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Case Report

A case of cardiogenic shock due to ventricular dyssynchrony resolved by atrial pacing

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ABSTRACT

We present a case of a man with ischemic cardiomyopathy and single chamber implantable cardioverter-defibrillator who developed sinus arrest creating sudden dependence on right ventricular (RV) pacing. He presented with cardiogenic shock secondary to abrupt onset ventricular dyssynchrony from RV pacing, which required emergent stabilization and completely resolved with atrial pacing.

Learning objective: To establish a basic understanding of cardiogenic shock management. To reinforce the adverse effects associated with right ventricular pacing.

Introduction

Chronic right ventricular (RV) pacing is an established risk factor for the development of left ventricular systolic dysfunction or heart failure [1]. Typically, heart failure induced by RV pacing is considered a chronic phenomenon and is sometimes referred to as pacing-induced cardiomyopathy [1]. However, recent studies have highlighted the possibility of acute decompensated heart failure and cardiogenic shock as a result of immediate RV pacing [2]. Herein, we present a case that reveals the possible role of RV pacing on clinical decompensation and worsening shock, as well as the importance of atrial pacing as a solution.

Case report

A 65-year-old man with a history of ventricular tachycardia and single chamber implantable cardioverter defibrillator (SC-ICD) presented to the emergency department with progressively worsening shortness of breath, chest pain, and orthopnea over the previous week. He additionally reported firing of his ICD the day before. His past medical history was significant for heart failure with reduced ejection fraction (HFrEF) secondary to ischemic cardiomyopathy, coronary artery disease with two-vessel coronary artery bypass grafting to the left anterior descending artery and obtuse marginal arteries, chronic obstructive pulmonary disease, diabetes mellitus, and chronic kidney disease.

On arrival, he was hypertensive, 165/145 mmHg, and bradycardic, 46 beats per minute (bpm). While being evaluated, he quickly became hypotensive to 80/50 mmHg. Examination was notable for labored breathing and tripod positioning in addition to significant jugular venous distention, bibasilar crackles, and cold extremities with decreased peripheral pulses. He was placed on bi-level positive airway pressure and started on intravenous (IV) epinephrine. At this time, differential diagnoses included cardiogenic shock, septic shock, takotsubo cardiomyopathy, and pulmonary embolism.

Laboratory findings were significant for lactic acidosis of 11.0 mmol/L, serum bicarbonate of 14 mmol/L, and pH 7.06. Serum creatinine was increased to 3.69 mg/dL from a baseline of 2 mg/dL. Aspartate transaminase and alanine transaminase levels were elevated to 3207 IU/L and 1409 IU/L respectively. B-type natriuretic peptide was 818 pg/mL (normal <100 pg/mL) and initial high-sensitivity troponin I was 210 pg/mL. Chest X-ray showed cardiomegaly and pulmonary edema. Electrocardiogram (ECG) showed non-specific T-wave inversions. The patient's transthoracic echocardiogram one month prior to admission showed a left ventricular ejection fraction (LVEF) of 30–35 % with akinesis of the inferior and inferolateral left ventricular (LV) segments along with moderate diastolic dysfunction (Fig. 1).

After being initiated on IV epinephrine, the patient's blood pressure improved, however he remained bradycardic. He developed complete sinus arrest and was entirely ventricularly paced at 40 bpm on his ECG. The pacing rate of his ICD was increased to 90 bpm for presumed

