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Case Report

Pacemaker syndrome mimicking heart failure in a DDD-paced patient: A case report

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Abstract

Introduction: Pacemaker syndrome is a clinical condition caused by loss of Atrioventricular (AV) synchrony, often associated with single-chamber pacing but also possible in dual-chamber systems due to device malfunction.

Case presentation: A 75-year-old woman with a history of DDD pacemaker implantation for carotid sinus hypersensitivity presented with worsening dyspnea, volume overload, and acute kidney injury. Echocardiography showed new-onset massive tricuspid regurgitation. Pacemaker interrogation revealed loss of atrial pacing due to battery depletion. After pacemaker box replacement, her symptoms and renal function improved, and tricuspid regurgitation decreased to moderate.

Discussion: This case highlights the potential for pacemaker syndrome in dual-chamber devices, particularly when battery depletion disrupts AV synchrony. The syndrome may mimic heart failure and contribute to cardiorenal complications.

Conclusion: In patients with pacemakers and unexplained volume overload or renal dysfunction, pacemaker syndrome should be considered. Prompt device evaluation can lead to full clinical recovery.

Keywords: Pacemaker; Heart failure; Battery depletion; Acute kidney injury; Dual chamber; Volume overload.

Introduction

Pacemaker syndrome is a constellation of symptoms resulting from suboptimal Atrioventricular (AV) or interventricular synchrony in patients with implanted pacemakers. It is most commonly observed in patients with single-chamber ventricular pacing but may also occur in dual-chamber systems, especially with device malfunction or programming issues [1]. Clinical manifestations can range from mild fatigue and dizziness to more severe presentations such as hypotension, syncope, and even heart failure. These symptoms may mimic other cardiovascular conditions, making the diagnosis of pacemaker syndrome clinically challenging and often underrecognized [2].

Carotid sinus hypersensitivity is a known indication for permanent pacemaker implantation, particularly in older adults, where excessive vagal tone may result in bradycardia and syncope [3]. Dual-chamber pacing (DDD mode) is generally considered optimal for maintaining AV synchrony in these patients. However, pacemaker-related complications can still arise over time, particularly as devices near battery depletion, leading to inappropriate pacing modes or loss of synchrony [4].

We report the case of a 75-year-old woman with a previously implanted DDD pacemaker for carotid sinus hypersensitivity, who presented with signs of volume overload and acute kidney injury. She was found to have new-onset massive tricuspid regurgitation and pacemaker desynchrony, consistent with pacemaker syndrome, which resolved following battery replacement.

Case presentation

A 75-year-old woman known to have hypertension, Chronic Obstructive Pulmonary Disease (COPD), diabetes mellitus type II, carotid hypersensitivity status post dual-chamber pacemaker implantation several years ago presenting to the hospital on March 8, 2025 for shortness of breath of two weeks duration that was gradually worsening over time to become at rest over the last 3 days prior to presentation. She was admitted to the Coronary Care Unit (CCU) for workup and evaluation.

The patient had been hospitalized on February 23, 2025 for signs of volume overload due to decompensated heart failure and was discharged at that time on furosemide 20 mg daily. This dose was increased to two times daily after the increased shortness of breath.

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Upon presentation, vital signs were normal without desaturation, physical examination revealed bilateral lung crackles with plus 1 lower extremity edema but otherwise other physical findings were normal. Laboratory investigations on admission showed a creatinine of 1.46 mg/dL (baseline: 1.1 mg/dL), meeting criteria for acute kidney injury (AKI). Other findings included a BUN of 31 mg/dL, Pro-BNP of 12,227 pg/mL, and a CRP of 7.1 mg/L; troponin was not elevated. An EKG was done and it showed ventricular pacing, and retrograde non paced p waves (Figure 1).

A transthoracic echocardiogram performed on March 10, 2025, revealed normal right and left ventricular systolic function with a Left Ventricular Ejection Fraction (LVEF) of 60–64%, and newly developed massive Tricuspid Regurgitation (TR) (Figure 2). Compared to a prior echocardiogram on February 24, 2025, which showed only mild TR, this was a significant new finding. Both pulmonology and cardiology teams were consulted to evaluate the etiology of the abrupt TR progression. Pulmonology deemed the change unrelated to her stable COPD and bronchiectasis. Cardiology proceeded with pacemaker interrogation, which revealed ERI mode (Elective Replacement Indicator), and a loss of atrial pacing with ventricular-only pacing, most likely due to battery depletion.

Nephrology was consulted for further evaluation of AKI. Urine studies revealed a fractional excretion of urea of 46%, suggesting possible intrinsic renal cause. The renal impairment was attributed to a cardiorenal mechanism, likely related to volume overload and impaired cardiac output.

