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Peripartum Cardiomyopathy: A prospective case series in a tertiary care hospital in Kolkata.

*Dr. Pradip Kumar Saha[1], DCH, MD. Dr. Shreya Sharma[2], MS, DNB. Dr. Kabir Hossain Purkait[*3], MS. Dr. Mandira Dasgupta[4], MS,DNB,MRCOG.*
[1]Associate Professor, [2] Senior Resident, [3]Junior Resident, [4]Prefessor
Department of Obstetrics & Gynaecology, RG Kar Medical College & Hospital.
**Corresponding author.*

Abstract

Objectives: This study aimed to analyze the clinical presentation, management, and outcomes of peripartum cardiomyopathy (PPCM) in a tertiary care setting, emphasizing early diagnosis, multidisciplinary care, and maternal-fetal outcomes.

Methods: A prospective case series of three primiparous women (ages 22–33) diagnosed with PPCM during the third trimester or postpartum. Data included clinical features, echocardiographic findings (LVEF <45%), and therapeutic interventions (diuretics, beta-blockers, ACE inhibitors). Management involved a multidisciplinary team (cardiologists, obstetricians, intensivists), with delivery via cesarean section in all cases.

Results: All patients presented with acute heart failure (NYHA III–IV), tachycardia, and reduced LVEF (24–37%). Risk factors included hypothyroidism (2/3), above 25 years of age (2/3), primi gravida (3/3), pre-eclampsia (1/3) and anemia (2/3). Post-stabilization, two underwent emergency cesarean delivery due to obstetrical complications. At discharge, LVEF improved in one case (24% to 42% at 6 months). No fetal complications were observed.

Conclusion: PPCM demands high clinical suspicion, prompt echocardiography, and tailored therapy. Multidisciplinary care optimizes outcomes, though long-term follow-up is essential to monitor recovery and recurrence risks.

Keywords: Peripartum cardiomyopathy, heart failure, pregnancy, echocardiography, multidisciplinary management.

1. INTRODUCTION

Peripartum cardiomyopathy (PPCM), one of the pregnancy-associated cardiovascular disorders, is a potentially lethal syndrome in a previously healthy woman [1].It is one of the leading causes of maternal morbidity and mortality due to a non-obstetric cause which cannot be prevented and predicted hence early diagnosis and management is vital in preventing maternal mortality.

The European society of Cardiology, 2018 describes PPCM as HF secondary to LV systolic dysfunction towards the end of pregnancy and in the months following delivery, with the majority diagnosed post-partum. Careful history taking is necessary to identify and exclude other causes of HF. The LV may be non-dilated, but the EF is usually <45%. Patients

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frequently present with acute HF, but also with ventricular arrhythmias and/or cardiac arrest. Hence, the diagnosis depends on the temporal relation of the symptoms with pregnancy as well as the exclusion of other cardiomyopathies [2]

The incidence of PPCM varies widely, depending mostly on racial and geographical background of the patients [3]. Data about incidence from India are lacking. A study from a South Indian tertiary care centre showed an incidence of 1:1374 live births but was limited by a low number of women [1].Risk factors for the disease include advanced age, black race, hypertensive disorders, multiple gestation, anaemia, and long tocolysis [4].Its aetiology is unclear and appears to be multifactorial and polygenic. Genetic predisposition, inflammation, autoimmune reaction, oxidative stress, low selenium levels, viral infections, as well as the effects of antiangiogenic factors, are all potential contributory factors. 16 kDa, an antiangiogenic mini sub fragment generated by prolactin hormone oxidative stress, may cause PPCM by inducing endothelial injury and having proapoptotic features [5].

PPCM requires a multidisciplinary team approach comprising of cardiologist, physician, paediatrician, anaesthetist, intensivist and an obstetric team.