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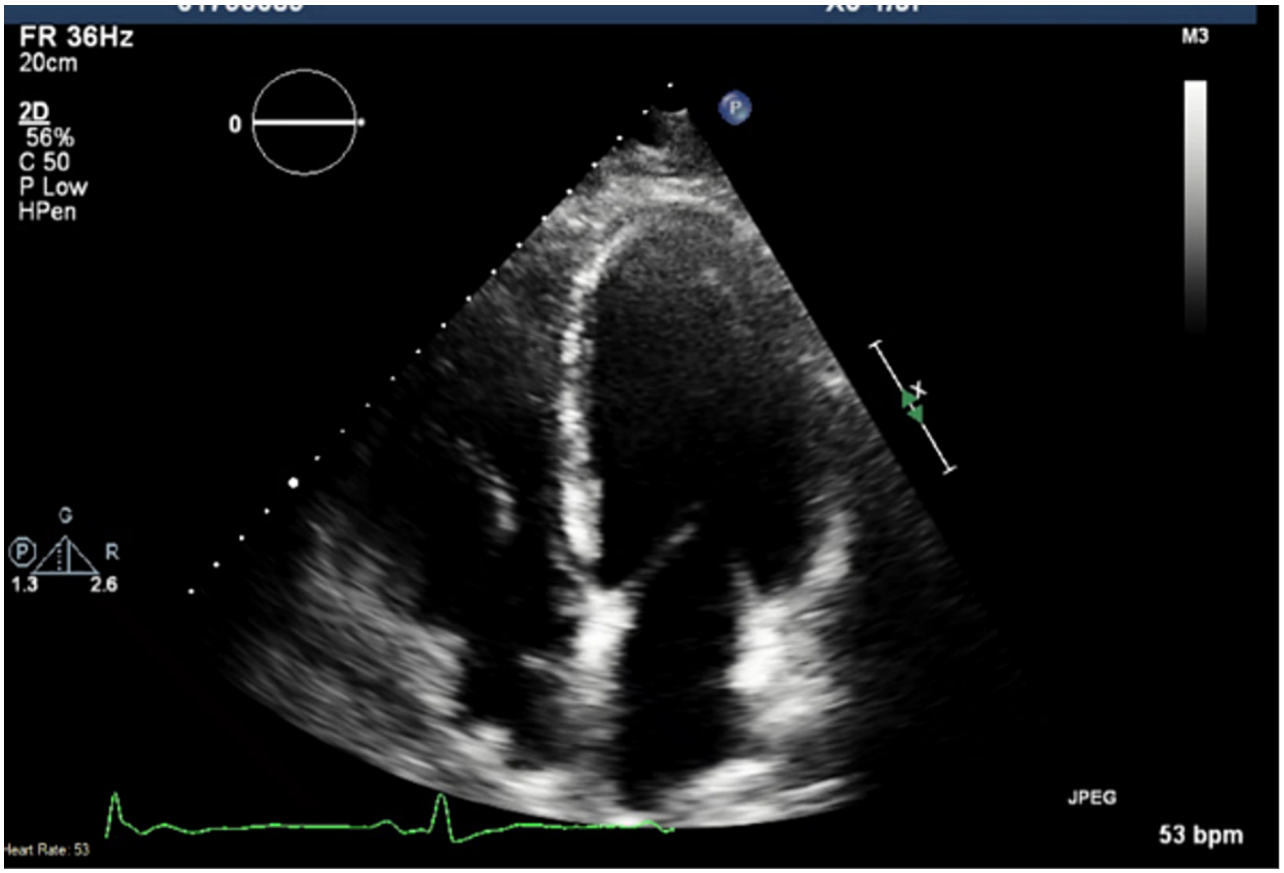

Fig. 1. Echocardiogram with inferior and inferolateral wall akinesis.

