

## Case Report

### *Development of Heart Failure Following Pace Maker Implantation in a Patient With Congenital Heart Block*

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#### ABSTRACT

Congenital complete heart block (CCHB) is a rare anomaly and is a potential indication for pacemaker implantation. However, pacemakers might lead to cardiomyopathy. Here we describe a young woman with CCHB presenting with heart failure early postpartum and pacemaker implantation.

**Keywords:** Complete heart block● Pacemaker-induced cardiomyopathy● Peripartum cardiomyopathy

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Congenital complete heart block (CCHB) can be isolated or occur in the setting of systemic autoimmune disorders or structural heart disease.<sup>1</sup> The patients could be asymptomatic or have minimal symptoms, so the diagnosis may be delayed and symptoms interpreted for other causes.<sup>1</sup> Once CCHB is diagnosed, pacemaker implantation is indicated for patients with symptomatic and significant bradycardia.<sup>1</sup> On the other hand, one potential complication of pacemakers in this group is reported to be the development of cardiomyopathy.<sup>2</sup>

We herein describe a patient with CCHB, who received a permanent pacemaker (PPM) in her postpartum period and presented with early-onset congestive heart failure.

#### CASE REPORT

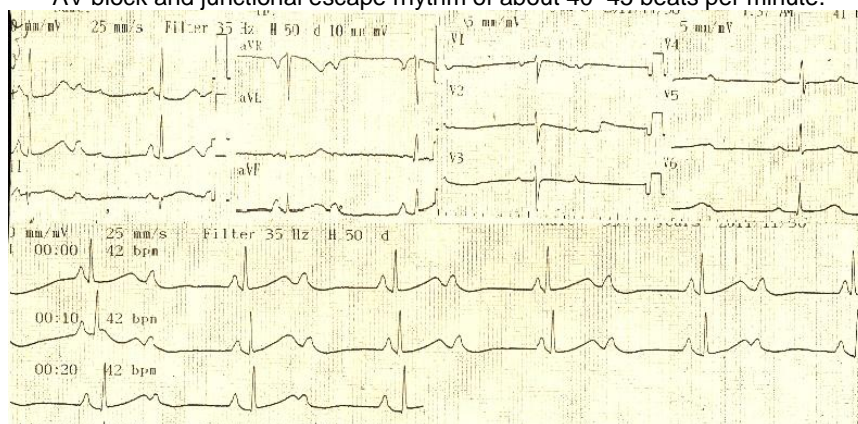
A 27-year-old 1st gravid woman, in her 38 weeks of gestation, who was admitted to our

hospital for termination of pregnancy, was found to have significant bradycardia during routine cardiac monitoring. ECG showed a heart rate of about 40 bpm with recorded narrow QRS complexes having no relation to atrial contraction (A-V dissociation) (Fig. 1). She had no history of syncope but mentioned episodes of exertional dizziness and easy fatigability. She had no past medical history of significant systemic illnesses or drug use. Cardiology consult was performed and because the patient was currently asymptomatic and had a narrow QRS response, she was scheduled to have natural vaginal delivery under cardiac monitoring with percutaneous temporary pacemakers being kept at hand in case she became more bradycardic or developed asystole. The delivery course was uneventful, and cardiac monitoring was continued in the postpartum phase for 5 days, during which she remained in complete heart block. Transthoracic

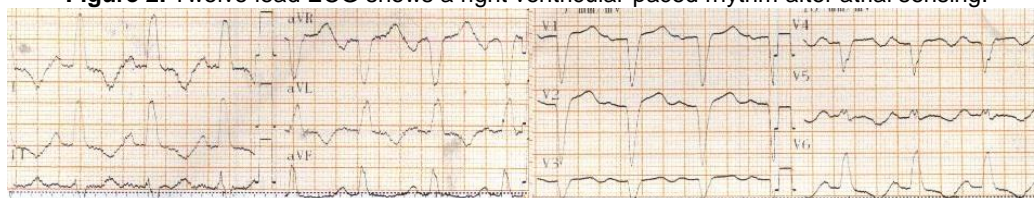
echocardiography was done; it showed normal left ventricular (LV) size and systolic function (left ventricular ejection fraction [LVEF] = 55%) as well as normal right

ventricular (RV) size and function with no significant valvular heart disease. A PPM was implanted for the patient, and she was discharged home in a good condition (Fig. 2).

**Figure 1.** Twelve-lead ECG depicts 2:1 atrioventricular (AV) block with intermittent complete AV block and junctional escape rhythm of about 40–45 beats per minute.



