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Peripartum Cardiomyopathy With Reduced Ejection Fraction In The Third Trimester: A Case Of Severe Left Ventricular Dysfunction With Successful Acute Management

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Abstract
This case report describes the case of a 31-year-old primigravida with no previous cardiac history who was referred from Civil Hospital during her third trimester with a known reduced ejection fraction (EF). She subsequently delivered via vacuum-assisted vaginal delivery. Following delivery, she was found to have a severely decreased LVEF (Left Ventricular Ejection Fraction) of 20-25%, and evidence of bilateral pedal edema. She was also noted to be tachycardic (140 BPM), hypotensive, and desaturated (SpO2 of 84% on room air). Based on these findings she was diagnosed with PPCM (Peripartum Cardiomyopathy) with severe LV (left ventricular) dysfunction and acute PE (pulmonary edema). She was treated in the ICU with diuretics, vasopressors, and standard heart failure therapy. Once stabilized, she was transferred to the cardiology ward for continued treatment.
We conclude that early identification and prompt management of PPCM is critical as it has significant implications for improving maternal health and survival. Although PPCM is extremely rare, it should be ruled out in all pregnant patients who experience symptoms consistent with heart failure during the puerperium. Furthermore, early diagnosis, treatment by multiple disciplines, and standard heart failure therapy are key factors in improving maternal health outcomes.

Keywords: *Peripartum cardiomyopathy; Heart failure; Postpartum complications; Left ventricular dysfunction; Maternal morbidity*

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I. Introduction

Peripartum Cardiomyopathy (PPCM), is an Idiopathic Cardiomyopathy that causes Heart Failure through Left Ventricular Systolic Dysfunction at the end of Pregnancy, or Postpartum Period; and when No Other Cause of Heart Failure can be Found (1).

The first descriptions of PPCM were published during the 1930's. Estimated Incidence of PPCM ranges from 1 in every 1000 to 1 in every 4000 Live Births; however the actual rate may vary depending on the Geographic Region (2).

Although the exact cause of PPCM has yet to be determined, several Mechanisms have been Proposed including Viral Myocarditis, Autoimmune Processes, Hormonal Influences, and Oxidative Stress (3).

As a result of its unpredictable nature, PPCM is a Leading Cause of Maternal Morbidity and Mortality. Outcomes can range from Complete Recovery to Chronic Heart Failure, Need for Transplantation, and/or Death (4).

We are reporting a case study of a 31 year old woman who developed PPCM post Cesarean Delivery, and will provide the Clinical Presentation, Diagnostic Approaches, and Management of PPCM as it relates to our patient.

II. Case Presentation

Here ia a case of 31 years Primigravida at 37 weeks gestation. No significant prior medical history was reported, and she had received routine prenatal care during her entire pregnancy without any recorded or documented issues or complications. Her vital signs upon admission were normal with a blood pressure of 130/70 mmHg; pulse rate of 140 beats per minute; and oxygen saturation of 98% on room air.

She had been referred from Civil Hospital during her third trimester with a known reduced ejection fraction (EF). At 37 weeks of gestation, she was admitted and subsequently delivered via vacuum-assisted vaginal delivery. The patient delivered a healthy female infant weighing 3.595 kg, with good apgars. The delivery was completed without any immediate perioperative complications.

At 3:30 am on the first post-operative day (day 2 post-partum), the patient began experiencing sudden onset shortness of breath, tachypnea, and oxygen desaturation (spO2 = 84% on 6 L/min O2). Examination revealed bilateral rales on lung auscultation; elevated jugular venous pressure; and pedal edema (grade I) bilaterally. Tachycardia with a heart rate of 152 bpm, and hypertension with a blood pressure of 168/110 mmHg were noted on cardiovascular examination. The patient was promptly transported to the Emergency Medical Services Intensive Care Unit for further treatment.

