

CASE REPORT

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Preemptive cardiac resynchronization therapy to prevent cardiac arrest in peripartum cardiomyopathy: a case report

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Abstract

Select patients with peripartum cardiomyopathy are candidates for implantable cardioverter-defibrillator (ICD) or cardiac resynchronization therapy (CRT) due to the increased risk for life threatening arrhythmias and sudden death. We present a case of peripartum cardiomyopathy awaiting CRT placement who suffered cardiac arrest during the surgical dilation and curettage (D&C) procedure for pregnancy termination.

Key words: Postpartum cardiomyopathy; Peripartum cardiomyopathy; Cardiac arrest; Implantable cardioverter defibrillator; Cardiac resynchronization therapy; Dilation and curettage; Hysteroscopy, Vasovagal, Vagal, Pulseless electrical activity

Abbreviations: PPCM - Peripartum cardiomyopathy, ICD - Implantable cardioverter defibrillator, AICD - automated implantable cardioverter defibrillator, CRT - Cardiac resynchronization therapy, D&C - Dilation and curettage, PEA - Pulseless electrical activity

Citation: Dhoon TQ, Kim S, Eshraghi Y, Perry R, Hameed A, Rajan GRC, Przybysz A. Preemptive cardiac resynchronization therapy to prevent cardiac arrest in peripartum cardiomyopathy: a case report. *Anaesth. pain intensive care* 2020;24(3):358-362.

Received: 15 February 2020, Reviewed: 25 June 2020, Accepted: 27 June 2020

1. Introduction

Peripartum cardiomyopathy (PPCM) is a form of idiopathic heart failure secondary to left ventricular (LV) systolic dysfunction that occurs in an otherwise healthy woman towards the end of pregnancy or in the first few months postpartum. It is a diagnosis of exclusion. The incidence of PPCM is estimated at approximately 1 in 3,200 deliveries in the United States. Fortunately, LV function normalizes (LVEF > 45%) in 30 to 50% of these women within 2 to 6 months after delivery.¹⁻² Women with PPCM who fail

to recover LV function despite standard heart failure therapy may be candidates for implantable cardioverter defibrillator (ICD) or cardiac resynchronization therapy (CRT) due to an increased risk for arrhythmias, and sudden death. We report a case of PPCM awaiting CRT placement who suffered cardiac arrest during the surgical dilation and curettage (D&C) for termination of pregnancy. Written informed consent was obtained from the patient according to our institutional policy.

2. Case Description

A 60 kg 30-year-old gravida 3, para 2 presented for D&C at 10 weeks gestational age. She was diagnosed with PPCM four months following her previous pregnancy and was scheduled for a cardiac resynchronization therapy with defibrillator (CRT-D); however, the procedure was postponed as she was found to be pregnant. Her medical history was significant for severe congestive heart failure, with ejection fraction (EF) 26%. She underwent multidisciplinary counseling with the obstetrician and the cardiologist regarding treatment options and elected to proceed with termination of pregnancy and placement of a subdermal contraceptive implant. Her preoperative laboratory workup was within normal limits; however, electrocardiogram showed sinus tachycardia (110/min) and intraventricular conduction delay. Transthoracic echocardiogram showed severely decreased left ventricular (LV) systolic function with EF of 26%, severe diastolic dysfunction, elevated pulmonary artery systolic pressure (RVSP 41 mmHg), and moderate right ventricular (RV) dysfunction with mitral valve regurgitation and tricuspid regurgitation. In view of her very high perioperative cardiac risk, the anesthetic plan involved combination of monitored anesthesia care with light sedation and paracervical block. The patient was premedicated with midazolam 2 mg and fentanyl 50 µg followed by an infusion of inj propofol at 50 µg/kg/min.

