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Case Report

Peripartum cardiomyopathy: A challenging case

Sanjay Ponde[1], Vidya Gaikwad[1], Ashton Dsouza[1]*

[1]Dept. of Obstetrics and Gynecology, Dr. D.Y. Patil Medical College, Dr. D.Y. Patil Vidyapeeth (Deemed to be University), Pimpri, Maharashtra, India

Abstract

Peripartum cardiomyopathy (PPCM) is a rare life-threatening condition which is associated with pregnancy. It is characterized by cardiac failure due to left ventricular (LV) systolic dysfunction. Here, we report the case of a 24-year-old G3P1L1A1 female who presented at 36 weeks of gestation with gestational hypertension, bilateral pedal edema, and a non-reactive non-stress test. She underwent emergency lower segment cesarean section for fetal distress and developed palpitations and dyspnea in the postoperative period. Echocardiography showed severe LV systolic dysfunction (LVEF 20%), global hypokinesia, severe pulmonary artery hypertension (PAH), and a left ventricular clot. Treatment with diuretics, beta-blockers, and anticoagulants showed significant improvement over five months postpartum, with an LVEF recovery to 45%. This case highlights the critical need for early diagnosis and a multidisciplinary approach in the management of PPCM to improve maternal outcomes and prevent complications such as thromboembolism.

Keywords: Cardiomyopathy, Pregnancy Complications, Echocardiography, Tachycardia.

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1. Introduction

Peripartum cardiomyopathy (PPCM) is a rare condition which carries a life-threatening potential. It is characterized by the dysfunction of left ventricular (LV) during the end of pregnancy or within the first few months postpartum.[1] The incidence varies globally, and in India the it has been reported to occur in 1 among 1340 live births in India.[2] The variation in the reported incidence can be attributed to several factors, including differences in demographics, diagnostic criteria, and underreporting.[3] Many times, a lack of awareness or misunderstanding of the condition leads to misinterpretation or missed diagnoses, making it harder to get an accurate picture of the incidence.[4] Although the exact etiology remains unknown, several hypotheses, coronary artery abnormalities, hormone dysregulation, immunological responses to fetal antigens, toxaemia and myocarditis, have been proposed.[3] A significant recurrence rate in subsequent pregnancies necessitates the importance of early diagnosis and vigilant monitoring.[5]

Diagnosis relies on echocardiographic criteria, including a left ventricular end-diastolic dimension of 2.7 cm/m² or more, fractional shortening on M-mode of 30% or less, or a left ventricular ejection fraction of 45% or lower.[6] This condition significantly contributes to morbidity and mortality among women of childbearing age worldwide. Here, we present an intriguing case of PPCM encountered at our institution, emphasizing the importance of understanding this condition to deliver the most effective and appropriate care to our patients.

2. Case Presentation

A 24-year-old woman, with a gestational age of 36 weeks and 1 day, presented to the hospital with complaints of bilateral pedal edema lasting for 10 days. The swelling had no specific aggravating or relieving factors. She denied symptoms such as dyspnea, palpitations, or chest pain. Previous medical records revealed gestational hypertension being diagnosed at 32 weeks of pregnancy which was managed with oral

*Corresponding author: Ashton Dsouza
Email: dsouzaashton1410@gmail.com

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labetalol (100 mg twice daily). Routine antenatal investigations, including blood parameters, were normal.

On general examination, the patient had stable vital signs, but bilateral pedal edema was noted. Obstetric examination revealed a relaxed uterus which was consistent with the gestational age. The fetus was in cephalic presentation, and fetal heart sounds were auscultated at 176 beats per minute (bpm) over the left spino-umbilical line. A non-stress test (NST) was performed and which was found to be non-reactive, necessitating an emergency lower segment cesarean section (LSCS) for fetal distress. Intraoperatively, thick meconium-stained liquor was noted. The patient delivered a male infant weighing 2.4 kilograms. The immediate post-operative period was uneventful. However, on the fourth post-operative day (POD 4), she developed palpitations and breathlessness. Clinical examination revealed a pulse rate of 124 bpm and respiratory rate was noted to be 24 breaths per minute. Oxygen saturation of 92% recorded on room air. The blood pressure was within the normal limits. Auscultation of the chest revealed basal crepitations over the lower lung fields. X-ray was done, which showed cardiomegaly (**Figure 1**).

Further a cardiology consultation was sought. An electrocardiogram (ECG) showed sinus tachycardia. Two-dimensional (2D) ECHO was done which revealed severely depressed LV systolic function and ejection fraction was found to be 20%. Additionally, a left ventricular end-diastolic diameter of 56 mm, and global hypokinesia was also noted. The findings also indicated grade III diastolic dysfunction, severe pulmonary artery hypertension (PAH), severe tricuspid regurgitation (TR), grade II mitral regurgitation (MR), and a thin rim of pericardial effusion. These findings confirmed the diagnosis of PPCM associated with severe LV dysfunction and PAH. The patient was started on intravenous furosemide (40 mg three times daily), oral eplerenone (25 mg twice daily), ramipril (2.5 mg twice daily), and carvedilol (3.125 mg twice daily). On further inquiry, the patient revealed a history of a similar condition following her previous delivery, during which she had been admitted to the intensive care unit (ICU). However, we were unable to elicit correct history from patient as this history had been concealed during her antenatal visits.

On POD 10, the patient was prescribed oral diuretics (a combination of furosemide 20 mg and spironolactone 50 mg, twice daily) after leaving the hospital against the medical advice. Meticulous documentation of events was done in discharge summary. She returned to the cardiology outpatient department on POD 22 with complaints of chest pain for two days. Repeat echocardiography revealed a large left ventricular apical clot measuring 2.8 × 0.8 cm, attached to the apical lateral wall, with no significant change in systolic function. The patient was started on oral rivaroxaban (20 mg once daily), dapagliflozin (10 mg once daily), and sacubitril/valsartan (24/26 mg twice daily).