Laboratory results included:
* Hemoglobin: 12.8 g/dL
* Total Leukocyte Count: 15,240/mm^3
* Serum Urea: 39 mg/dL
* Serum Creatinine: 0.8 mg/dL
* Liver Function Tests and Electrolyte Panel: Normal
* Chest X Ray revealed Bilateral Pleural Effusion with Pulmonary Edema
* Electrocardiogram: Sinus Tachycardia with no Specific ST/T Wave Changes

A Transthoracic Echocardiogram was performed revealing Severely Reduced Left Ventricular Ejection Fraction (20-25%) with Global Hypokinesis and Dilated Cardiac Chambers. Diastolic Dysfunction Grade 1, and No Significant Valvular Abnormalities were identified. Based on the clinical presentation and echocardiographic findings, a diagnosis of Peripartum Cardiomyopathy with Acute Decompensated Heart Failure and Pulmonary Edema was made (5).

The Patient was treated with:
* Initial Intravenous Furosemide 40 mg dose with subsequent Stat Doses of 20 mg for Diuresis
* Supplemental Oxygen Therapy (initially 6 L/min via Face Mask that was gradually weaned as her condition improved)
* Injection Noradrenaline Infusion for Adequate Tissue Perfusion
* Initiation of standard heart failure medications, including Injection Lasix, Oral Torsemide, and Injection Ramipril
* Commencement of Beta-Blocker Therapy with Injection Carvedilol and Up-titration as Tolerated (6)

Monitoring during her ICU stay revealed sequential improvements in oxygen saturation (94% on Room Air by Day 3); Resolution of Tachypnea; and Stabilization of Blood Pressure (120/80-130/80 mmHg). Improvement in tachycardia was noted and treated with Injection Isolazine. Serial Cardiac Biomarker Monitoring and Optimization of Medical Management were performed.

Transfer to the Cardiology Ward for Continued Management and Monitoring of the patient was made on Post Partum Day 5. The patient showed Progressive Clinical Improvement, Resolved Breathlessness and Decrease in Pedal Edema. She was discharged on Oral Diuretics (Torsemide, Furosemide), Angiotensin-Converting Enzyme Inhibitor (Ramipril), Beta Blocker (Carvedilol), and Spironolactone for Heart Failure Management (7, 8). Additionally, she was started on Bromocriptine for Suppression of Lactation as Breastfeeding is Generally Contraindicated in PPCM Patients on Multiple Cardiac Medications (9).

When discharged, the patient was Hemodynamically Stable with Improved Functional Status. The patient's discharge medications included Oral Furosemide, Torsemide, Ramipril, Carvedilol, Spironolactone, and Bromocriptine. The patient was instructed to Avoid Exertion, Follow an Immunization Schedule as per Protocol, Practice Exclusive Bottle Feeding for 6 Months, Avoid Conception for 1-2 Years, Use Contraceptive Counseling, Attend Preconceptional Counseling and Regular Follow-Up in the Cardiology Outpatient Department for Monitoring of Cardiac Function and Adjustment of Medications (10).

III. Discussion

A diagnosis of peripartum cardiomyopathy (PPCM) is one of exclusion that is defined by the development of heart failure in the final month of pregnancy or in the five months after delivery in a woman without known heart disease (11). According to the European Society of Cardiology PPCM is defined by four elements: heart failure developed toward the end of pregnancy or in the months after delivery, there is no identifiable reason for the development of heart failure; no evidence of recognized heart disease existed prior to the last month of pregnancy; and a left ventricular systolic dysfunction with an ejection fraction of <45% (1).

Our patient demonstrated all the classic signs of PPCM in that she had developed acute heart failure symptoms on her first postoperative day following cesarean delivery and she had significantly decreased left ventricular ejection fraction (LVEF) (20-25%) with global hypokinesis as evidenced by echocardiogram.