The patient was treated with diuretics, and a plan was made to proceed with pacemaker box replacement. Despite medical management, her creatinine continued to rise, reaching 1.86 mg/dL on March 12, 2025. She underwent pacemaker box change on March 14, 2025, with close post-procedure monitoring. From the day following the intervention, her renal function began to improve, and within a few days, her creatinine returned to baseline. A repeat echocardiogram on March 16, 2025, showed an improvement in TR severity from massive to moderate. The patient was discharged the same day on furosemide 40 mg orally once daily.

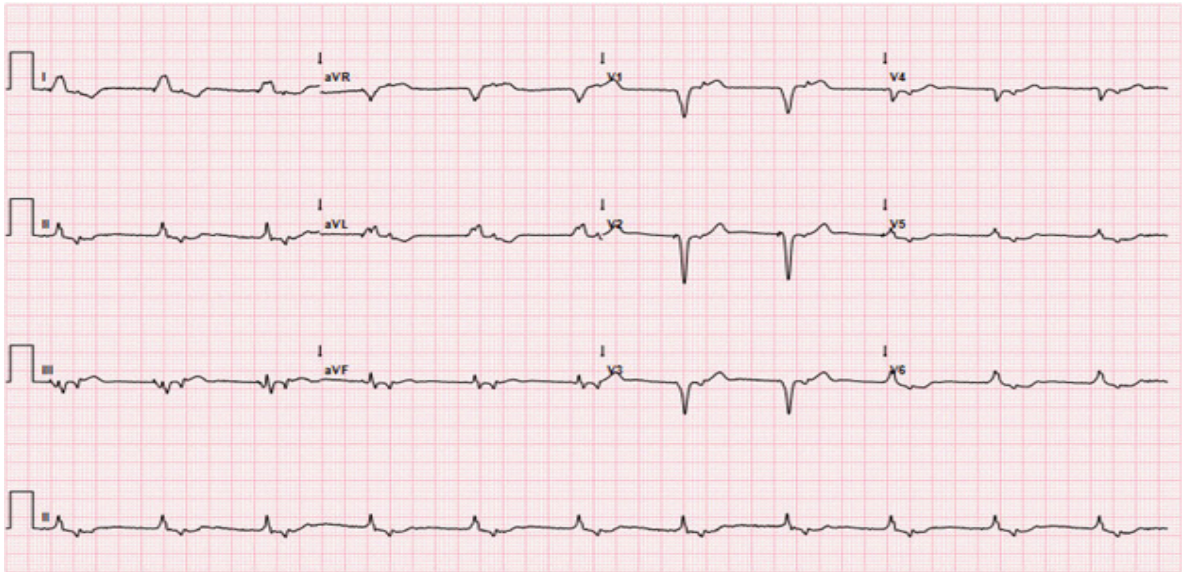

Figure 1: EKG upon presentation. Black arrow: Ventricular pacing. Red arrow: Retrograde P waves.

