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Pacemaker Syndrome Presenting as Acute Decompensated Heart Failure After an ERI-Associated Mode Change: A Case Report

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Abstract

Background: Pacemaker syndrome is a recognized complication of atrioventricular (AV) dyssynchrony and may occur in patients with dual-chamber pacemakers following automatic mode changes associated with battery depletion.

Case Presentation: A 75-year-old woman with a dual-chamber pacemaker implanted for carotid sinus hypersensitivity presented with acute decompensated heart failure, acute kidney injury, and newly developed massive tricuspid regurgitation. Device interrogation revealed that the pacemaker had reached recommended replacement time (RRT), corresponding to the elective replacement indicator (ERI) state, resulting in automatic transition to ventricular-only pacing with loss of AV synchrony. Following generator replacement and restoration of dual-chamber pacing, the patient experienced rapid improvement in symptoms, renal function, and tricuspid regurgitation severity.

Discussion: This case demonstrates that ERI-associated loss of AV synchrony can precipitate acute heart failure, cardiorenal dysfunction, and reversible functional tricuspid regurgitation despite preserved ventricular systolic function. It underscores the importance of considering pacemaker syndrome in patients with implanted devices who present with otherwise unexplained clinical deterioration and highlights the value of prompt device interrogation.

Conclusion: Early recognition of ERI-associated pacemaker syndrome and timely generator replacement can restore AV synchrony and lead to rapid reversal of hemodynamic compromise and clinical recovery.

Keywords: Pacemaker syndrome; Dual-chamber pacemaker; Atrioventricular synchrony; Recommended replacement time (RRT); Tricuspid regurgitation; Acute decompensated heart failure; Acute kidney injury

Introduction

Pacemaker syndrome is a constellation of clinical symptoms and hemodynamic disturbances resulting from loss of optimal atrioventricular (AV) synchrony in patients with implanted pacemakers. It is classically associated with single-chamber ventricular pacing but may also occur in patients with dual-chamber systems when AV synchrony is disrupted by device programming or automatic mode changes.

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Clinical manifestations range from fatigue, dizziness, and exercise intolerance to hypotension, dyspnea, systemic congestion, and heart failure. Because these manifestations may overlap with other cardiovascular conditions, pacemaker syndrome can be underrecognized, particularly in patients with multiple comorbidities [1,2].

Carotid sinus hypersensitivity is an established indication for permanent pacemaker implantation in selected patients, particularly when associated with cardioinhibitory responses and recurrent syncope [3]. Dual-chamber pacing is generally used to preserve AV synchrony and maintain the atrial contribution to ventricular filling. However, as pacemakers approach battery depletion, they may enter a manufacturer-specific elective replacement or recommended replacement state, during which automatic changes in pacing mode may occur. In susceptible patients, this loss of AV synchrony may result in clinically significant pacemaker syndrome [4].

We report the case of a 75-year-old woman with a dual-chamber Enura DR MRI pacemaker implanted in 2014 for carotid sinus hypersensitivity who presented with acute decompensated heart failure, acute kidney injury, and newly developed massive tricuspid regurgitation. Device interrogation demonstrated that the pacemaker had reached recommended replacement time (RRT), with loss of dual-chamber pacing and ventricular-only pacing accompanied by retrograde atrial activation. The temporal relationship between the device mode change, clinical deterioration, and subsequent improvement following generator replacement highlights the importance of device interrogation in patients with implanted pacemakers who present with otherwise unexplained heart failure.

Case Presentation

A 75-year-old woman with a medical history of hypertension, chronic obstructive pulmonary disease (COPD), type 2 diabetes mellitus, and carotid sinus hypersensitivity status post dual-chamber pacemaker implantation presented to the hospital on March 8, 2025, with progressively worsening shortness of breath. Her symptoms had begun approximately two weeks earlier and had gradually progressed to dyspnea at rest during the three days preceding presentation. She was admitted to the coronary care unit for further evaluation.

