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OPIS PRZYPADKU
CASE REPORT

One patient, multiple cardiac implantable devices, a shared threat: T-wave oversensing in the course of hyperkalemia

Jeden pacjent, wiele kardiologicznych urządzeń wszczepialnych, wspólne zagrożenie: *oversensing* załamka T w przebiegu hiperkaliemii

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ABSTRACT

Implantable cardiac devices, such as implantable cardioverter-defibrillators (ICDs) and pacemakers, play a crucial role in the management of patients with severe cardiac arrhythmias. These devices enable continuous monitoring and regulation of cardiac activity, thereby preventing life-threatening arrhythmic events. However, their function may be compromised by electrolyte imbalances, among which hyperkalemia is particularly significant. This condition may result in inappropriate shocks or misdetection of arrhythmias. We present the case of a 42-year-old male with a history of chronic heart failure and end-stage renal disease, who was admitted to a cardiology center for cardiovascular assessment and further management. In 2023, the patient underwent implantation of a subcutaneous implantable cardioverter-defibrillator (S-ICD) due to progressive heart failure. During the course of treatment, the patient experienced an inappropriate S-ICD shock triggered by hyperkalemia (serum potassium concentration of 7.21 mmol/L, which led to increased T-wave oversensing and subsequent inappropriate high-energy therapy). Implantable devices are a cornerstone in the treatment of patients with advanced cardiac arrhythmias; however, their effectiveness is highly dependent on the proper monitoring of serum electrolyte levels. Hyperkalemia can interfere with device sensing and therapy delivery, emphasizing the importance of regular electrolyte assessment, particularly potassium, in patients with implanted cardiac devices.

KEYWORDS

cardiac arrhythmias, hyperkalemia, T-wave oversensing, implantable cardioverter-defibrillator, cardiac pacemaker

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STRESZCZENIE

Kardiologiczne urządzenia wszczepialne, takie jak kardiowertery-defibrylatory (*implantable cardioverter-defibrillators* – ICDs) oraz stymulatory serca, pełnią kluczową rolę w leczeniu pacjentów z brady- i tachyarytmiami. Ich działanie może jednak zostać zakłócone zaburzeniami elektrolitowymi, do których należy hiperkaliemia. Taki stan może być przyczyną nieadekwatnych wyładowań lub błędnej detekcji arytmii. W pracy przedstawiono przypadek 42-letniego mężczyzny z przewlekłą niewydolnością serca i schyłkową chorobą nerek w wywiadzie, przyjętego do ośrodka kardiologicznego w celu oceny układu krążenia i dalszego leczenia. Pacjent przeszedł implantację podskórnego kardiowertera-defibrylatora (*subcutaneous implantable cardioverter defibrillator* – S-ICD) z powodu niewydolności serca ze znacznie zredukowaną frakcją wyrzutową lewej komory. Po implantacji pacjent doznał nieadekwatnego wyładowania S-ICD spowodowanego hiperkaliemią (stężenie potasu sięgało 7,21 mmol/L), która doprowadziła do zjawiska *oversensingu*, czyli nadczułości załamka T i nieadekwatnej terapii wysokoenergetycznej. Urządzenia wszczepialne to podstawowa metoda elektroterapii pacjentów z niewydolnością serca. Hiperkaliemia może prowadzić do zakłóceń w działaniu tych urządzeń, co podkreśla konieczność regularnej kontroli stężenia elektrolitów, zwłaszcza potasu, u pacjentów z implantowanymi urządzeniami.

SŁOWA KLUCZOWE

zaburzenia rytmu serca, hiperkaliemia, *oversensing* załamka T, kardiowerter-defibrylator, stymulator serca

INTRODUCTION

Heart failure (HF) represents a growing public health issue, with an estimated global prevalence of approximately 37.7 million patients [1,2]. It is associated with high mortality and constitutes a significant burden on healthcare system, being one of the leading causes of hospitalization among adults and the elderly [2]. The incidence of HF increases with population aging, improved prognosis of patients with ischemic heart disease, and greater societal access to effective therapies that prolong patient survival [3]. The etiology of heart failure is diverse and depends, among others, on sex, age, race, comorbidities, and environmental factors. Most cases of HF develop on the basis of coronary artery disease, chronic obstructive pulmonary disease (COPD), arterial hypertension, as well as valvular heart disease and diabetes mellitus [4,5]. The most common cause of HF is coronary artery disease [6].

