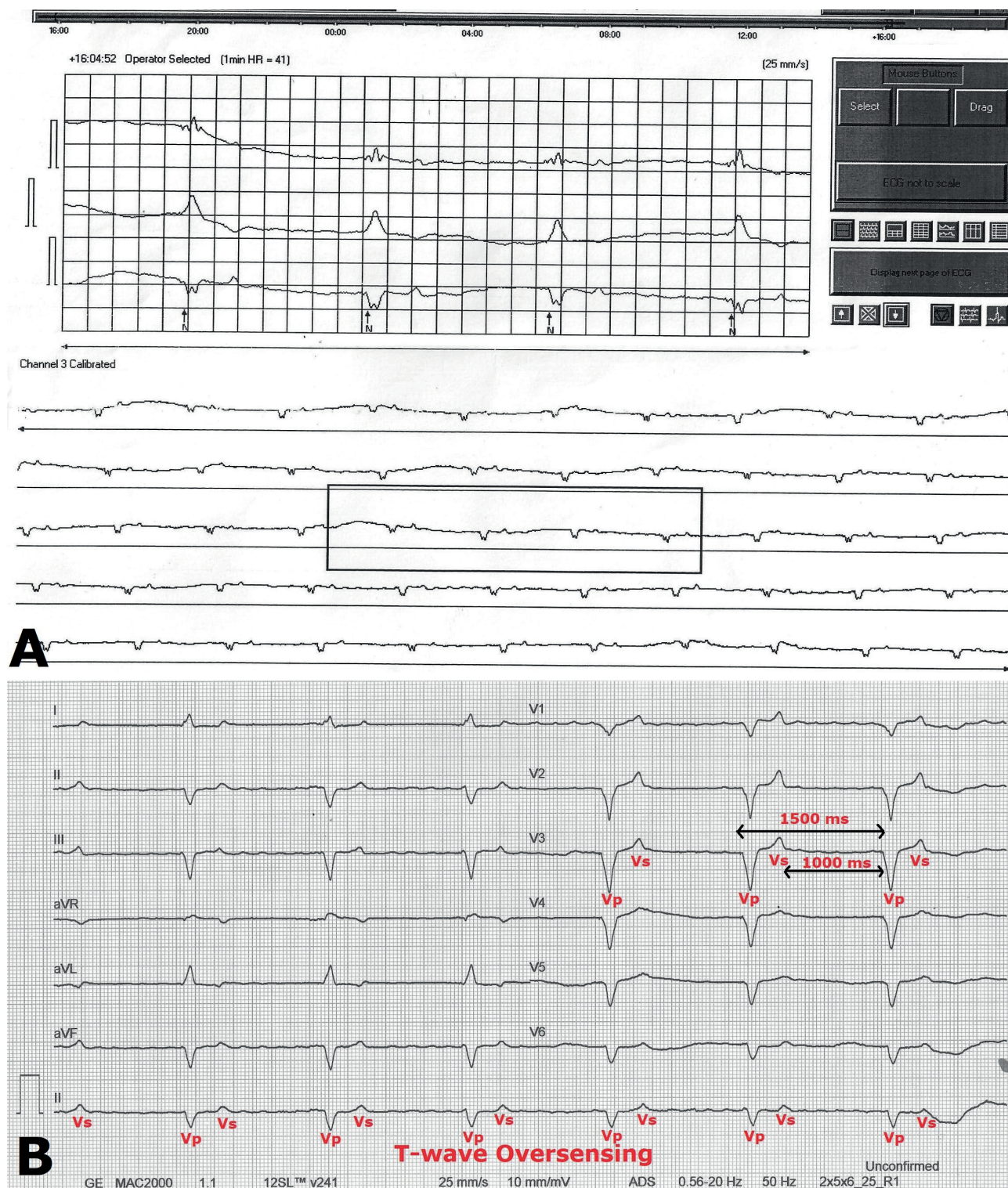


Figure 2. A) A 24-hour Holter monitoring showed intermittent bradycardia episodes (ventricular pacing rate of 40 bpm) during his symptoms. B) The 12-lead ECG showed an intermittent bradycardia episode with a ventricular pace rate of 40 bpm despite the pacemaker basal rate setting being 60 bpm