The patient had previously been hospitalized on February 23, 2025, for clinical volume overload attributed to decompensated heart failure. Her labs at that time showed creatinine of 1.21 mg/dL and N-terminal pro-B-type natriuretic peptide (NT-proBNP) of 10,088 pg/mL. A transthoracic echocardiogram (TTE) was done on February 24 and showed normal RV and LV systolic function with an estimated LVEF 65-69%, grade 2 diastolic dysfunction, mild TR, with mildly elevated pulmonary arterial systolic pressure. PSAP: 42 mmHg, with RAP estimated at 5 mmHg. Her diastolic parameters showed LA volume of 54.16 ml, E/E' mean 7.52, E' velocity 8.48 cm/s, LV mass 75.9 g/m2. She was discharged on furosemide 20 mg once daily, which was subsequently increased to twice daily after few days because of worsening dyspnea.

On presentation, her vital signs showed: blood pressure 118/78 mmHg, temperature 37 degrees, pulse 90 bpm, respiratory rate 12 per minute, SpO2 100%. Physical examination revealed bilateral pulmonary crackles and 1+ bilateral lower-extremity edema. Laboratory investigations demonstrated a creatinine level of 1.46 mg/dL compared with a baseline of approximately 1.1 mg/dL, consistent with acute kidney injury. Additional laboratory findings included a blood urea nitrogen level of 31 mg/dL, NT-proBNP of 12,227 pg/mL, and C-reactive protein of 7.1 mg/L. Troponin was not elevated. Spot urine studies showed Albuminuria of 35 mg/L, urine creatinine 43.8 mg/dL, Albumin/Creatinine 79.7 mg/g, protein/creatinine 320 mg/g.

A 12-lead electrocardiogram demonstrated ventricular-paced rhythm with consistent ventricular pacing spikes preceding each QRS complex. Her ventricular rate was 65 bpm, and QRS duration was 157 ms. Small atrial deflections compatible with retrograde P waves were observed following each paced ventricular complex with a fixed ventriculoatrial relationship, consistent with preserved retrograde ventriculoatrial conduction (Figure 1).

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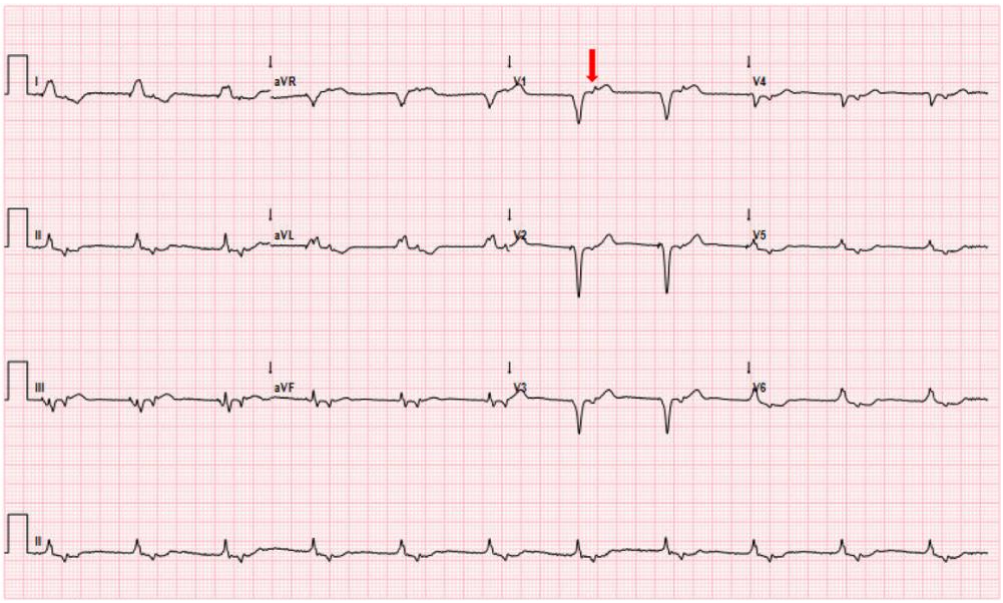

Figure 1: Twelve-lead electrocardiogram demonstrating ventricular-paced rhythm. Black arrows indicate ventricular pacing spikes preceding each paced QRS complex. Red arrows indicate retrograde P waves occurring after the paced QRS complexes, consistent with preserved retrograde ventriculoatrial conduction.