The patient was placed in lithotomy position and received a paracervical block with 18 mL of 0.5% lidocaine. During the very first moments at the start of cervical dilation, the patient went into profound bradycardia and severe hypotension, which quickly deteriorated into pulseless electrical activity (PEA). The surgery was halted, propofol infusion was stopped, and the patient was quickly placed in supine position and cardiopulmonary resuscitation (CPR) was initiated. After two minutes of CPR and 1 mg intravenous bolus of epinephrine, return of spontaneous circulation was established. The patient was intubated, a radial arterial catheter inserted and central venous access established. The infusions of dobutamine 5 µg/kg/min and vasopressin 0.04 units/min were started. We proceeded with the D&C and placement of subdermal contraceptive implant once patient's hemodynamics stabilized.

The postoperative course was immediately complicated by encephalopathy characterized predominantly by behavioral agitation and transient aphasia. A computed tomography of the brain was negative for ischemia or intracranial hemorrhage and her continuous electroencephalogram showed excessive theta waves nonspecific for mild

encephalopathy. The patient's neurologic deficits self-resolved by postoperative day (POD) 2. On POD 3 the patient underwent placement of a CRT-D under a left pectoralis nerve block and monitored anesthesia care. Her subsequent inpatient hospital course was unremarkable, and the patient was discharged home on POD 4.

3. Discussion

In our case, during the preoperative preparation our multidisciplinary team had serious concerns about the patient's tenuous cardiac status with possibility of developing intraoperative malignant arrhythmias and sudden cardiac death. The cardiologist recommended CRT-D placement at the earliest instance. In hindsight, preemptive placement of CRT-D prior to therapeutic abortion may have prevented intraoperative cardiac arrest and PEA. Vasovagal episodes are well documented in relation to cervical dilation, hysteroscopy and endometrial biopsy.³⁻⁵ CRT-D placement prior to D&C would have changed both management and outcome in this case. If a CRT-D had been placed prior to the procedure it would have been possible to prevent the cardiac arrest and the subsequent hypoxic encephalopathy.

Patients with PPCM are at heightened risk of progressive heart failure, sudden death and arrhythmias.⁶ Cardiac resynchronization therapy (CRT) is a form of biventricular ICD. CRT is recommended for patients with cardiac failure (particularly those with a low EF (< 35%)), intraventricular conduction delay, or prolonged QRS > 120 ms. Biventricular stimulation improves quality of life, exercise tolerance and may partially reverse maladaptive cardiac remodeling. CRT may reduce morbidity and mortality among patients with cardiac failure and intraventricular conduction delay.⁷

Complications of PPCM include thromboembolism, heart failure, arrhythmias, and sudden cardiac death.^{1,8} Over 80% of the complications usually present within the first six months of diagnosis.¹ Interestingly, 10-17% of patients with PPCM have evidence of LV thrombus on preliminary diagnostic echocardiography.¹ Once the diagnosis of PPCM is established and subsequent to the delivery of the baby, reliable contraception with intrauterine device or contraceptive implant must be considered. Further,