DISCUSSION

Hyperkalemia is a common clinical problem that is most often a result of impaired urinary potassium excretion due to acute or chronic kidney disease (CKD) and/or systemic disorders or drugs inhibiting

the renin-angiotensin-aldosterone system (RAAS). Daily clinical practice shows that it is common among elderly patients undergoing optimized heart failure treatment (with potassium-sparing drugs)

especially if there is coexisting renal insufficiency 2. In the TREAT HF (Turkish Research Team-Heart Failure) study, its prevalence has been reported as 17.7% among patients with heart failure and chronic renal disease 3. The most serious manifestations of hyperkalemia are muscle weakness or paralysis, cardiac conduction abnormalities, and cardiac arrhythmias. These manifestations usually occur when the serum potassium concentration is ≥ 7.0 mEq/L with chronic hyperkalemia or possibly at lower levels with an acute rise in serum potassium 1. Hyperkalemia may be associated with a variety of changes on the ECG. Tall peaked T waves with a shortened QT interval are usually the first findings. As the hyperkalemia gets more severe, there is a progressive lengthening of the PR interval and QRS duration, the P wave may disappear, and ultimately the QRS widens further to a sine wave pattern 4. Hyperkalaemia is the most common electrolyte abnormality causing loss of pacing capture. In patients with a CIED, hyperkalemia causes the widening of the paced QRS complex (and paced P-wave if it is seen) because of delayed myocardial conduction, and increased atrial and/or ventricular pacing thresholds causing failure to capture, and oversensing of the peaked T-waves causing inappropriate ICD shocks 2. Although we managed our patient per the current heart failure guidelines regarding optimal medications and close follow-up for electrolyte and renal functions, reminded our patient to stop RAAS inhibitors accordingly, hyperkalemia with significant signs and symptoms was developed because of the continuation of RAAS inhibitors by the patient. It is well known that ICD leads were more sensitive to intracardiac signals than conventional pacemaker leads. T-wave

oversensing due to hyperkalemia caused a delay in subsequent ventricular pacing as the patient was pacemaker dependent and the ventricular rate was dropped to 40 bpm in our patient. It was managed by changing the sensing polarity in the acute management and improved after appropriate treatment of hyperkalemia.

In conclusion, hyperkalemia induced by optimal heart failure medications is an important clinical condition that should be suspected, diagnosed, and managed appropriately. It can also cause significant changes in patients with CIEDs including pacemakers or ICDs. Device interrogation and prolonged rhythm monitorization with 24-hour Holter ECG are essential in pacemaker-dependent patients with bradycardia (pacing rate is slower than basal rate setting of pacemaker) and secondary causes like hyperkalemia should be suspected in the absence of primary pacemaker abnormality.

Author contribution

Study conception and design: UC, SZ and KA; data collection: UC and SZ; analysis and interpretation of results: UC, SZ and KA; draft manuscript preparation: SZ and UC. All authors reviewed the results and approved the final version of the manuscript.

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Conflict of interest

The authors declare that there is no conflict of interest.

REFERENCES

- [1] Altay H, Cavusoglu Y, Celik A, et al. Management of Hyperkalemia in Heart Failure. *Turk Kardiyol Dern Ars.* Oct 2021;49(Supp1):1-32.
- [2] Barold SS, Herweg B. The effect of hyperkalaemia on cardiac rhythm devices. *Europace.* Apr 2014;16(4):467-476.
- [3] Murat S, Kaya H, Cavusoglu Y, Yilmaz MB. Hyperkalemia in chronic heart failure with renal dysfunction or diabetes mellitus: Results from the TREAT HF study. *Turk Kardiyol Dern Ars.* Apr 2021;49(3):198-205.
- [4] Littmann L, Gibbs MA. Electrocardiographic manifestations of severe hyperkalemia. *J Electrocardiol.* Sep - Oct 2018;51(5):814-817.