A TTE performed on March 10, 2025, demonstrated preserved left and right ventricular systolic function, with a left ventricular ejection fraction of 60–64%, but newly developed massive tricuspid regurgitation (TR) (Figure 2). Those parameters were measured- Tricuspid ERO area: 0.42 cm2, regurgitant volume: 27 ml, mildly elevated pulmonary arterial systolic pressure, PASP: 44 mmHg, with RAP estimated at 20 mmHg. This represented a marked change from a transthoracic echocardiogram performed on February 24, 2025, which demonstrated only mild TR.

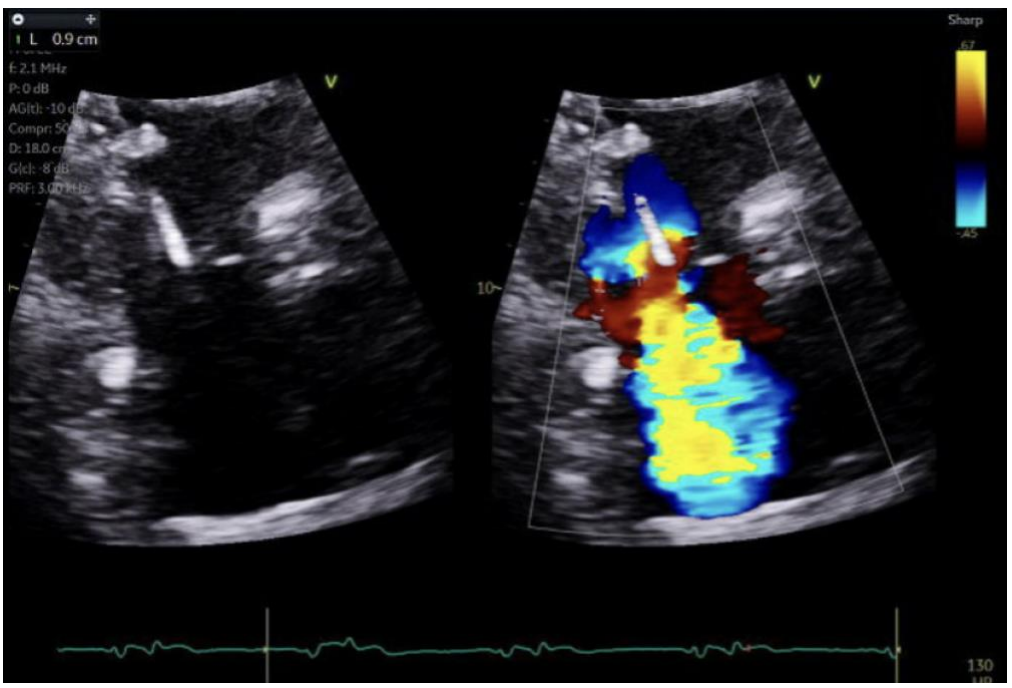

Figure 2: Massive TR on TTE.

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Subsequently, a transesophageal echocardiography (TEE) was done and showed massive TR with the cardiac device lead restricting the septal leaflet.

Given the abrupt change in TR and the patient's clinical deterioration, both pulmonology and cardiology were consulted. Pulmonology did not consider the patient's stable COPD and bronchiectasis sufficient to explain the acute clinical deterioration. Cardiology subsequently performed pacemaker interrogation. The patient had a dual-chamber Enura DR MRI pacemaker that had been implanted in 2014 for carotid sinus hypersensitivity. Prior to the current episode, the pacemaker had been programmed in DDD mode. Device interrogation demonstrated that the generator had reached RRT, corresponding to the elective replacement indicator (ERI) state, because of battery depletion. The device had subsequently transitioned to ventricular-only pacing, resulting in loss of AV synchrony. The presence of retrograde P waves on the presenting electrocardiogram indicated preserved ventriculoatrial conduction. Together, these findings were consistent with ERI-associated loss of AV synchrony and pacemaker syndrome.