Risk factors for PPCM have included: being >30 years of age, being a multiparous female, having African ancestry, having a multiple gestation, having a hypertensive disorder of pregnancy, and receiving prolonged tocolytic therapy (2,12). Our patient was 31 years of age, a primigravida and had none of these risk factors.

The exact pathophysiologic mechanism of PPCM is still unknown; however, several theories exist. The most commonly accepted theory is that prolactin is cleaved into a 16 kDa fragment by cathepsin D, resulting in both anti-angiogenic and pro-apoptotic effects on cardiomyocytes (3). Other potential mechanisms for PPCM include: oxidative stress, inflammation, auto-immune responses, viral myocarditis, and pregnancy-related hormonal effects (13). There is also evidence to suggest genetic predisposition exists in patients with PPCM based on sarcomeric protein gene mutations found in some individuals (14).

Presentation of PPCM is similar to other causes of heart failure and includes symptoms such as dyspnea, orthopnea, paroxysmal nocturnal dyspnea, fatigue, and peripheral edema (5). A physical exam may demonstrate tachycardia, elevated jugular venous pressure, pulmonary rales, S3 gallop, and peripheral edema (5). Our patient demonstrated symptoms of acute heart failure including acute shortness of breath, tachycardia and bilateral pedal edema.

Evaluation of PPCM should include electrocardiogram (ECG), chest x-ray, brain natriuretic peptide (BNP) levels and comprehensive echocardiogram. Echocardiogram is the gold standard of diagnosing PPCM, usually demonstrating left ventricular dilation, decreased left ventricular ejection fraction and often global hypokinesia (6). Cardiac magnetic resonance imaging (MRI) can provide further information regarding myocardial inflammation or fibrosis, but is rarely required to diagnose PPCM.

Management of PPCM is similar to managing non-pregnant heart failure with consideration for pregnancy and lactation issues. Initial treatment of PPCM includes oxygen therapy, diuretics for volume overload, vasodilators if the patient's blood pressure allows, and inotropes if needed (7). Long-term management of PPCM includes use of ACE inhibitors or ARBs (contraindicated during pregnancy), beta-blockers, MRA and diuretics as needed (8). Use of bromocriptine as an adjunct to treatment to inhibit prolactin production has shown promise in some studies and is becoming increasingly used (9).

Recovery from PPCM varies among patients. Some recover completely whereas others develop chronic heart failure or require cardiac transplantation. Prognostic indicators of PPCM include: initial LVEF of <30%, increased left ventricular end diastolic diameter, delay in diagnosis, and African ethnicity (15). Most patients show improvement in cardiac function by six months postpartum if they will improve at all. Our patient improved clinically during her hospital stay, and long term follow-up will be important to determine whether she has recovered cardiac function.

It is important to counsel women with PPCM regarding future pregnancies as the risk of relapse is extremely high (20-50%) in subsequent pregnancies, regardless of whether the patient has completely recovered cardiac function (10). In fact, women with persistent left ventricular dysfunction are generally advised against future pregnancies due to high maternal mortality (10).

This case demonstrates the need for a high level of suspicion for PPCM in women experiencing heart failure symptoms in the peripartum period. Prompt recognition, initiation of heart failure treatment, and multidisciplinary care of obstetricians, cardiologist, and intensivist, are all critical to optimal patient outcome.

IV. Conclusion

The postpartum period is when peripartum cardiomyopathy (PPCM) commonly develops. Promptly identifying this condition and using an aggressive treatment plan will save many lives. While PPCM occurs infrequently, health care providers need to be extremely vigilant for signs and symptoms of heart failure in all pregnant women and new mothers; therefore, it is vital that clinicians diagnose PPCM as soon as possible and manage the patient with a multidisciplinary approach using standard heart failure therapies. In addition, it is very important to counsel patients about future pregnancies so that they can make informed decisions about their future childbearing. Furthermore, long-term follow up of these patients will help clinicians assess the extent of recovery of the patient's cardiac function and allow them to adjust management appropriately.

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