HF, depending on left ventricular ejection fraction (LVEF), is classified into:

– HF with reduced ejection fraction (HFrEF), where LVEF is less than or equal to 40%
– HF with mid-range ejection fraction (HFmrEF), where LVEF is 41–49%
– HF with preserved ejection fraction (HFpEF), where LVEF is greater than or equal to 50% and symptoms of heart failure persist [6,7].

The main symptoms of HF are fatigue, dyspnea, and signs of fluid overload, including peripheral edema and pulmonary congestion [8]. Pharmacotherapy constitutes the foundation of HF treatment and should be initiated as first-line therapy, in parallel with non-pharmacological interventions. The main goals of such therapeutic management are the reduction of mortality, prevention of hospitalizations due to HF exacerbations, and improvement of clinical status, exercise capacity, and patient quality of life [7]. The cornerstone pharmacological agents are angiotensin-converting enzyme inhibitors (ACEIs), angiotensin receptor-neprilysin inhibitors (ARNIs), beta-blockers, and mineralocorticoid receptor antagonists (MRAs). Their mechanism of action is based on modulation of the renin–angiotensin–aldosterone system and the sympathetic nervous system, which improves the general condition of patients [9,10,11].

Despite optimal pharmacotherapy, a significant proportion of deaths among patients are due to sudden cardiac death, often caused by arrhythmias such as ventricular tachyarrhythmias, bradycardia, and asystole [7].

Implantable cardioverter-defibrillators (ICDs) are devices designed to detect and treat life-threatening ventricular arrhythmias. Their operation is based on the detection of ventricular tachycardia or ventricular fibrillation and the application of antitachycardia pacing, and if ineffective, the delivery of a shock to terminate the arrhythmia [12]. The transvenous, or conventional, ICD additionally has pacing functions in cases of bradyarrhythmias. Although ICDs prolong survival in HF patients by preventing sudden cardiac death, the intracardiac leads of conventional ICDs carry a risk of complications such as dysfunction or infective endocarditis. A response to these issues is the development of subcutaneous ICD systems (S-ICD), in which the leads are placed extrathoracically, outside the heart chambers. However, these systems have limitations, namely the lack of capability for continuous cardiac pacing in cases of bradycardia [13].

In addition to S-ICD, another innovation in implantable cardiac devices is represented by leadless pacemakers (Medtronic Micra™ TPS or Abbott Aveir), which constitute an effective alternative to conventional transvenous pacemakers in patients with contraindications to such implantation. They can also be used in combination with S-ICD [14].

Despite the dynamic development of various types of electrotherapy, these modalities are not free from

problems and complications. One such complication, already mentioned above, is lead dysfunction. Another involves device sensitivity issues, such as oversensing or undersensing, both of which may result, in extreme cases, in lack of pacing or inappropriate arrhythmia detection. One such phenomenon is T-wave oversensing (TWOS), which may occur in conventional ICDs, S-ICDs, as well as pacemakers. It is characterized by the incorrect recognition of a T-wave as a ventricular depolarization, which may result in inappropriate ICD or S-ICD detection of tachyarrhythmia [15]. This situation can be triggered by various factors, including hyperkalemia or ischemia. In pacemakers, this may lead to pacing inhibition, whereas in ICDs – to inappropriate shocks [16,17].

Hyperkalemia affects up to 8% of hospitalized patients. Among cardiology patients, it most commonly occurs in elderly individuals treated for HF with concomitant renal failure. It is the most frequent electrolyte disturbance leading to dysfunction of implantable cardiac devices [18]. In the electrocardiographic recording, hyperkalemia manifests with tall, peaked T-waves, progressive flattening of P-waves, and widening of QRS complexes. When plasma potassium exceeds 7.0 mmol/L, cardiovascular manifestations such as atrioventricular blocks, asystole, ventricular tachycardia, or atrial fibrillation may occur. Individual patient sensitivity to elevated potassium levels may vary significantly [19].

