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Case Series

Peripartum cardiomyopathy: case series from a tertiary care centre in India

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ABSTRACT

Peripartum cardiomyopathy (PPCM) is a rare condition characterized by the onset of left ventricular systolic dysfunction and heart failure symptoms in the late stages of pregnancy or the early postpartum period. This paper presents a case series of Peripartum Cardiomyopathy (PPCM) from a tertiary care center in India. Seven cases are described, highlighting the diverse clinical presentations, diagnostic challenges, and treatment outcomes encountered. The majority of patients were young primigravidae, emphasizing the importance of considering PPCM even in low-risk individuals. Diagnosis relied on echocardiographic findings and exclusion of other causes of heart failure. Treatment strategies included pharmacological interventions aimed at reducing afterload, preload, and enhancing contractility. Despite the high mortality rate associated with PPCM, timely critical care managed by cardiologists prevented fatalities in our cohort. This series underscores the significance of early recognition, prompt referral, and collaborative management in optimizing outcomes for PPCM.

Keywords: Cardiomyopathy, 2D ECHO, Pregnancy, Heart failure, Peripartum

INTRODUCTION

Peripartum cardiomyopathy (PPCM) is an uncommon but life-threatening form of dilated cardiomyopathy that manifests during the last month of pregnancy or within five months post-delivery.[1] The aetiology of the condition is largely unknown, though multiple risk factors like pre-eclampsia, and chronic hypertension, have been proposed.[2] The incidence varies in different geographical regions, but predominantly prevailed in the low- and middle-income settings.[1,3] The incidence is unknown in the Indian setting. Clinically, PPCM is characterized by symptoms such as dyspnoea, oedema, and fatigue, which are often overlooked as they mimic normal pregnancy adjustments, complicating timely diagnosis.[4] The diagnostic criteria for PPCM include idiopathic onset, development in the peripartum period, left ventricular systolic dysfunction leading to heart failure, and a decrease in left ventricular ejection fraction to below 45% or fractional shortening below 30%.[5] The severity of untreated PPCM can lead to dire complications including cardiogenic shock and fatal arrhythmias, underscoring the importance of early detection and a high clinical suspicion towards the end of pregnancy.[4,6] Management requires a coordinated care approach, typically involving specialists across multiple disciplines.[3] This paper presents a series of five cases of PPCM presented in an Indian setting, emphasizing successful outcomes achieved through early intervention and comprehensive care highlighting the essential knowledge needed for effective clinical practice.

Methodology

PPCM is characterized by heart failure occurring in the last month of pregnancy or up to five months postpartum, without prior heart failure and not attributed to other identifiable causes. It is diagnosed by echocardiographic evidence of left ventricular systolic dysfunction, including

a left ventricular ejection fraction (LVEF) less than 45%, fractional shortening less than 30%, and an enlarged left ventricular end diastolic dimension greater than 2.7 cm/m2 of body surface area (Demakis criteria).[7] Clinical assessments involved collecting data on patient age, gestational age at presentation, parity, symptoms, and risk factors. Exclusion of other heart failure causes was thorough, using ECGs, cardiac enzymes and thyroid tests. Echocardiography was performed using the acuson cypress system, analyzing parameters like LV end diastolic dimension, LV fractional shortening, and LVEF with media logic software.

Management by a multidisciplinary team including intensivists, obstetricians, and cardiologists focused on preload optimization, afterload reduction, and enhancing cardiac contractility. Treatment regimens included diuretics, digoxin, vasodilators, and β-blockers. Postpartum patients might also receive ACE inhibitors, inotropes, and anticoagulants. Dietary modifications like fluid and salt restriction were implemented to manage blood pressure. Delivery methods were tailored based on obstetric needs, and pregnancy outcomes were carefully documented. Complications such as pulmonary edema, acute kidney injury, multiple organ dysfunction syndrome (MODS), and pleural effusion were managed as needed. Recovery criteria were an LVEF of 50% or higher, LV fractional shortening of 30% or more, and achieving New York Heart Association (NYHA) functional class I, with or without ongoing heart failure medication. All newborns received care according to the hospital protocol. This study adhered to ethical guidelines approved by the Hospital Ethics Committee, ensuring proper clinical care, informed consent, appropriate laboratory investigations, and adherence to the study protocol, including manuscript submissions for publication.