Nephrology was consulted for evaluation of the acute kidney injury. Urine studies demonstrated a fractional excretion of urea of 46%, raising the possibility of an intrinsic renal component. However, in the setting of marked volume overload and acute cardiac decompensation, the renal dysfunction was considered multifactorial, with cardiorenal physiology and venous congestion likely contributing.

The patient was treated with diuretic therapy, but renal function continued to deteriorate, with serum creatinine increasing to 1.86 mg/dL on March 12, 2025(Peak value). Given the device findings and clinical presentation, generator replacement was planned. The pacemaker generator was replaced on March 14, 2025, with restoration of dual-chamber pacing and AV synchrony.

Beginning the day after generator replacement, renal function improved, and serum creatinine returned to approximately baseline within several days. A repeat transthoracic echocardiogram performed on March 16, 2025, demonstrated improvement in TR severity from massive to moderate. Given the concomitant diuretic therapy, the improvement in TR was considered likely multifactorial, although restoration of AV synchrony was temporally associated with the improvement.

The patient was discharged on March 16, 2025, on furosemide 40 mg orally once daily. She reported marked improvement in dyspnea and energy following generator replacement.

Discussion

Pacemaker syndrome is characterized by symptoms and hemodynamic abnormalities resulting from impaired AV synchrony. Although it is most commonly recognized in patients with single-chamber ventricular pacing, it may also develop in patients with dual-chamber pacemakers when appropriate AV synchrony is lost [5,6].

In the present case, the patient's pacemaker had been implanted in 2014 and programmed in DDD mode to provide coordinated atrial and ventricular pacing. During the current admission, interrogation demonstrated that the generator had reached recommended replacement time (RRT), resulting in a change from dual-chamber pacing to ventricular-only pacing. Importantly, the device had not simply become nonfunctional. Rather, the clinical deterioration occurred in the setting of an expected device behavior associated with battery depletion and RRT. The presenting electrocardiogram demonstrated ventricular pacing with retrograde P waves, indicating preserved ventriculoatrial conduction.

The combination of ventricular-only pacing and intact retrograde ventriculoatrial conduction can result in marked AV dyssynchrony. Retrograde atrial activation may occur during or shortly after ventricular systole, when the atrioventricular valves are closed. This can impair effective atrial contribution to ventricular filling, increase atrial and systemic venous pressures, reduce forward cardiac output, and produce the clinical manifestations of pacemaker syndrome [5,6]. In this patient, the temporal association between the transition to ventricular-only pacing, development of heart failure symptoms, and subsequent improvement following generator replacement strongly supports AV dyssynchrony as an important precipitating factor.

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The development of massive TR was a particularly notable feature of this presentation, especially that all the echocardiography images were done at the same lab at AUBMC and under same condition. The patient had only mild TR on echocardiography approximately two weeks before presentation, whereas the repeat study demonstrated massive TR with preserved biventricular systolic function. Mechanical lead-related tricuspid regurgitation remains an important explanation in patients with trans-tricuspid pacing leads, especially that the TEE done on the patient showed that the cardiac device lead is restricting the septal leaflet. However, the abrupt progression from mild to massive TR over approximately two weeks argues against slowly progressive chronic leaflet injury as the sole mechanism. Therefore, the temporal association between ERI-associated loss of AV synchrony, acute decompensation, and subsequent improvement following restoration of dual-chamber pacing suggests a significant functional component, with a possible contribution from lead-related TR. The acute change may have reflected a predominantly functional process related to altered right-sided hemodynamics, AV dyssynchrony, and systemic venous congestion. The subsequent improvement from massive to moderate TR following generator replacement further supports a potentially reversible functional component. However, because the patient also received diuretic therapy during this period, improvement in TR cannot be attributed exclusively to restoration of AV synchrony.