CASE REPORT

A 36-year-old patient was transferred in May 2018 from a district hospital to a cardiology center due to decompensated heart failure in NYHA class III. The patient reported a progressive decline in exercise tolerance and exertional dyspnea over the preceding month. Medical history included chronic kidney disease requiring dialysis, secondary hyperthyroidism, and gastroesophageal reflux disease. He had not previously been under cardiology care.

Echocardiographic examination revealed severe impairment of left ventricular systolic function (LVEF = 23%) without significant valvular defects. Standard heart failure therapy was initiated. Endomyocardial biopsy confirmed chronic active myocarditis. Steroid therapy was commenced, resulting in improvement of contractility and hemodynamic stabilization. Holter ECG monitoring showed no sustained ventricular arrhythmias. Laboratory tests revealed persistently elevated creatinine levels, N-terminal pro-B-type natriuretic peptide (NT-proBNP) concentration above 35,000 pg/mL, and hyperkalemia (5.47–7.03 mmol/L). The estimated glomerular filtration rate (GFR) was approximately 7 mL/min/1.73 m².

Following clinical stabilization, the patient was discharged with a recommendation for rehospitalization in September 2018 for assessment of ICD implantation indications. Follow-up examinations demonstrated improvement of LVEF to 37%, absence of significant valvular pathology, and nocturnal sinus bradycardia episodes. No sustained ventricular arrhythmias were recorded. Due to improvement in left ventricular function, ICD implantation was not pursued.

In September 2023, the patient presented to the clinic for cardiovascular assessment and potential elective implantation of a subcutaneous implantable cardioverter-defibrillator (Boston Emblem MRI S-ICD). Left ventricular ejection fraction was 22%. The device, with an electrode positioned along the left sternal border, was implanted using the three-incision technique. The procedure was performed under general anesthesia without complications. Subsequent Holter ECG revealed 232 episodes of sinus bradycardia, with a minimum heart rate of 37 beats per minute. Given the history of syncope and bradycardia episodes, the patient was qualified for leadless pacemaker implantation. In January 2024, a 42-year-old man with chronic heart failure, left ventricular dilatation, and end-stage renal disease was admitted for leadless pacemaker implantation due to numerous bradycardia episodes. The procedure and hospitalization were uneventful, and device parameters were within normal limits. S-ICD function was also assessed as normal. The patient was discharged in good general condition.

In March 2024, the patient was electively admitted to the cardiology department for heart transplantation qualification. Echocardiography demonstrated LVEF of 25%, with no valvular defects. Oxygen consumption during cardiopulmonary exercise testing was 15.4 mL/kg/min, and the six-minute walk distance was 418 m. Considering the patient's overall condition, further qualification testing was discontinued.

In June 2024, the patient was urgently admitted following teletransmission confirming an S-ICD shock. Laboratory tests revealed hyperkalemia (7.21 mmol/L). Medical history confirmed that the patient had discontinued sodium polystyrene sulfonate (used to prevent hyperkalemia) several days earlier. ECG demonstrated tall, peaked T-waves. Echocardiographic examination showed no new abnormalities. Hemofiltration was initiated. Leadless pacemaker interrogation revealed no significant abnormalities or arrhythmias. S-ICD interrogation confirmed TWOS and inappropriate detection of ventricular tachycardia, which triggered a high-energy shock. After correction of electrolyte disturbances, no recurrence of TWOS was observed. Following stabilization of electrolyte

abnormalities, the patient was discharged. During one--year follow-up, TWOS did not recur, and the function of both devices remained normal.

DISCUSSION

Over the past decades, significant technological development has occurred in the field of implantable devices. An alternative to conventional transvenous systems has become the S-ICD, which does not require the insertion of electrodes into the heart chambers [20]. The S-ICD represents a particularly beneficial solution in younger patients, in whom the preservation of the venous system is important, as well as in patients with difficult vascular access or a high risk of infections associated with transvenous leads [20,21]. Modern ICDs are equipped with advanced arrhythmia detection algorithms that enable differentiation between supraventricular and ventricular rhythms. This significantly reduces the risk of inappropriate interventions [22].