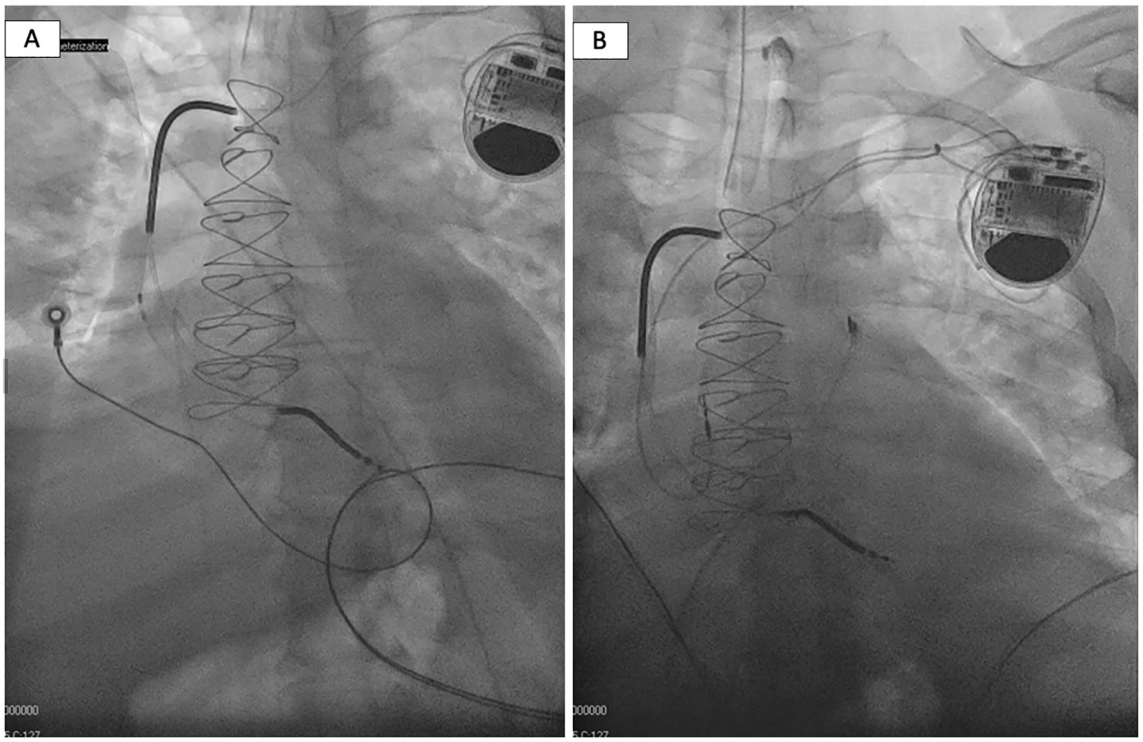

Fig. 2. (A) Temporary pacemaker was placed in the right atrium, which improved clinical condition. (B) Permanent right atrial lead added, and new dual chamber implantable cardioverter-defibrillator placed.

hemodynamically unstable bradycardia. In addition to causing the patient to feel subjectively worse, he immediately developed worsening hypotension at the higher pacing rate. The hemodynamic deterioration at higher pacing rates led to suspicion that he had severe intolerance to ventricular dyssynchrony created by RV pacing. Following an interdisciplinary discussion within the cardiology sub-specialty teams, a decision was made to take the patient emergently to the cardiac catheterization laboratory for right heart catheterization (RHC) and possible atrial pacing. The patient was intubated due to worsening acute hypoxic respiratory failure prior to transport.

RHC was performed with findings of significantly elevated biventricular filling pressures, mean pulmonary artery (PA) pressure, and pulmonary capillary wedge pressures consistent with cardiogenic shock. Atrial pacing was performed via a temporary pacemaker wire at a rate of 80 bpm which resulted in immediate and profound hemodynamic improvement; mean arterial pressure increased by 30 mmHg (Fig. 2A). A leave-in PA catheter was placed for close hemodynamic monitoring and a right-sided femoral intra-aortic balloon pump (IABP) was introduced for circulatory support. Coronary angiogram was not performed due to severe renal dysfunction and low suspicion