CASE SERIES

Case 1

A 23-year-old primigravida at 40 weeks and 2 days gestation presented with one day of NYHA class III breathlessness. She had no significant past medical history. Examination revealed a sick-looking patient in severe respiratory distress with a respiratory rate of 30 breaths per minute, BP of 130/90 mmHg, HR of 130 BPM, and oxygen saturation of 70% on room air (94% on 5l/min oxygen). Bilateral basal crepitations were noted. Clinically, she was in heart failure of unknown etiology. In the second stage of labor, the labor was augmented, and she delivered a healthy male infant (2.98 kg) via vacuum-assisted vaginal delivery due to fetal distress. Laboratory findings showed a WBC count of 27,000 and D-dimer level of 5000 µg/dl. Chest X-ray, ECG, and echocardiogram findings are summarized in Table 2. She was diagnosed with new-onset PPCM in cardiac failure and admitted to the ICU, where she was intubated and treated as per cardiologist's orders (Table 3). Nebulization and chest physiotherapy were administered. By day 3, her condition improved, and she was transferred to the stepdown HDU on oxygen support. Asymptomatic, she was discharged on day 12 with medications from the cardiac clinic.

Case 2

A 26-year-old primigravida at 41 weeks and 1 day gestation presented in latent labor with a three-week history of a non-productive, non-blood-tinged cough. She had no significant past medical history and was not considered high risk during ANC visits. Examination showed stable vitals, bilateral lower limb pitting edema, and bilateral coarse lung crepitations. Suspecting a lower respiratory tract infection, conservative management was initiated. Due to non-progression of labor despite four doses of misoprostol (25 mcg), she underwent an uneventful emergency LSCS under spinal anesthesia. Post-surgery, she developed breathlessness with oxygen saturation dropping to 80% (rising to 92% with 5 L/min oxygen), pulse rate of 120 BPM, and respiratory rate of 32 breaths per minute.

Lab investigations were normal, but chest X-ray, ECG, and echocardiogram (Table 2) revealed a low ejection fraction of 35%. Diagnosed with PPCM, she was admitted to the ICU and treated per cardiologist's recommendations (Table 3). Clinically stable by day 2, she was moved to the stepdown HDU on day 5. She was discharged on day 16 with prescribed medications and remained well without complications.

Case 3

A 28-year-old G2A1 woman at 39 weeks and 2 days gestation was referred due to preeclampsia. On admission, BP was 140/90 mm Hg; other vitals were stable. She had a history of increased BP two weeks before delivery and was on antihypertensive medications, with regular antenatal visits. Laboratory results were normal except for a WBC count of 22,000. The COVID-19 test was negative, and D-dimer levels were normal. She underwent an emergency LSCS due to doppler changes indicating uteroplacental and fetoplacental insufficiency and fetal growth restriction. On the day of surgery, she developed breathlessness and a productive cough, with saturation dropping to 82% on room air, a pulse rate of 126 BPM, and a respiratory rate of 30 breaths per minute. Auscultation revealed reduced air entry in the right lower lung lobe. She was moved to the ICU with oxygen support. Chest X-ray, ECG, and echocardiogram (Table 2) led to a diagnosis of new-onset PPCM in cardiac failure by cardiologists. Treatment was initiated as per Table 3. By day 4, oxygen was weaned off, and she was discharged on day 10. She has remained well since, with no complications.

Case 4

A 20-year-old primigravida at 35 weeks and 1 day gestation was referred for antepartum eclampsia. She had

a two-day history of NYHA class III cough and breathlessness. There was no history of increased bp in previous visits. On admission, her oxygen saturation was 77% on room air, pulse rate was 136 bpm, and respiratory rate was 30 breaths per minute. Auscultation showed bilateral reduced air entry. She was shifted to the ICU with oxygen support. Laboratory results were normal except for a WBC count of 22,000. The COVID-19 test was negative, and D-dimer levels were normal. Chest X-ray, ECG, and echocardiogram (Table 2) were performed. Obstetric scan revealed intrauterine fetal demise with abruptio placentae. Diagnosed with new-onset PPCM in cardiac failure, she was treated as per cardiologist's advice (Table 3). Following induction, she delivered vaginally. She had one episode of cardiac arrest 10 minutes post-delivery, but was successfully revived with two cycles of CPR. By day 2 post-delivery, her symptoms improved, and oxygen was weaned off by day 5. She was discharged on day 17 with prescribed medications. She has remained well since.