Despite numerous advantages, implantable cardiac devices are not free from limitations and complications. One example of their malfunction is the possibility of inappropriate shock delivery associated with misinterpretation of T waves (TWOS), which may be caused by factors such as hyperkalemia or repolarization changes [23,24,25]. TWOS is a serious and potentially dangerous phenomenon occurring in patients with implantable cardiac devices [26]. The mechanism of TWOS consists of the device erroneously recognizing a physiological T wave as an independent ventricular impulse, which results in an overestimation of the detected heart rate and may lead to an incorrect diagnosis of ventricular arrhythmia [27,28]. Risk factors for TWOS include young patient age, high left ventricular ejection fraction, low QRS amplitude in lead I of the ECG, repolarization variability (e.g., due to hyperkalemia, physical exertion, or alcohol consumption), and diseases such as Brugada syndrome [29,30,31]. Cases have also been reported in which TWOS appeared only years after ICD implantation [30]. TWOS is one of the main causes of inappropriate ICD interventions, especially in subcutaneous systems (S-ICD) [32,33].

Management of TWOS is primarily based on adjustment of device settings. This may include changes in detection sensitivity, modification of device parameters, and ICD software updates to improve R- and T-wave discrimination [28,34]. In cases resistant to software modification, generator replacement may be considered. An important role is also played by the identification and correction of secondary causes, such as electrolyte disturbances, in particular hyperkalemia, which significantly alters T-wave morphology and may exacerbate oversensing [16,28,35].

In the literature, cases of TWOS in S-ICD have been reported for various reasons, including one similar to that described herein, namely in a female patient with chronic heart failure and end-stage renal disease, in whom hyperkalemia was the main factor triggering TWOS. Consequently, this led to inappropriate recognition of ventricular rhythm by the device and the delivery of high-energy therapy. In this case, the patient experienced symptoms of general clinical deterioration. As in the present case, the key element of treatment was the correction of electrolyte disturbances. However, additional reprogramming of the ICD was required [25].

CONCLUSIONS

1. TWOS, causing inappropriate S-ICD discharges, is a clinical problem that requires immediate medical intervention.
2. Hyperkalemia may be a cause of inappropriate discharges of implantable cardiac devices.
3. Correction of electrolyte disturbances allows for the limitation of TWOS episodes in S-ICD.

Authors' contribution

Study design – M. Krawiec, A. Hakało, A. Radziewicz, E. Jędrzejczyk-Patej
Data collection – M. Krawiec, A. Hakało, A. Radziewicz, E. Jędrzejczyk-Patej, B. Średniawa
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REFERENCES

1. Greene SJ, Bauersachs J, Brugts JJ, Ezekowitz JA, Lam CSP, Lund LH, et al. Worsening heart failure: nomenclature, epidemiology, and future directions: JACC review topic of the week. J Am Coll Cardiol. 2023;81(4):413–424. doi: 10.1016/j.jacc.2022.11.023.

2. Ziaeian B, Fonarow GC. Epidemiology and aetiology of heart failure. Nat Rev Cardiol. 2016;13(6):368–378. doi: 10.1038/nrcardio.2016.25.

3. Savarese G, Becher PM, Lund LH, Seferovic P, Rosano GMC, Coats AJS. Global burden of heart failure: a comprehensive and updated review of epidemiology. Cardiovasc Res. 2023;118(17):3272–3287. doi: 10.1093/cvr/cvac013.

4. Shams P, Malik A, Chhabra L. Congestive heart failure. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2025, https://www.ncbi.nlm.nih.gov/books/NBK430873/ [accessed on 13 June 2025].

5. Knap K. Niewydolność serca. Mp.pl, 19.07.2018 [online] https://www.mp.pl/pacjent/choroby-ukladu-krazenia/choroby/191351 [accessed on 15 May 2025].