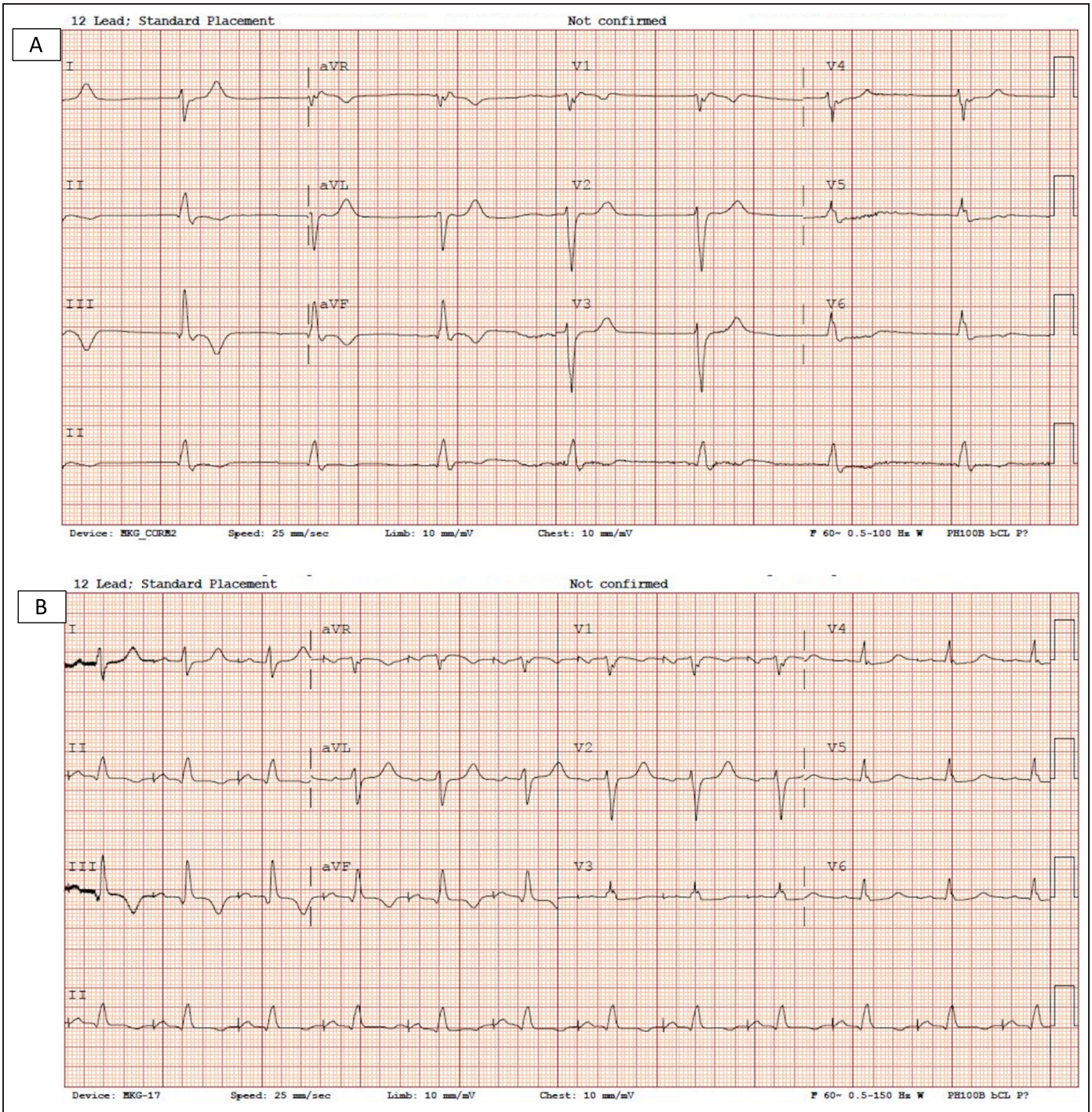

Fig. 3. (A) Electrocardiogram showing bradycardia and ventricular pacing. (B) Electrocardiogram after implantable cardioverter-defibrillator upgrade.

that interval ischemia triggered the patient's decline in sinus node function. Given the patient's significant hemodynamic improvement from temporary atrial pacing, an emergent device upgrade to a new dual-chamber ICD device and right atrial lead placement was performed by the electrophysiology team immediately afterward (Fig. 2B). The patient was subsequently transferred to the cardiac intensive care unit in critical but stable condition on IABP support, mechanical ventilation, and norepinephrine and vasopressin infusions. ECG post-procedure with comparison to baseline is seen in Fig. 3.

A repeat echocardiogram post-lead implantation showed a similarly reduced ejection fraction, however over the following 3 days, he had improvement with successful weaning of all vasoactive drips. The IABP was weaned and removed within 24 h. On hospital day 2 he was extubated. His acute kidney injury and acute ischemic hepatic injury both entirely resolved over the next several days and he was discharged in stable condition on hospital day 10.