Case 5

A 33-year-old G2P1L1 woman at 37 weeks and 1 day gestation presented with two days of NYHA class III breathlessness and fever. She had no significant past medical history and had not undergone any cardiac evaluation. On examination, she appeared sick with severe respiratory distress: respiratory rate of 30 BPM, BP of 130/90 mm Hg, HR of 130 BPM, and oxygen saturation of 85% on room air (98% on 6l oxygen). Bilateral crepitations were heard.

Lab results showed a WBC count of 27,000, positive COVID-19 RTPCR, and D-dimer level of 5000 µg/dL. Chest X-ray, ECG, and echocardiogram findings are summarized in Table 2. Diagnosed with new-onset PPCM in cardiac failure with COVID-19, she was admitted to the ICU, intubated, and treated as advised by a cardiologist (Table 3). Nebulization and chest physiotherapy were also initiated. In latent labor, she underwent an emergency LSCS on the same day, delivering a healthy baby (Table 1). By day 3, her condition improved, and she was transferred to the stepdown ward on oxygen. She was discharged on day 24 with medications from the cardiac clinic and has remained well without further complications.

Case 6

A 23-year-old primigravida at 35 weeks and 2 days gestation with dichorionic diamniotic twins was referred for preeclampsia. She presented with one day of NYHA class III breathlessness. She had no significant past medical history.

On examination, she appeared sick with a respiratory rate of 26 BPM, BP of 120/90 mm Hg, HR of 92 BPM, and oxygen saturation of 88% on room air. Bilateral crepitations were heard. Lab results showed hemoglobin of 8 g/dl and a negative COVID-19 RTPCR. Chest X-ray, ECG, and echocardiogram findings are summarized in Table 2. Diagnosed with new-onset PPCM in cardiac failure, she was admitted to the ICU with 10l of oxygen, intubated, and treated per cardiologist's recommendations (Table 3) along with chest physiotherapy. She underwent an uneventful emergency LSCS due to twin gestation (Table 1). By day 10, her condition improved, and she was transferred to the stepdown ward on oxygen. She was discharged on day 16 with medications from the cardiac clinic.

Case 7

A 26-year-old primigravida at 36 weeks gestation was referred for imminent eclampsia. On admission, BP was 140/90 mmHg, with a two-day history of elevated BP. Other vitals were stable. She was on antihypertensive medications and had regular antenatal visits. Lab results showed normal hematological levels except for a WBC count of 22,000. COVID-19 RTPCR was negative, and D-dimer levels were normal. She underwent an emergency LSCS due to fetal distress.

Post-LSCS, the patient desaturated to 75% on 10L oxygen, with BP 150/100 mmHg, HR 88 BPM, and respiratory rate 20 BPM. She was moved to the ICU, initially on BiPAP, then non-invasive ventilation, maintaining 98% saturation. BNP was 2250 pg/ml, and 2D echo showed LVEF of 25%, diagnosing PPCM. Cardiology recommendations were followed. By day 6, she improved symptomatically and was moved to the stepdown HDU. She was discharged on day 13.

Table 1: Clinical characteristics of the patients.

Clinical characteristics	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7
Maternal age (years)	23	26	28	20	33	23	26
Gestational age (weeks)	40 weeks 2 days	41 weeks 1 day	39 weeks 2 days	35 weeks 1 day	37 weeks 1 day	34 weeks 2 days	30 weeks
Clinical history	No	H/o LRTI since 3 months	Preeclampsia	Antepartum eclampsia with	Covid positive status	Preeclampsia	Imminent eclampsia

Continued.

Clinical characteristics	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7
				abruptio placentae			
Parity	Primigravida	Primigravida	Spontaneous abortion	Primigravida	Previous LSCS	Primigravida	Primigravida
Type of delivery	Vaginal delivery	Emergency LSCS	Emergency LSCS	Vaginal delivery	Emergency LSCS	Emergency LSCS	Emergency LSCS
Delivery complications	No	No	No	No	No	No	No
Newborn	Alive	Alive	Alive	IUD	Alive	Alive	Alive
Clinical onset	1 day prior to delivery	On the day of surgery	On the day of surgery	1-day prior delivery	2 days prior delivery	On the day of surgery	On the day of surgery
Clinical debut	Heart failure	Heart failure	Heart failure	Heart failure	Heart failure	Heart failure	Heart failure
Length of stay in hospital (days)	12	16	10	17	24	16	13

Table 2: Summary of the radiological investigations.

