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Case Report

Right Ventricular Pacing-Induced Hypotension and Pulmonary Edema

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Abstract

We hereby report a case of hypotension and pulmonary edema induced by right ventricular pacing. Ventricular Intrinsic Preference (VIP) algorithm was inadequate in maintaining intrinsic ventricular rhythm, prolongation of Atrioventricular Delay (AVD) aided in restoration of normal hemodynamics.

Case report

92 year-old female with past medical history including hypertension, Parkinson's disease, coronary artery disease, chronic kidney disease, hypothyroidism, LV diastolic dysfunction, paroxysmal atrial fibrillation and sinus node dysfunction; she had a dual-chamber permanent pacemaker in 2016. Patient was admitted for hip fracture following a mechanical fall. Patient underwent cephalomedullary nail fixation. After surgery, patient developed delirium, acute kidney injury, elevated liver enzymes, acute hypoxemic respiratory failure attributed to worsening pulmonary edema (Figure 1). She was transferred to ICU due to persistent hypotension and worsening hypoxemia, and was ultimately started on mechanical ventilation. Acute hypoxemic respiratory failure was thought to be due to hydrostatic/cardiogenic pulmonary edema triggered by right ventricular pacing. Infectious process was not likely in view of normal white blood cell count and absence of fever with negative cultures. Pulmonary embolism was ruled out. Patient was briefly on vasopressor support, she underwent IV diuresis with adequate urine output and improvement in oxygen saturations; however, all attempts to wean off vasopressors and mechanical ventilation were not successful. Ventricular pacing was found to be correlating with BP measured manually and by arterial line (Figure 2). Ventricular Intrinsic Preference (VIP, St. Jude Medical, Sylmar, CA) algorithm was programmed on and led to cyclical ventricular sensed events; however, it failed to maintain persistent ventricular sensed events and adequate BP. VIP was turned off and Atrioventricular Delay (AVD) was extended to 350ms which resulted in persistent ventricular sensed events and stable BP and cardiac output.

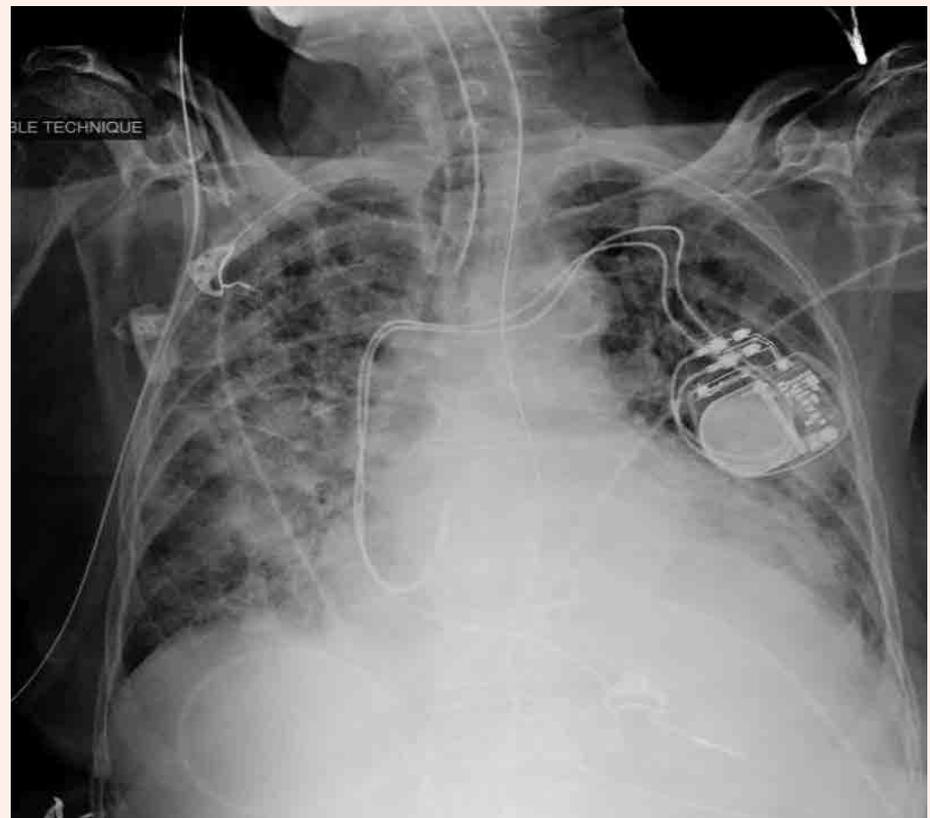


Figure 1: Bilateral diffuse infiltrates consistent with pulmonary edema, dual-chamber pacemaker with right ventricle lead positioned at the apex

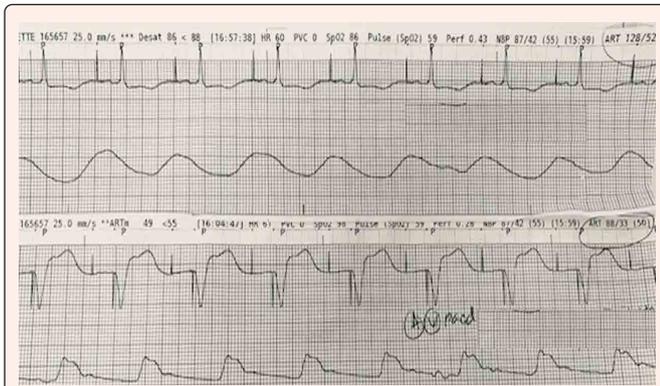


Figure 2: Right ventricular pacing correlating with BP measured manually and by arterial line

Few hours after pacemaker programming adjustments, the patient was successfully weaned off vasopressor support and successfully extubated. Patient remained hemodynamically stable and was transferred to rehabilitation facility.

Discussion

This case highlights how right ventricular pacing can be detrimental and can lead to acute hemodynamic compromise. In our patient and while pacemaker initial settings were present before, we hypothesize that her acute illness and surgery along with ventricular pacing led together to the hemodynamic decompensation manifesting as a drop in her cardiac output and blood pressure along with increased filling pressure and pulmonary edema. There are now abundant studies illustrating the profound hemodynamic deleterious effects due to ventricular dyssynchrony created by right ventricular pacing, especially when pacing from the apex [1-5]. While VIP is designed to minimize ventricular pacing, we think it has failed in maintaining overall adequate BP, manual programming is required at times.

Very prolonged AVD has been described to result in hemodynamic derangement including shortening and impairment of LV filling, increased left atrial pressure and, in some patient, symptoms resembling pacemaker syndrome [6-7]. However, individualizing programming can be favorable like in our patient.

Conclusion

This case report highlights that high burden of right ventricular pacing can lead to acute and detrimental hemodynamic compromise. Pacemaker reprogramming to minimize right ventricular pacing can result in a remarkable and rapid clinical improvement.

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Conflict of interest ; None

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