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Case Report On A Near Miss Case Of Peripartum Cardiomyopathy With Triumphant Feto-Maternal Outcome

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Abstract

Peripartum cardiomyopathy (PPCM) can be classified as a variant of dilated cardiomyopathy usually identified in the last month of pregnancy prior to delivery or within the first five months after delivery. The incidence of peripartum cardiomyopathy (PPCM) varies geographically. Diagnosis of PPCM is difficult and requires a multidisciplinary approach for proper management. It has high morbidity and mortality rate.

Keywords: Peripartum Cardiomyopathy(PPCM),

Introduction

Peripartum cardiomyopathy (PPCM) can be identified as an idiosyncratic type of dilated cardiomyopathy usually diagnosed in the 3rd trimester, specifically in the last month of gestation prior to delivery or within the first five months postpartum. The occurrence of peripartum cardiomyopathy (PPCM) varies from place to place. The incidence varies in different racial groups, it differs from 1 in 1,000 to 1 in 4,000 in the United States.(1) The incidence in southern India was noted to be 1 case per 1374 live births in a study by Pandit V et. al.(2) Risk factors for PPCM include African descendants, advanced maternal age, hypertensive disorders like pre-eclampsia, multifetal pregnancies, etc. (1)

Diagnosing PPCM is cumbersome and challenging because its features can often be perceived as a continuum of pregnancy and postpartum events. It has fatal complications and high mortality rate when misdiagnosed and treatment is delayed.

Case Report

A 27-year-old primigravida at 34.5 weeks gestation had presented to the emergency department of a tertiary care centre, Lokmanya Tilak Municipal Medical College and General Hospital, Mumbai with complaints of sudden onset breathlessness and perspiration since 6 to 7 hours. Patient also had complaint of cough with expectoration since 1 day. The patient was a known case of preeclampsia on treatment with tablet Labetalol 100 mg TDS and tablet Nifedipine (R) 10mg BD. ANC profile of the patient was within normal limits. The patient had been primarily registered at a peripheral health care centre and was repeatedly advised admission previously for evaluation and management of pregnancy induced hypertension. On examination, general condition of the patient was moderate with Pulse rate 140 bpm, Blood Pressure 180/110 mmHg, Respiratory rate of 30 /min maintaining SpO_2 of 76% on room air, 88% on O_2 nasal prongs and arterial partial pressure of oxygen was 41mmHg on ABG analysis. On Respiratory examination the patient had bilateral coarse crepitations. The patient had bilateral pedal edema and did not have any premonitory symptoms of fulminant preeclampsia. There was no history of chronic hypertensive disorder, congenital cardiac disorder, valvular dysfunction, carditis, cardiomyopathy, autoimmune disorders, or any other major medical/ surgical disorder. Patient did not have any significant past obstetric history/ family history suggestive of any predisposing factor and there was no history of any known allergy/ addictions. The