Cases	Chest radiography	Electrocardiograph	Echocardiogram
Case 1	Bilateral non homogenous fluffy opacity	Sinus tachycardia	No regional wall motion abnormality (RWMA) RHD, severe MS, LV EF-60%; mild AR, TR & PAH; dilated LA, LV; MVOA-0.8 cm^2.
Case 2	Bilateral non homogenous fluffy opacity	Sinus tachycardia	Global hypokinesia of left ventricle with normal chambers, dimensions LVEF of 35%; Mild MR/TR/AR/PAH; Moderate LV systolic dysfunction
Case 3	Cardiomegaly	Sinus tachycardia	Severe biventricular systolic and diastolic dysfunction; LVEF 24.2%; Moderate MR, Dilated chamber with global hypokinesia of LV
Case 4	Bilateral pulmonary odema	Sinus tachycardia	Global hypokinesia of LV, normal chamber dimensions, mild MR, severe LV systolic dysfunction, LVEF: 25%
Case 5	Cardiomegaly	Sinus tachycardia	Global hypokinesia of LV, Dilated LV grade II MR, Mild AR/ PR, Moderate TR, Mild PAH, Thin interatrial septum, mild LV systolic dysfunction, No clot/vegetations/PE; LVEF 30-35%.
Case 6	Bilateral homogenous opacity	Sinus tachycardia	Global hypokinesia of LV; normal chamber dimensions; mild MR, AR, TR and PAH; Severe LV systolic dysfunction; moderate bilateral pleural effusion; LVEF 20%.
Case 7	Cardiomegaly	Sinus tachycardia	Global hypokinesia of LV; Normal chamber dimensions, mild MR, AR, PR, and TR; RSVP- 28 mmHg; Severe LV systolic dysfunction; LVEF: 20-25%; no clot/veg; mild pericardial effusion, mild pleural effusion (right and left)

DISCUSSION

In this series, we observed seven cases of peripartum cardiomyopathy (PPCM) presenting during pregnancy or labor. Symptoms included breathlessness, cough, and respiratory distress, sometimes leading to severe complications such as cardiac arrest. Diagnosis was often made post-delivery, confirmed through echocardiography and exclusion of other causes. Treatment primarily involved intensive care management, including oxygen support, medications prescribed by cardiologists, and, in some cases, ventilation. Despite the severity of presentations, all patients showed improvement with treatment and were discharged following stabilization. Peripartum cardiomyopathy (PPCM) is an idiopathic primary myocardial disease that occurs during pregnancy, with its exact etiology remaining unclear, necessitating diagnosis by exclusion.[1,8] Our study supports previous findings from India indicating that PPCM often presents in young primigravid women. While PPCM is typically associated with women at the extremes of childbearing age and higher parity, it is not worthy that 24% to 37% of cases occur in young primigravid individuals. Similar trends were observed in a large cohort from Haiti.[9] Additionally, risk factors such as obesity, smoking, excessive alcohol consumption, malnutrition, and a history of heart disease are significant.[10] Thus, educating patients about these risk factors during the antenatal period is crucial for early identification and prevention.

PPCM continues to be diagnosed through exclusionary methods. No specific criteria have been established to differentiate peripartum patients with new-onset heart failure and left ventricular systolic dysfunction as PPCM from other types of dilated cardiomyopathy. Hence, all alternative causes of dilated cardiomyopathy with heart failure must be thoroughly ruled out before confirming the diagnosis of PPCM.[1] Recent findings from Haiti indicate the possibility of a latent form of PPCM without apparent clinical symptoms.[9] Diagnosis hinges on echocardiographic identification of new-onset left ventricular systolic dysfunction, along with depressed fractional shortening and ejection fraction for a short period during parturition.[7] In our study, fortunately, timely diagnosis through 2D echocardiography could aid significantly in the diagnosis. PPCM presents similarities with other non-ischaemic cardiomyopathies; however, a notable difference lies in the higher rate of spontaneous recovery of ventricular function observed in affected women. Treatment goals focus on reducing afterload and preload while enhancing contractility.[11] ACE inhibitors are typically employed to mitigate afterload through vasodilation post-pregnancy, while hydralazine (with or without nitrates) substitutes ACE inhibitors during gestation due to potential fetal toxicity. β blockers are administered to address high heart rate, arrhythmias, and sudden death risks in PPCM patients. Digitalis, an inotropic agent, is considered safe during pregnancy, aiding contractility and rate control but requires close monitoring to avert adverse outcomes associated with excessive serum digoxin concentrations. Diuretics are utilized safely to diminish preload and alleviate symptoms. Given the heightened thromboembolic risk, heparin use is often deemed necessary, followed by warfarin in individuals with left ventricular ejection fractions below approximately 35%.[8,12]