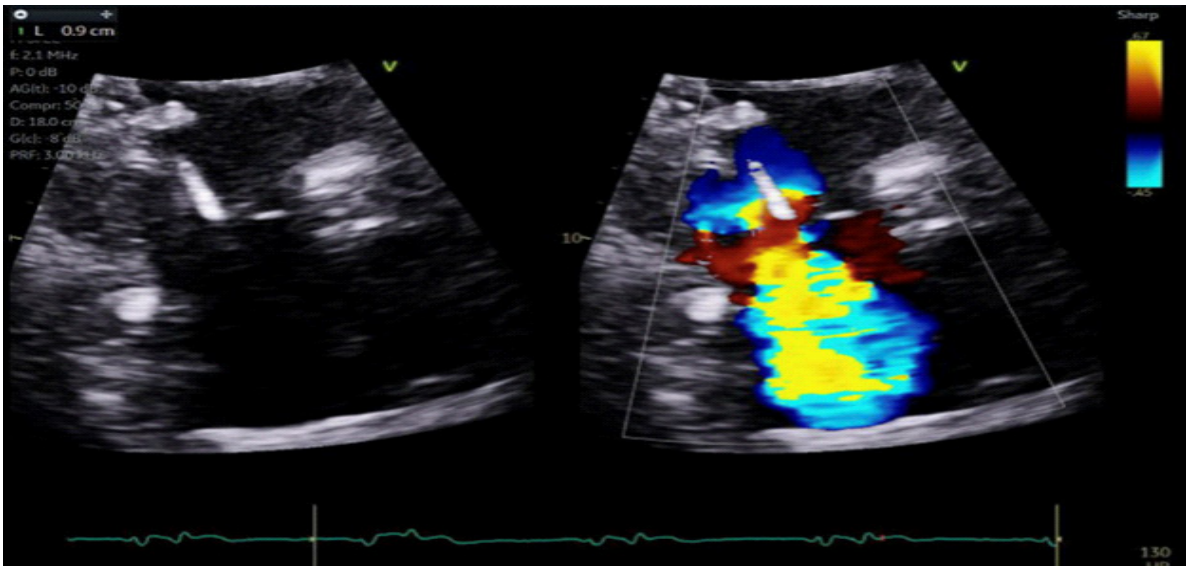

Figure 2: Massive TR on TTE.

Discussion

Pacemaker syndrome is a clinical entity characterized by a constellation of symptoms and hemodynamic disturbances resulting from loss of Atrioventricular (AV) synchrony, which impairs cardiac output and contributes to systemic congestion [5]. Although it is traditionally associated with single-chamber Right Ventricular (RV) pacing, it can also occur in patients with dual-chamber pacemakers if the AV synchrony mechanism is disrupted-such as in cases of battery depletion, lead malfunction, or device programming issues [6].

In this case, the patient had a dual-chamber pacemaker intended to coordinate atrial and ventricular contraction, preserving both the atrial contribution to ventricular filling and overall cardiac efficiency. However, battery exhaustion rendered the device non-functional, effectively reverting the heart to an unsynchronized rhythm, likely with ventricular escape beats or intrinsic conduction that was not coordinated with atrial activity [5]. This situation leads to mistimed atrial contractions-often occurring during ventricular systole when the AV valves are closed-which results in elevated atrial and venous pressures, decreased stroke volume, and retrograde flow into the systemic and pulmonary venous systems [6]. Repeated atrial contractions against a closed tricuspid valve, along with mechanical trauma from right ventricular pacing leads, can also lead to tricuspid annular dilation or leaflet damage, contributing to or exacerbating massive tricuspid regurgitation [7].

The patient's presentation with volume overload-clinically manifesting as peripheral congestion-is a hallmark complication of this impaired hemodynamic state. Chronic or acute venous congestion due to ineffective forward flow not only worsens heart failure symptoms but also contributes to renal dysfunction, as seen in this case with Acute Kidney Injury (AKI). The pathophysiology of AKI in the context of pacemaker syndrome is likely multifactorial, involving both decreased renal perfusion from low cardiac output and elevated renal venous pressure, which reduces glomerular filtration. This phenomenon parallels the pathogenesis of cardiorenal syndrome, a well-recognized consequence of heart failure-related volume overload [8].

This case also raises the importance of differential diagnosis in pacemaker patients presenting with new symptoms: Lead malfunction (e.g., dislodgement, fracture) was ruled out via interrogation. Autonomic dysfunction was less likely given structural abnormalities. Infection, such as device-related endocarditis, was excluded clinically and by normal inflammatory markers and absence of fever or local signs.

While pacemaker syndrome is often underdiagnosed, it is clinically important to recognize it in the setting of device malfunction, especially when symptoms such as fatigue, orthopnea, hypotension, and AKI emerge without another clear etiology. Notably, even in functioning dual-chamber systems, emerging data have shown that ventricular desynchrony-independent of AV timing-can also contribute to pacemaker syndrome-like symptoms, highlighting the broader relevance of maintaining both AV and Interventricular (VV) synchrony [9].

Treatment of pacemaker syndrome involves addressing the root cause of desynchrony. In this case, battery depletion was the precipitating factor, and timely pacemaker battery replacement is critical to restore AV synchrony and improve hemodynamics [6]. Following restoration of pacing, symptoms typically resolve quickly, renal function improves, and volume status stabilizes. In patients with persistent symptoms or suboptimal device settings, upgrading to more physiologic pacing strategies, such as His-bundle or biventricular pacing, may be warranted to further enhance cardiac function [5].

Patient perspective: The patient expressed that she had been feeling increasingly fatigued and short of breath without understanding the cause. She noted a dramatic improvement in her breathing and energy levels following pacemaker replacement, stating, "I was feeling worse every day and didn't know why. After they changed the pacemaker battery, I could breathe better and my energy came back. I'm thankful it was something fixable."

Conclusion

Pacemaker syndrome can occur in dual-chamber devices when AV synchrony is lost, especially due to battery depletion. It may mimic acute heart failure and contribute to secondary complications like acute kidney injury. Early recognition through device interrogation is essential for accurate diagnosis. Timely correction can lead to rapid and complete clinical improvement.

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