The Unusual Presentation of Peripartum Cardiomyopathy Leading to Fatal Outcomes in Two Young Parturients: Case Reports

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ABSTRACT

Peripartum cardiomyopathy (PPCM) is a rare but serious condition characterised by heart failure during late pregnancy or early postpartum. This report discusses two cases of PPCM in young parturients highlighting variable presentations and outcomes. Case one involved a 26-year woman who deteriorated rapidly in the postoperative period despite initial stabilisation. This deterioration led to multi-organ failure and death. Case two was a 29-year woman who also experienced acute decompensation of heart failure, progressing to cardiac arrest and death despite aggressive management. These cases highlight the unpredictable nature of PPCM and the challenges in diagnosis and management. Early recognition and prompt and aggressive therapy are crucial to improve outcomes in PPCM, emphasising the need for heightened clinical suspicion and multidisciplinary care in pregnant and postpartum patients.

Key Words: *Peripartum cardiomyopathy, Young parturients, Heart failure.*

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INTRODUCTION

Peripartum cardiomyopathy (PPCM) is a systolic dysfunction of the left ventricle that occurs in the last month of pregnancy or within five months postpartum.[1] The incidence of PPCM in Pakistan is not documented. However, the reported incidence in Europe was one in 4,950 deliveries.[2] PPCM can manifest as mild symptoms of dyspnoea and fatigue, mimicking pregnancy symptoms, to severe manifestations such as pulmonary oedema, arrhythmias, thromboembolic events, cardiogenic shock, or sudden cardiac death.[3] Diagnosis of PPCM involves clinical assessment, echocardiography for left-ventricular dysfunction, and laboratory tests measuring *BNP/NT-pro BNP* and troponin levels, supported by ECG findings.[4] Early recognition is vital for prompt intervention and improved outcomes. The treatment of PPCM includes heart failure management with diuretics, beta-blockers, and angiotensin-converting enzyme inhibitors or angiotensin receptor blockers.[5] Bromocriptine, by inhibiting prolactin secretion, reduces the myocardial stress associated with lactation, potentially improving cardiac function and outcomes in women with PPCM.[6]

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In severe cases, mechanical circulatory support or heart transplantation may be considered. Understanding the unusual presentations and their potential implications in PPCM is crucial for early recognition, intervention, and ultimately, for improving maternal and foetal outcomes. This paper explores two cases of PPCM in young parturients, highlighting the challenges in diagnosis and management, and the importance of heightened clinical suspicion and vigilance in obstetric care settings.

CASE 1

A 26-year lady G2, P1, A0, presented at 37 weeks of gestation with foetal distress for emergency caesarean section. The patient had a history of shortness of breath on exertion with no history of orthopnoea and paroxysmal nocturnal dyspnoea. Her previous pregnancy was uneventful, and she had caesarean section for the breech presentation of the foetus. Her past medical history, medicine history, and family history were negative. The period of gestation remained uneventful throughout. On examination, the patient had a pulse rate of more than 100 beats per minute. The pulse was regular and of good volume. The patient had low blood pressure (BP) of 87 / 56 mm/Hg. On chest auscultation, vesicular breathing was normal overall lung zones with normal heart sounds. Oxygen saturation was 96%. Due to low systolic BP and the urgency of caesarean section, the case proceeded under general anaesthesia. Induction was smooth and uneventful. Intraoperatively, BP improved with fluid resuscitation. A healthy baby was delivered. The patient was extubated successfully and shifted to the postoperative area, vitally stable and pain-free. After two hours, there was a call from the postoperative team that the patient became irritable and had low BP readings repeatedly. The patient was

managed and shifted to the intensive care unit (ICU) immediately for further monitoring and work-up.

In ICU, the patient developed tachypnoea, diffuse fine crackles at the base of the chest, and oxygen saturation of 88% within an hour of presentation. The patient was put on noninvasive ventilation and diuresis was started. However, due to respiratory distress, the patient was intubated and mechanical ventilation was initiated. Later on, the patient went on inotropic support. Echo revealed a low ejection fraction of 25% *pro BNP* and Troponin were elevated. ECG showed poor progression of the R wave. Bromocriptine and ivabradine were started. Despite aggressive treatment, including inotropic support and ventilatory assistance, the patient's condition deteriorated rapidly, leading to multiorgan failure and death.

CASE 2

A 29-year G2, P1, A0 presented for emergency caesarean due to foetal distress at 37 weeks of gestation. Preoperatively, the patient had no history of orthopnoea, paroxysmal nocturnal dyspnoea, or dyspnoea on exertion. The previous pregnancy was uneventful. There was no significant medical history, medicine history, or family history. The general physical examination was also unremarkable. The case proceeded under spinal anaesthesia. Immediately after the delivery of a healthy baby, the patient developed hypotension. Nor-epinephrine support was started. After a few hours, the patient developed tachypnoea and fine crackles at the bases of both lungs. The patient was shifted to ICU, and an echo was performed immediately. Echo revealed an ejection fraction of 30% and grade 1 mitral regurgitation. *Pro BNP* and Troponin T were elevated. ECG showed asymmetrical T wave inversions in I, AVL, and V1-V4. Dobutamine was added as another inotropic support along with digoxin and bromocriptine. Due to the worsening of dyspnoea, the patient was intubated the next morning. Despite aggressive management, invasive haemodynamic monitoring, and escalating doses of inotropes, the patient went into cardiac arrest and could not be revived.

DISCUSSION

These two cases showed the variable presentations and outcomes of PPCM in young parturients, emphasising the rapid progression of the disease and its devastating consequences. In both cases, the patients had mild to no symptoms before hospital presentation but they deteriorated rapidly to the stage of acute decompensation of heart failure. Especially, the stage of auto-transfusion from the uterus and massive fluid shifts intraoperatively, resulted in a shift from stage A of heart failure to stage D.[7,8] In these types of scenarios, clinicians should maintain a high index of suspicion for PPCM in pregnant patients presenting with even mild cardiac symptoms.[9] Both cases demonstrated a rapid progression of PPCM, leading to catastrophic outcomes within hours to days postpartum. Thus, the importance of timely diagnosis by echocardiography and biomarkers is crucial to mitigate the risk of adverse outcomes.[7] The prompt initiation of medical therapy, including diuretics, vasodilators, beta-blockers, and inotropic support, is essential in stabilising patients with PPCM.[10] Additionally, close monitoring in the ICU allows for the timely detection of haemodynamic changes and optimisation of therapy.[11] Despite aggressive management, the prognosis of PPCM can be unpredictable, emphasising the need for multidisciplinary care and advanced cardiac support in specialised centres. Further research is needed to elucidate the pathophysiology of PPCM and to identify novel biomarkers for early diagnosis and risk stratification. Additionally, the strategies for prevention and long-term management of PPCM remain areas of ongoing investigation.

PATIENTS' CONSENT:

Consent was taken from immediate blood relatives of the patients to report these cases.

COMPETING INTEREST:

The author declared no conflict of interest.

AUTHORS' CONTRIBUTION:

AA: Data collection and drafting of case report, revision of the manuscript critically for the important intellectual content.

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