PPCM typically exhibits a mortality rate of 15-50%.[1,7] In a South African study of 100 PPCM patients, 15% succumbed to the condition, and only 23% achieved normal left ventricular function after six months of optimal medical therapy with ACE inhibitors and β blockers.[12] Similarly, a study in Haiti reported a 15% mortality rate, with 31.5% of patients recovering normal left ventricular function over five years.[9] Timely critical care overseen by cardiologists in our hospital prevented maternal mortality, but majority had to undergo caesarean delivery increasing maternal morbidity. Additionally, the intrauterine death in our observation underscores the importance of considering PPCM in peripartum patients with unexplained illness. A broad index of suspicion is crucial, given the possibility of atypical presentations. Clinical deterioration after discharge, recurrence in the next pregnancy, and high mortality has been observed through follow-up studies.[1] Therefore, in resource-limited settings, prompt referral of suspected PPCM cases to tertiary care centers is essential to access specialized diagnostic tools and expertise, enabling early treatment initiation and complication prevention through collaborative efforts between primary and tertiary care facilities. No follow-up of these women after discharge was made and hence, we could not observe the prognosis and complications among them.

CONCLUSION

In conclusion, our study reaffirms the demographic trends of peripartum cardiomyopathy (PPCM) in young primigravid women, highlighting the need for timely diagnosis and management of the condition. With a focus on exclusionary diagnosis and treatment aimed at reducing afterload, enhancing contractility, and managing thromboembolic risk, collaborative efforts are key for optimizing outcomes and reducing the potential mortality in PPCM.

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REFERENCES

1. Sliwa K, Fett J, Elkayam U. Peripartum cardiomyopathy. Lancet. 2006;368(9536):687-93.
2. Kolte D, Khera S, Aronow WS, Palaniswamy C, Mujib M, Ahn C, et al. Temporal trends in incidence and outcomes of peripartum cardiomyopathy in the United States: a nationwide population-based study. J Am Heart Assoc. 2014;3(3):1056.
3. Abboud J, Murad Y, Chen-Scarabelli C, Saravolatz L, Scarabelli TM. Peripartum cardiomyopathy: a

comprehensive review. Int J Cardiol. 2007;118(3):295-303.
4. Elkayam U. Clinical characteristics of peripartum cardiomyopathy in the United States: diagnosis, prognosis, and management. J Am Coll Cardiol. 2011;58(7):659-70.
5. Demakis JG, Rahimtoola SH, Sutton GC, Meadows WR, Szanto PB, Tobin JR, et al. Natural Course of Peripartum Cardiomyopathy. Circulation. 1971;44(6):1053-61.
6. Elkayam U, Tummala PP, Rao K, Akhter MW, Karaalp IS, Wani OR, et al. Maternal and fetal outcomes of subsequent pregnancies in women with peripartum cardiomyopathy. N Engl J Med. 2001;344(21):1567-71.
7. Braunwald E, Mann DL, Zipes DP, Libby P, Bonow RO. Braunwald's heart disease: A textbook of cardiovascular medicine. In: Braunwald's heart disease: A textbook of cardiovascular medicine. 2024;9:1776-7.
8. Prasad GS, Bhupali A, Prasad S, Patil AN, Deka Y. Peripartum cardiomyopathy – case series. Indian Heart J. 2014;66(2):223-6.
9. Fett JD, Christie LG, Carraway RD, Murphy JG. Five-year prospective study of the incidence and prognosis of peripartum cardiomyopathy at a single institution. Mayo Clin Proc. 2005;80(12):1602–6.
10. Martin S, Short D, Wong CM, McLellan D. A change of heart: case series of peripartum cardiomyopathy. Case Rep Obstet Gynecol. 2013;563158.
11. Murali S, Baldisseri MR. Peripartum cardiomyopathy. Crit Care Med. 2005;(10):340-6.
12. Sliwa K, Förster O, Libhaber E, Fett JD, Sundstrom JB, Hilfiker-Kleiner D, et al. Peripartum cardiomyopathy: inflammatory markers as predictors of outcome in 100 prospectively studied patients. Eur Heart J. 2006;27(4):441-6.

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