The patient was seen in the heart failure clinic 2 weeks post-discharge, at which time he reported slow progress in his exertional capacity and no recurrent ICD discharges or worsening heart failure symptoms. Additionally, RHC performed one month after the procedure showed normal hemodynamics.

Discussion

Chronic RV pacing is known to cause detrimental effects on cardiac function which in turn leads to arrhythmias, heart failure, and cardiac mortality, broadly referred to as pacing-induced cardiomyopathy when no other attributable cause is identified [1]. It is especially noted to worsen preexisting LV dysfunction and can be detrimental in the setting of preexisting HFrEF [1,2].

RV pacing is known to have a depressant effect on LV function because of changes in intrinsic myocardial contractile properties, and several potential mechanisms behind this effect have been theorized [3]. RV apical pacing results in an activation pattern similar to that of a left bundle branch block by causing a delay in electrical activation that results in impaired mechanical contraction. These overall effects result in abnormal myocardial metabolism, elevated cardiac filling pressures, and reduced cardiac output [4]. RV pacing has also been shown to affect myocardial perfusion [5]. Nielsen et al., in a study of 30 patients with sick sinus syndrome, showed that chronic DDD pacing reduced inferior, septal, and global mean myocardial blood flow, and led to a reduced LVEF [5]. Moreover, studies have also demonstrated that secondary repolarization changes persist after cessation of ventricular pacing, manifesting as cardiac memory [4]. As witnessed in the DAVID and MOST trials, increased rates of death or hospitalizations for heart failure, and increased incidence of atrial fibrillation was witnessed in patients with a cumulative RV pacing of >40 % [6,7].

Although the adverse remodeling associated with RV pacing is generally thought to be a chronic process requiring months to years to manifest as a cardiomyopathy, significant changes in LVEF can be observed within hours of instituting RV pacing, as witnessed in our patient [2]. An analysis of 12 patients with baseline normal LVEF and forced RV pacing demonstrated a significant drop in LVEF within 2 h of RV pacing and subsequent progressive deterioration of LV function over time [4]. These acute adverse hemodynamic effects of RV pacing may result not just from abnormal electrical activation but also from a direct impact on ventricular function [2,4].

We present a case of cardiogenic shock in a patient with chronic heart failure secondary to ischemic heart disease who developed sinus arrest and the sudden need for RV pacing. Although the initial suspicion was that the back-up heart rate of 40 bpm on his primary prevention device was simply insufficient to support adequate cardiac output once he developed sinus arrest, this was not supported by further hemodynamic deterioration that he demonstrated at the increased ventricular pacing rate. Although chronic RV pacing is a known cause of worsening LV systolic dysfunction, abrupt heart failure decompensation, particularly complicated by cardiogenic shock, is not a well-known consequence of RV pacing in patients that have underlying depressed LV systolic function [8,9]. It is possible that the patient's hemodynamics improved not only from elimination of intraventricular dyssynchrony but also from restoration of atrioventricular (AV) synchrony, given that AV conduction was preserved and all that was required was atrial pacing. The immediate hemodynamic improvement noted upon a trial of temporary atrial pacing far exceeded any benefit that the patient had demonstrated with other measures including epinephrine infusion and IABP support. It is important to note that while the patient had in fact developed complete sinus arrest, the AV conduction remained intact therefore the atrial pacing measure allowed proper heart rate support without ventricular pacing, thereby correcting both the bradycardia and the ventricular dyssynchrony.

Conclusion

In patients with underlying LV systolic dysfunction and SC-ICDs placed for primary prevention, a sudden increase in the RV pacing burden can cause acute hemodynamic instability related to ventricular dyssynchrony. While rare, this should be included in the differential of precipitating factors for cardiogenic shock secondary to acute on chronic heart failure. This can be safely and effectively corrected with addition of a right atrial lead provided that AV conduction remains intact.

Consent statement

Written informed consent was obtained from the patient for publication of this case report.

Declaration of competing interest

The authors declare that there is no conflict of interest.

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