**Figure 2.** Twelve lead ECG shows a right ventricular-paced rhythm after atrial sensing.



After 45 days, the patient referred to the emergency department complaining of dyspnea on exertion (New York Heart Association's functional class 3), fatigue, palpitation, and chest pain. She was admitted for further evaluation. ECG showed normal function of the pacemaker, but transthoracic echocardiography revealed a normal LV size with severe LV systolic dysfunction (LVEF = 10%–15%). On chest X-ray, the pacemaker leads were seen in proper position in the RV apex and the right atrial appendage. Analysis of the PPM showed a normal pacemaker function. Laboratory tests, including troponin I and renal and liver function tests, as well as rheumatologic evaluations revealed no abnormality. Due to persistent chest pain, coronary angiography was performed to evaluate for an abnormal course, coronary slow flow, or other abnormalities.

Nonetheless, all the coronary arteries were patent and had normal courses.<sup>3</sup>

Medical therapy for heart failure was started with lisinopril, spironolactone, and carvedilol. The patient's symptoms were relieved a few days after the beginning of the therapy, and she was discharged after a week.

Serial follow-up transthoracic echocardiographic examinations showed gradual improvement in LV contractility and there was a reported LVEF of 35% twelve months after the medical therapy for heart failure.

## DISCUSSION

Our patient was a case of asymptomatic and most probably CCHB. She developed rapid-onset congestive heart failure within 45 days after delivery and receiving a PPM.

The underlying cause of this event remains ambiguous. Indeed, did the patient suffer from coincidental peripartum cardiomyopathy (PPCM) or did she actually develop pacemaker-induced cardiomyopathy?

PPCM is an uncommon cardiomyopathy that results in myocardial damage in the late phases of pregnancy and the early postpartum period, causing maternal morbidity and mortality.<sup>4</sup> The reported incidence is variable in different communities and ethnicities, and diverse risk factors have been implicated.<sup>4</sup> PPCM is clinically similar to dilated cardiomyopathy, but the course and prognosis are unpredictable.<sup>4</sup> The diagnosis is made based on the occurrence of *de novo* heart failure during the terminal months of pregnancy or the initial 5 months after the delivery in the absence of cardiac anomalies before pregnancy. It is usually reached by the exclusion of other possible causes. The transthoracic echocardiography criteria<sup>4</sup> include LVEF < 45%, fractional shortening < 30%, and LV end-diastolic dimension > 2.7 cm/m<sup>2</sup>. Despite the definitions, PPCM could occur beyond the mentioned time period either earlier in the pregnancy or later postpartum. Indeed, about 13% of the cases are thought to happen outside the defined timeline, leading to underdiagnosis.<sup>4</sup>

The standard therapy for CCHB and significant bradyarrhythmias is the implantation of PPMs. The RV lead is often placed in the apical area. In this situation, electrical impulse conduction happens through the myocardial cells and outside the normal conduction pathway.<sup>5, 6</sup> The stimulation starts from the RV, which causes dyssynchronous cardiac stimulation and a wide QRS complex of left bundle branch block morphology.<sup>5,7</sup> Pacemaker-induced heart failure is a known complication of RV pacing, with a reported incidence of 10% in patients with CCHB and 9%–10% in the general population in different studies.<sup>2,8</sup> The more the RV is paced (> 40% of cardiac cycles), the greater the risk of cardiomyopathy will be. Pacemaker-induced

cardiomyopathy is defined echocardiographically as an LVEF < 45% and a decrease >5% in the LVEF as compared to the baseline EF in the 1st year post PPM implantation. That is the reason why some studies have suggested that individuals who are completely pace-dependent receive biventricular pacemakers regardless of baseline patient characteristics.<sup>2,9</sup>

The definite diagnosis is challenging in this case due to the time overlap of the responsible pathologies. However, the question is whether there is a greater tendency of the development of pacemaker-induced cardiomyopathy in the early postpartum period.

Pregnancy leads to significant hemodynamic changes, including increases in plasma volume and heart rate, which could be damaging in mothers with baseline cardiac diseases<sup>10</sup> or perhaps render the myocardium more vulnerable to the pacing effects early after delivery. Future molecular and genetic studies might be able to elucidate the exact pathologic insult at the myocyte level.

## CONCLUSIONS

PPCM and pacemaker-induced cardiomyopathy are 2 rather uncommon entities leading to variably reversible cardiomyopathy. In the current case, the 2 mentioned pathologies seemed to coincide with the possibility of simultaneous or additive harmful effects on the myocardium.

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