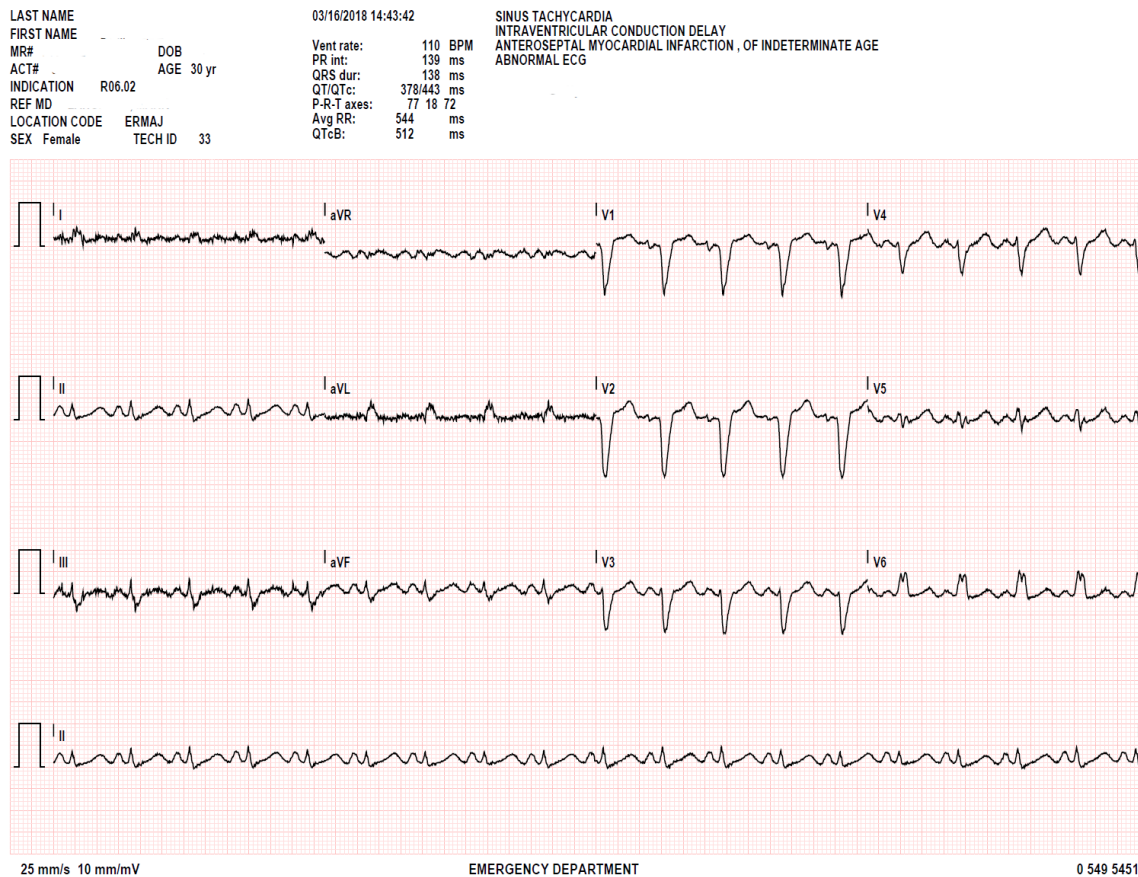


Figure 1: Preoperative ECG demonstrating sinus tachycardia with an intraventricular conduction delay

permanent sterilization should be discussed with the patient and her family.

PPCM is defined as idiopathic cardiac failure secondary to LV systolic dysfunction ($EF \leq 45\%$) in a previously healthy woman with clinical features similar to non-ischemic dilated LV. PPCM typically presents late in pregnancy (mean 32-38 weeks) or in the 5 to 6 months following fetal delivery.^{1-2,9-10} Of note, 45% of patients present during the first week following delivery and 75% do so within the first month.¹¹ The incidence of PPCM has been estimated to be approximately 1 in 3,200 deliveries in the United States.^{1,9} PPCM is a diagnosis of exclusion. Differential diagnosis includes disease states that may be unmasked by pregnancy including idiopathic dilated cardiomyopathy, familial dilated cardiomyopathy, and valvular heart disease, as well as hypertensive heart disease, pre-existing unrecognized congenital heart disease, pregnancy associated

myocardial infarction, pulmonary embolism, and HIV/AIDS cardiomyopathy.¹²

The pathophysiology of PPCM is currently unclear but likely multifactorial and include viral infections, abnormal immune response, unbalanced cardiomyocyte oxidative stress, angiogenic imbalance (preeclampsia & VEG-F signaling), and response to hormonal triggers (i.e. prolactin and its cleaved products).^{1-2,9-10} PPCM is associated with older maternal age, obesity, smoking, gestational hypertension, preeclampsia, multiple gestations, and African American descent.⁸⁻⁹ Further, geographical hotspots of PPCM have been observed in Haiti, South Africa and Nigeria.^{1-2,8,10} Diagnosis of PPCM is often missed or delayed due to the overlapping of physiological changes of normal pregnancy and those of heart failure. Women reporting less severe symptoms of fatigue, shortness of breath, cough, asthma, palpitations, or edema may be ignored by the healthcare providers resulting in delays in diagnosis.