An additional important consideration is the possibility of underlying heart failure with preserved ejection fraction (HFpEF). The patient's age, hypertension, diabetes, and prior episode of volume overload could indicate pre-existing susceptibility to HFpEF and progressive TR. Therefore, an underlying cardiac substrate cannot be excluded. Nevertheless, the abrupt clinical deterioration, newly developed severe TR, device interrogation demonstrating ERI-associated ventricular-only pacing, presence of retrograde atrial activation, and subsequent improvement after generator replacement support loss of AV synchrony as an important precipitating factor in the acute decompensation.

The patient's acute kidney injury was also likely multifactorial. Cardiorenal syndrome in acute heart failure is mediated not only by reduced renal arterial perfusion but also by increased renal venous pressure and systemic venous congestion. In this patient, the marked volume overload and right-sided hemodynamic abnormalities provide a plausible mechanism for the observed renal dysfunction. The improvement in renal function shortly after restoration of dual-chamber pacing and improvement in cardiac congestion supports a relationship between the acute cardiac deterioration and renal impairment, although a direct causal relationship between pacemaker syndrome and AKI cannot be established from a single case.

Several alternative explanations were considered. Lead malfunction, including lead dislodgement or fracture, was not identified on device interrogation. Device-related infection was not supported by the absence of fever, local device findings, or significant inflammatory abnormalities. The patient's pulmonary disease was stable and was not considered sufficient to account for the abrupt cardiac and echocardiographic changes. Underlying HFpEF remains a possible contributing factor, as discussed above.

This case also highlights the importance of directly interrogating pacemakers in patients presenting with unexplained heart failure. Device-related causes may be overlooked when attention is focused on more common etiologies of dyspnea and volume overload. Importantly, a pacemaker reaching RRT/ERI does not necessarily represent complete device failure. Depending on the device and manufacturer, automatic changes in pacing mode may occur, potentially resulting in clinically important loss of AV synchrony. Recognition of this mechanism requires correlation of the patient's symptoms and electrocardiographic findings with device interrogation.

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Previous reports have described pacemaker syndrome associated with ERI-related changes from dual-chamber to ventricular pacing and subsequent improvement following generator replacement [10,11]. Other reports have described severe TR and cardiorenal manifestations in association with pacemaker syndrome [11]. The present case adds to this literature by illustrating the combination of ERI-associated AV dyssynchrony, abrupt worsening of TR, acute decompensated heart failure, and acute kidney injury in a patient with preserved biventricular systolic function. Rather than representing a previously undescribed mechanism, the case emphasizes the potential severity and reversibility of an established device-related phenomenon.

The principal treatment for pacemaker syndrome caused by inappropriate loss of AV synchrony is correction of the underlying pacing abnormality. In this patient, generator replacement restored dual-chamber pacing and was followed by improvement in symptoms, renal function, and TR severity. More physiologic pacing strategies, including His-bundle or left bundle branch area pacing, may be considered in selected patients with persistent symptoms or other indications for pacing optimization; however, such strategies were not required in this case [5,6].

Conclusion

Pacemaker syndrome can occur in patients with dual-chamber pacemakers when AV synchrony is disrupted, including during automatic mode changes associated with generator battery depletion and ERI. In susceptible patients, this may present as acute decompensated heart failure with systemic congestion, worsening tricuspid regurgitation, and cardiorenal dysfunction. Device interrogation is therefore an essential component of evaluating unexplained heart failure in patients with implanted pacemakers. Recognition of ERI-associated loss of AV synchrony and timely generator replacement may lead to rapid clinical improvement and improvement in the associated hemodynamic complications.

Informed Consent

Written informed consent was obtained from the patient for publication of this case report and any accompanying clinical images. The patient was informed that identifying information would be omitted to protect confidentiality.

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