6. Heidenreich PA, Bozkurt B, Aguilar D, Allen LA, Byun JJ, Colvin MM, et al.; ACC/AHA Joint Committee Members. 2022 AHA/ACC/HFSA Guideline for the Management of Heart Failure: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. Circulation. 2022;145(18):e895–e1032. doi: 10.1161/CIR.0000000000001063.

7. McDonagh TA, Metra M, Adamo M, Gardner RS, Baumbach A, Böhm M, et al. 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure. Eur Heart J. 2021;42(36):3599–3726. doi: 10.1093/eurheartj/ehab368.

8. King M, Kingery J, Casey B. Diagnosis and evaluation of heart failure. Am Fam Physician. 2012;85(12):1161–1168.

9. Gayat E, Arrigo M, Littnerova S, Sato N, Parenica J, Ishihara S, et al. Heart failure oral therapies at discharge are associated with better outcome in acute heart failure: a propensity-score matched study. Eur J Heart Fail. 2018;20(2):345–354. doi: 10.1002/ejhf.932.

10. Crespo-Leiro MG, Anker SD, Maggioni AP, Coats AJ, Filippatos G, Ruschitzka F, et al. European Society of Cardiology Heart Failure Long-Term Registry (ESC-HF-LT): 1-year follow-up outcomes and differences across regions. Eur J Heart Fail. 2016;18(6):613–625. doi: 10.1002/ejhf.566.

11. McMurray JJV, Packer M, Desai AS, Gong J, Lefkowitz MP, Rizkala AR, et al. Angiotensin-neprilysin inhibition versus enalapril in heart failure. N Engl J Med. 2014;371(11):993–1004. doi: 10.1056/NEJMoa1409077.

12. Iqbal AM, Butt N, Jamal SF. Automatic internal cardiac defibrillator. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2023, https://www.ncbi.nlm.nih.gov/books/NBK538341/ [accessed on 31 May 2025].

13. Vio R, Forlin E, Čulić V, Themistoclakis S, Proietti R, China P. Systematic Review on S-ICD Lead Extraction. J Clin Med. 2023;12(11): 3710. doi: 10.3390/jcm12113710.

14. Doldi F, Biller B, Reinke F, Eckardt L. New developments in leadless pacing systems. [Article in German]. Herz. 2021;46(6):513–519. doi: 10.1007/s00059-021-05075-6.

15. Francis J. T wave oversensing. Indian Pacing Electrophysiol J. 2010; 10(6):236–238.

16. Sun Y, Zhang P, Li X, Guo J. Inappropriate ICD discharge due to T-wave oversensing in a patient with short QT syndrome. Pacing Clin Electrophysiol. 2010;33(1):113–116. doi: 10.1111/j.1540-8159.2009.02538.x.

17. Cohen MI, Anagnostopoulos PV, Papez A. An unusual resolution of T-wave oversensing in an implantable cardioverter defibrillator in a child with long QT syndrome. J Interv Card Electrophysiol. 2009;25(3):235–238. doi: 10.1007/s10840-008-9353-8.

18. Barold SS, Herweg B. The effect of hyperkalaemia on cardiac rhythm devices. Europace. 2014;16(4):467–476. doi: 10.1093/europace/eut383.

19. Weiss JN, Qu Z, Shivkumar K. Electrophysiology of hypokalemia and hyperkalemia. Circ Arrhythm Electrophysiol. 2017;10(3):e004667. doi: 10.1161/CIRCEP.116.004667.

20. Knops RE, Olde Nordkamp LR, Delnoy PPH, Boersma LV, Kuschyk J, El-Chami MF, et al. Subcutaneous or transvenous defibrillator therapy. N Engl J Med. 2020;383(6):526–536. doi: 10.1056/NEJMoa1915932.

21. Kaya E, Rassaf T, Wakili R. Subcutaneous ICD: Current standards and future perspective. Int J Cardiol Heart Vasc. 2019;24:100409. doi: 10.1016/j.ijcha.2019.100409.

22. Bryant RM. How implantable cardioverter-defibrillators work and simple programming. Cardiol Young. 2017;27(S1):S121–S125. doi: 10.1017/S1047951116002353.