PPCM may present insidiously over weeks or as acute symptomatic heart failure.¹⁰ Timely diagnosis of PPCM is of utmost importance as a delay in diagnosis of more than one week has been shown to increase morbidity.¹⁰

Management consists of a combination of beta-blockers, diuretics, spironolactone, ACE inhibitors (ACE), angiotensin receptor blockers (ARBs), intravenous or oral vasodilators, intravenous inotropes and digoxin.¹ Anticoagulants may be indicated in select cases to decrease the risk of thromboembolism. Treatment with intravenous immunoglobulins, pentoxifylline, or bromocriptine has also been reported; however, these are not the standard of care in the United States.^{6,13}

4. Conclusion

Implantable cardioverter defibrillator placement is often delayed among patients with PPCM in anticipation of LV recovery, which is usually expected within six months of diagnosis. However, such approach may leave a significant subset of patients at risk for malignant arrhythmias and sudden death. We recommend that women who exhibit depressed ejection fraction and intraventricular conduction delay should merit strong consideration for early implantation of cardiac resynchronization therapy.

5. Disclosures

The authors have no disclosures.

6. Authors' contribution

TD, SK: literature search, manuscript editing.

YE, RP, AH, GR, AP: manuscript editing

7. References

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MY CORONA STORY

DOI: 10.35975/apic.v24i3.1296

My own experience of COVID-19

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England has seen its worst days; past few months have been full of multiple challenges and hard struggle for the people of United Kingdom. No rules in the beginning of the current pandemic to few restrictions like social distancing and eventually implementation of a complete lockdown. To one's surprise, the restrictions were not very strictly followed in the beginning. I have the vivid memories of the fear surrounding us all by the daily reports from Italy. We were two weeks behind Italy and expected a similar surge in UK. Fortunately we were not the first worst effected country and had some time to think and plan based on the facts and reports. A meeting was called in our hospital in anticipation, involving all the departments e.g., physicians, surgeons and nursing staff. The plan also included a short ICU training run to other medical staff in order to deal with the surge.

I felt fine till the end of March, when on waking up one morning, I felt unwell and had cold like symptoms, which started getting better on third day of illness. I thought it was just simple cold but then there was severe myalgia, low grade fever, loss of taste and smell, headache, excessive sleepiness and tiredness and mild shortness of breath on exertion. My other family members got similar complaints too, but the intensity was mild. It flashed in my mind for a moment that I had contracted COVID-19, but wasn't 100% sure, as the COVID test was not being carried out in UK at that time, and the public health England's advice was to self-isolate at home.

I took paracetamol for myalgia and fever but the myalgia was so severe that I had to add ibuprofen at times. I also had sore throat and stuffy nose. Headache was severe at times. I used to think that why my symptoms were not getting better, especially myalgia was literally killing me and was probably the cause of excessive tiredness and sleepiness. I was fortunate enough not to have my diet effected by the illness and was still able to eat despite of loss of taste and smell.

I was confined to bed most of the time and hoped and prayed for a negative corona disease. It took nearly fourteen to sixteen days for me to become symptoms free. It gives me great pleasure to express my feeling that it gave me a great chance and ample time to read through the researches, updates and the articles on COVID-19, and to write something on it during that period.

I went back to work after I recovered completely and have been working fearlessly since then. It was only recently (3rd week of June) when I got confirmation of my illness to be COVID-19 on getting positive antibodies in my blood. My colleagues think that I am lucky to have positive antibodies at this stage; but I have a mixed feeling and often ask myself if it is really true as it could easily have gone the other way round.

Things are much better now in UK as we are approaching the end of June with most of the restrictions have been eased off. We wish that the situation keeps on improving and we never have to face the second wave of this virus. I wish that every frontliner stays motivated, healthy and carries on with hard work till the end of this pandemic.