antenatal period was uneventful, until the current episode. Chest X-ray indicated an enlarged heart shadow and bilateral opacities showing pulmonary oedema, and Arterial Blood Gas analysis revealed a PaO2 of 66 mmHg on supplemental oxygen at 15 L/min via a NRBM mask, antihypertensive NTG via iv infusion and diuretic, iv frusemide bolus were given. Ultrasound examination showed fetal parameters appropriate for gestational age and within normal limits. An Echocardiography done immediately revealed an ejection fraction of 25-30 % and global left ventricular hypokinesia with mild pulmonary arterial hypertension with moderate mitral regurgitation, mild tricuspid regurgitation, and a diagnosis of peripartum cardiomyopathy was made in view of no other identifiable cause for the left ventricular diastolic dysfunction in the patient. The patient's condition deteriorated progressively. Patient was shifted to non-invasive CPAP mode of ventilation and antihypertensive infusion drip with nitroglycerine and Lasix were continued. The patient developed a sudden cardiac arrest and immediate resuscitation was started. Patient developed refractory respiratory failure and thus was put on mechanical ventilation. SpO2 increased to 90% after mechanical ventilation with high PEEP (Positive End Expiratory Pressure) and 100% FiO2 (Fraction of inspired Oxygen). Aggressive cardiopulmonary resuscitation was given and the patient was revived and shifted on ionotropic supports. The patient was being managed by the multidisciplinary team in intensive cardiac critical care unit. Chronic heart diseases, hypertensive disorders, and other identifiable causes of cardiomyopathy were excluded. As the patient had rapidly deteriorating heart failure, was clinically diagnosed with PPCM in a known case of preeclampsia, and emergency cesarean section was proposed by the obstetrician and the intensivists considering both maternal and fetal prognosis. Emergency LSCS was performed under general anesthesia after informed consent was taken from the relatives within 5 hours of the admission and the patient delivered a female child of 1894 grams. The patient was shifted back to the ICCU and her condition improved drastically after the CS. The patient was extubated after 1 day and ionotropic support and Lasix infusion drip were tapered gradually. Postoperatively, the patients received iv antibiotics, Bisoprolol 2.5 mg BD, Co-enzyme and tablet Carnisure, and tablet Lasix. Tablet Isolazine (Isosorbide dinitrate (20mg) + Hydralazine hydrochloride(37.5mg)) was started from postoperative day 5 and was advised to continue for 3 months post-discharge. Tablet Bromocriptine 2.5 mg BD was given and injection unfractionated Heparin 5000 IU was given postoperatively for 7 days. Rest of the postpartum period was uneventful in the ward. Echocardiography was done before discharge, which showed a left ventricular ejection fraction of 45% with mild MR, and mild TR. The patient and neonate were in good condition on follow-up visits.

Discussion

Peripartum cardiomyopathy (PPCM) can be identified as an idiosyncratic type of dilated cardiomyopathy usually diagnosed in the 3rd trimester, specifically in the last month of gestation prior to delivery or within the first five months postpartum without a history of cardiovascular disease. The Heart Failure Association of the European Society of Cardiology Working Group, redefined PPCM in 2010 to "an idiopathic form of cardiomyopathy that presents with heart failure secondary to LV systolic dysfunction towards the end of pregnancy or in the months following delivery, with no other identifiable cause of heart failure".[3]

The diagnostic criteria include:[4]

1. Cardiac failure occurring during the last month of gestational period or the initial 5 months postpartum;
2. No other identifiable precipitating factor for the heart failure;
3. No demonstrable preexisting heart disease prior to pregnancy; and
4. Echocardiographic features
    a. Left ventricular end-diastolic dimension >2.7 cm/m^2
    b. M-mode fractional shortening <30%
    c. Left ventricular ejection fraction <45%

Our case met all the diagnostic criteria. Obstetricians play a vital role in the diagnosis of PPCM in the early stages during antenatal follow-ups. It is a form of diastolic heart failure with undetermined etiopathogenesis and has diverse clinical manifestations. Often, PPCM occurs in the postpartum period; less than 10% of cases occur in

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the antepartum period. Clinical features include dyspnea, orthopnea, paroxysmal nocturnal dyspnea, cough, hemoptysis, fatigue, malaise, chest and abdominal discomfort, palpitations, postural hypotension, etc.[4] These patients often present with class III or IV New York Heart Association (NYHA) dysfunction. Early diagnosis is crucial as initial symptoms of PPCM can be misjudged with physiologic changes of advanced gestation.

Management of PPCM requires a multidisciplinary approach involving obstetricians, cardiologists, and intensivists. The treatment plan is to improve symptoms via conventional medical therapies for heart failure and through targeted curative therapies. Beta-blockers and loop diuretics can be administered before delivery in patients with heart failure, but done cautiously as diuretics can diminish the placental blood flow which may result in fetoplacental insufficiency. Hence, the diuretic therapy should be used in the minimum possible and effective doses. During the postpartum period, the standard heart failure therapy is recommended in PPCM, which includes beta-blockers, angiotensin-converting enzyme (ACE) inhibitors, angiotensin II receptor blockers (ARBs), and diuretics. The use of inotropic infusion like dobutamine should be initiated only in cases of refractory hypotension. Inotropes are tapered with the return of blood pressure to normal, and the standard guidelines are followed thereafter. Early beta-blocker therapy has shown to be protective even at very low doses in patients with a severely depressed ejection fraction.[5]