23. Implantable cardioverter-defibrillators (ICDs). Mayo Clinic [online] https://www.mayoclinic.org/tests-procedures/implantable-cardioverter-defibrillators/about/pac-20384692 [accessed on 13 June 2025].

24. Larbig R, Bettin M, Motloch LJ, Fischer A, Bode N, Frommeyer G, et al. Management of inappropriate shocks/T-wave-oversensing in S-ICD®--patients. [Article in German]. Herzschrittmacherther Elektrophysiol. 2018;29(1):122–126. doi: 10.1007/s00399-018-0555-1.

25. Gonzalez A, Franqui H, Lopez J, Banchs H. Hyperkalemia-Induced T Wave Oversensing in an Implantable Cardioverter Defibrillator. Cureus. 2024;16(2):e54135. doi: 10.7759/cureus.54135.

26. Sun YX, Gao J, Jiang CY, Xue YM, Xu YZ, Liu G, et al. T Wave Safety Margin during the Process of ICD Implantation As a Novel Predictor of T Wave Oversensing. Front Physiol. 2017;8:659. doi: 10.3389/fphys.2017.00659.

27. Adhikari S, Arisha MJ, Assal C. T-wave oversensing precipitating cardiogenic shock in a pacer-dependent patient. HeartRhythm Case Rep. 2023;9(10):728–731. doi: 10.1016/j.hrcr.2023.07.015.

28. Strik M, Ploux S, Eschalier R, Mondoly P, Fontagne L, Ramirez FD, et al. T-Wave Oversensing with Contemporary Implantable Cardioverter--Defibrillators. J Cardiovasc Dev Dis. 2023;10(10):430. doi: 10.3390/jcdd10100430.

29. El-Chami MF, Harbieh B, Levy M, Leon AR, Merchant FM. Clinical and electrocardiographic predictors of T wave oversensing in patients with subcutaneous ICD. J Arrhythm. 2016;32(3):181–185. doi: 10.1016/j.joa.2016.01.002.

30. Topf A, Motloch LJ, Kraus J, Danmayr F, Mirna M, Schernthaner C, et al. Exercise-related T-wave oversensing: an underestimated cause of reduced exercise capacity in a pacemaker-dependent patient-a case report and review of the literature. J Interv Card Electrophysiol. 2020;59(1):67–70. doi: 10.1007/s10840-019-00698-6.

31. Rasania SP, Mountantonakis S, Patel VV. Inappropriate ICD shocks caused by T-wave oversensing due to acute alcohol intoxication. Pacing Clin Electrophysiol. 2012;35(9):e267–e271. doi: 10.1111/j.1540-8159.2012.03348.x.

32. Konishi S, Minamiguchi H, Ozu K, Mizuno H, Hikoso S, Yamaguchi O, et al. Routine exercise testing could not predict T-wave oversensing in a patient after a subcutaneous implantable cardioverter-defibrillator implant. Clin Case Rep. 2017;6(2):309–313. doi: 10.1002/ccr3.1345.

33. Ochman AR, Riesbeck M, Corbisiero M. Inappropriate shock caused by P- and T-wave oversensing in a subcutaneous implantable cardiac defibrillator in a patient in sinus rhythm. HeartRhythm Case Rep. 2020;7(7):433–435. doi: 10.1016/j.hrcr.2020.11.022.

34. Rodríguez-Mañero M, de Asmundis C, Sacher F, Arbelo E, Probst V, Castro-Hevia J, et al. T-wave oversensing in patients with Brugada syndrome: true bipolar versus integrated bipolar implantable cardioverter defibrillator leads: multicenter retrospective study. Circ Arrhythm Electrophysiol. 2015;8(4):792–798. doi: 10.1161/CIRCEP.115.002871.

35. Nathan K, Kamel O, Marks D, Russo AM. Treatment of symptomatic bradycardia due to T-wave oversensing with implantation of a new generator incorporating delayed decay and threshold start sensitization algorithms. HeartRhythm Case Rep. 2020;6(12):891–895. doi: 10.1016/j.hrcr.2020.06.024.