At a three-month follow-up, echocardiography showed resolution of the LV clot, with no vegetation, but the LVEF remained unchanged at 20%. By five months postpartum, significant improvement was observed, with the LVEF increasing to 45%. The patient remained stable on follow-up, with her medical regimen adjusted accordingly.

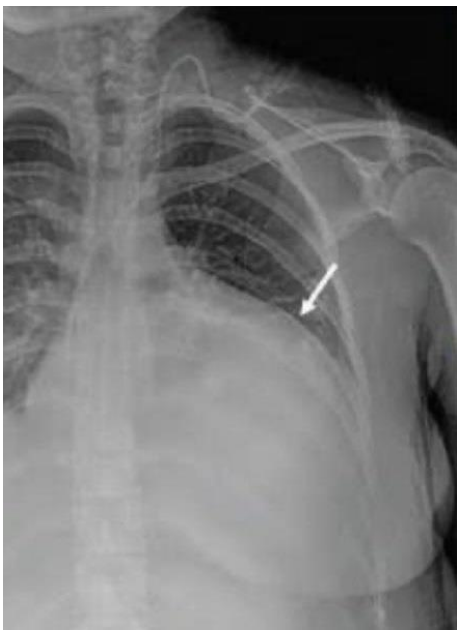

Figure 1: X-ray showing cardiomegaly

3. Discussion

Peripartum cardiomyopathy (PPCM) is a rare condition which carries a life-threatening potential. Several risk factors have been identified to contribute to the development of this condition.[3] Recent studies highlight oxidative stress, prolactin-mediated mechanisms, genetic predisposition, and inflammatory responses as critical contributors.[7] A genome-wide study has implicated a gene on chromosome 12 in PPCM pathogenesis.[8] Studies have also attributed elevated levels of tumor necrosis factor-alpha (TNF-α) and interleukin-6 suggest as an inflammatory etiology.[5,9] Other mechanisms, such as autoimmune responses to fetal cells in maternal circulation, and abnormal relaxin hormone effects, have also been postulated.[3]

The clinical presentation of PPCM includes heart failure symptoms such as tachycardia, tachypnea, elevated jugular venous pressure, cyanosis, pulmonary rales, basal crepitations, and peripheral edema.[3,7] Chest radiography often shows cardiomegaly and pulmonary venous congestion, while ECG findings typically indicate sinus tachycardia without specific changes.[10] Diagnosis requires meeting the criteria of development of heart failure in the last month of pregnancy or postpartum within five months with no previous history of heart condition, and evidence of LV systolic dysfunction demonstrated by echocardiography.[11-13] Echocardiography is essential for detecting associated complications such as LV thrombus, which can lead to severe

thromboembolic events. Patients with an LV ejection fraction (EF) ≤ 35% may benefit from anticoagulation therapy, such as novel oral anticoagulants (NOACs), to reduce thrombus formation. Post operative use of Low molecular weight Heparin or Novel Oral anticoagulants like Dabigatran is beneficial in reducing incidence of clots in such cases and would have prevented clot formation if used in our case. Yamamoto et al. reported successful LV thrombus resolution with dabigatran (220 mg/day).[14] Several biomarkers have been shown to be highly sensitive for identifying adverse maternal events and assessing heart failure risk.[15] It was seen that graduated compression stockings are effective in reducing risk of Deep vein thrombosis and its thromboembolic complications in patients who have undergone surgery.[21] Early ambulation and exercise is also beneficial in reducing Thromboembolic events. Management of PPCM involves a multidisciplinary approach, including beta blockers, diuretics, nitrates, and inotropic or ventilatory support as required.[7] For patients with hemodynamic instability unresponsive to medical management, advanced interventions like mechanical circulatory support or ventricular assist devices (VADs) are considered.[16] During pregnancy, the use of ACE inhibitors or angiotensin receptor blockers (ARBs) is contraindicated due to teratogenic risks, but these medications form a cornerstone of postpartum treatment.[17] Emerging therapies, such as bromocriptine and intravenous immunoglobulin, have shown promise but require further evaluation in larger clinical trials.[18]

The mode and timing of delivery in these cases depend on the maternal hemodynamic status. Hemodynamically stable patients may attempt vaginal delivery, while caesarean sections are recommended for those with severe cardiac decompensation. General anaesthesia may be preferred in patients with anticoagulant use to minimize sympathetic blockade and the risk of heart failure.[19] The prognosis varies, with more than 50% of women achieving complete recovery of LV function within six months postpartum. However, the mortality rate remains significant, ranging from 10–25%, despite treatment.[20] Subsequent pregnancies carry a high risk of recurrence (over 30%), and family planning counseling is crucial. Patients are advised to delay subsequent pregnancies until at least five years after EF normalization to reduce risks. In our case, Patient was 10th standard pass and belonged to low socioeconomic class and thus decided to hide past history of PPCM from us. Hence, education and financial status play an important role to ensure compliance.

4. Conclusion

Peripartum cardiomyopathy (PPCM) is a rare yet potentially life-threatening condition. Early recognition and timely, effective management significantly improve the chances of complete recovery while reducing associated mortality and morbidity. Our case highlight the diverse presentations. The case underscores the importance of maintaining a high index of suspicion for PPCM in its various forms. Familiarity with its spectrum of presentations enables prompt diagnosis and intervention, ultimately improving patient outcomes.

5. Source of Funding

None.

6. Conflict of Interest

None.

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