High-risk factors for thromboembolism include pregnancy, heart failure, and associated prolonged immobilization. The administration of anticoagulants (Unfractionated heparin /low molecular-weight heparin (LMWH) and warfarin) in the antepartum & postpartum period is suggested when the LV ejection fraction is less than 30%.[4] Presence of atrial fibrillation increases this risk even further. Bromocriptine, a dopamine agonist with prolactin inhibiting action, is also found to be fruitful in these patients. According to a study by Hakata et al., the use of bromocriptine leads to increased survival rates. It was contemplated that excess production of prolactin increased the accumulation of a 16-kDa prolactin fragment, which is antiangiogenic and plays an important role by negatively impacting myocardial micro-vascularization. Bromocriptine therapy decreases the amount of the prolactin fragment and helps improve the cardiac function.[5]

In this case, the patient was diagnosed with peripartum cardiomyopathy in critical stage and prompt decision-making saved the lives of both the mother and the baby. The decision regarding when to terminate the pregnancy is vital in the management. Emergent CS should be done immediately when the patient's condition does not improve in spite of exhaustive conservative treatment. In the present case, we ventured to stabilize the patient's condition, but her condition worsened instead. Recovery is defined as the return of LVEF to 50% or improvement by 20%. It usually takes 3- 6 months post-delivery for recovery, but might occur as late as 48 months postpartum.[4] Recurrence in following pregnancies is reported as high as 46%, especially among women with pre-pregnancy LVEF <0.55. The mortality rate in the United States associated with PPCM is estimated to be 6% to 10%.[4] Up to 50% of patients with PPCM improve with standard medical treatment of heart failure; however, up to 25% of patients develop chronic heart failure while some perish to have a fatal course. Subsequent pregnancy should be avoided if the LVEF does not return to normal values. However, normalization of LV function does not endorse a normal consequent pregnancy; approximately 20% of these patients are also at risk of moderate to severe LV dysfunction, which may persist even after delivery in 20% to 50% of patients. Early antenatal registration and screening and referral of high-risk cases to a higher center enables us to diagnose these patients before acute clinical presentation.[6]

Conclusion

In this case report we present a near-miss case of PPCM, the patient was successfully treated with triumphant maternal and fetal outcomes via a multidisciplinary team involving emergency cesarean section combined with intensive critical cardiac care and comprehensive medical care for heart failure. The findings here establish that early identification and referral to a tertiary care institute, timely intervention, and prompt decision for termination of pregnancy form the bottom-line in the management of PPCM.

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References:

1. Peripartum Cardiomyopathy Review - American College of Cardiology. Available from:
2. https://www.acc.org/latest-in-cardiology/ten-points-to remember/2020/01/13/15/18/peripartum-cardiomyopathy
3. Pandit V, Shetty S, Kumar A, Sagir A. Incidence and outcome of peripartum cardiomyopathy from a tertiary hospital in South India. http://dx.doi.org/101258/td2008080353. 2009 Jun 17;39(3):168–9.
4. Davis MB, Arany Z, McNamara DM, Goland S, Elkayam U. Peripartum Cardiomyopathy: JACC State-of-the-Art Review. Vol. 75, Journal of the American College of Cardiology. NLM (Medline); 2020. p. 207–21.
5. Cardiomyopathy P. 8. Vol. 39, Tex Heart Inst J. 2012.
6. Chinweuba GC, Rutkofsky IH. Unveiling the Mystery of Peripartum Cardiomyopathy: A Traditional Review. Cureus. 2020 Oct 4;
7. Joshi A v., Fonseca MN, Kharat-Kapote DS. A study of peripartum cardiomyopathy in a tertiary care center in India. Int J Reprod Contracept Obstet Gynecol. 2017 Jan 31;6(2):523.

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