2. CASE PRESENTATION

2.1 Case One

A 27-year-old primiparous woman with a history Hypothyroidism and Pre-eclampsia presented at 35 weeks' gestation with sudden onset respiratory distress for a week (NYHA III). On examination, she was tachycardic and was maintaining a saturation of 95 % on 4 lit oxygen. Her ECG done showed sinus tachycardia,Echocardiography done revealed LVEF of 37%,Global hypokinesia of Left Ventricle,Moderate LV systolic dysfunction, Grade II left Ventricular Diastolic Dysfunction with a possibility of peripartum cardiomyopathy. Trop T and NT Pro BNP done was negative. Multidisciplinary team approach including a cardiologist, obstetrician, anaesthetist and paediatrician was taken. She was started on diuretics (Inj.Lasix 20 mg IV BD) and anti-hypertensives (Tab. Labetalol 100 mg TDS) which were titrated according to her BP. Blood Investigations done was suggestive of anaemia (Hb-8.7 gm%)She was planned for Elective C-section after stabilisation of her vitals. However, she had PPROM at 35 weeks 2 days of gestation and she underwent Emergency C - Section and delivered a live boy baby of 2.605 gm, she also received 1-unit PRBC transfusion intra-operatively. Post operatively she was managed conservatively with Anti-hypertensives, Diuretics, Beta Blockersand Anti-coagulants which was titrated accordingly, Hb on post op day 1 was 10.6 gm %. Her ECG post-operatively on Day –6 was within normal limits with Echo showing Global Hypokinesia, with EF of 36%. She was discharged after two weeks. Her 6 month follow up is awaited.

2.2 Case Two

A 23-year-oldprimiparous woman with history of hypothyroidism and shortness of breath for 1 week (NYHA III) presented at 36 weeks of gestation with PPROM. On examination, tachycardia was present with added sounds on auscultation of heart, she was maintaining a saturation of 100% on 4 L oxygen. Her ECG done showed-sinus tachycardia with premature ventricular complexes, ECHO showed-Left ventricular dilatation with severe systolic dysfunction, Left Ventricle generalized wall hypokinesia with paradoxical motion of left ventricular septum, mild pulmonary arterial hypertension, with LVEF -30%.The patient underwent emergency LSCS under GA after consultation with Dept. of cardiology andAnesthesia and delivered a live boy baby of 2.6 kgs. Neonate did not show any congenital

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affection. Post operatively she was started on ACE Inhibitors (Tab. Ramipril 2.5 mg OD), Diuretics (Tab. Lasix 20 mg BD, Tab. Spironolactone 25 mg OD) and Beta Blockers (Tab. MetXL 12.5 mg OD) which were gradually titrated. Repeat Echocardiogram done on post op day 5 showed LVEF-34%, global hypokinesia, moderate MR,TR,other valves normal.IAS/IVS was intact. She was discharged after 2 weeks after stabilization and gradual titration of her medications. 6 months follow up is awaited.

2.3 Case Three

33 years old primigravida with history of fibroid in pregnancy presented at 33 weeks of gestation with complaints of shortness of breath (NYHA IV) along with palpitations for 4 days. On examination she was tachycardic with raised jugular venous pressure.ECG revealed sinus tachycardia. Echocardiographic monitoring showed dilated left ventricle and atrium with global hypokinesia of left ventricle with EF-24%. She was then shifted to ICU where she was conservatively managed with digoxin, diuretics, potassium supplements, heparin and inotropes. After stabilization of heart failure, she underwent an elective Caesarean section at 36 weeks 5 days, under epidural anesthesia and delivered a live female baby of 2.49 kg. Post operatively she developed cardiogenic shock which required ICCU admission and multidisciplinary support. She was discharged after two weeks and asked to be on follow-up. Neonate did not show any congenital affection. Follow-up echocardiography at 6 month showed relatively improved left ventricular systolic function with EF of 42%.For contraception she was advised progesterone only pill.

	CASE1	CASE 2	CASE 3
Age/Parity	**27/ Primi**	**22/ Primi**	**33/ Primi**
Associated comorbidity	**Hypothyroidism, Pre-eclampsia, Anemia**	**Hypothyroidism, Anemia**	**Anemia**
Time of onset of symptoms	**35 weeks/ Sudden**	**36 weeks/ Sudden**	**33 weeks/Sudden**
Presenting complaints	**NYHA-III Respiratory Distress**	**NYHA-III Respiratory Distress**	**NYHA IV Respiratory Distress**
On Examination	**Tachycardic and was maintaining a saturation of 95 % on 4 lit oxygen.**	**Tachycardic, Added sounds on auscultation of heart, maintaining a saturation of 100 % on 4 lit oxygen**	**Tachycardic, Raised JVP, maintaining a saturation of 95 % on 4 lit oxygen.**
ECG Changes/ ECHO	**Sinus tachycardia, LVEF of 37%,**	**Sinus Tachycardia, Premature Ventricular Ectopic, LVEF-30%**	**Sinus Tachycardia, LVEF-24 %**

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Initial Stabilization	**Oxygen, Diuretics and Anti-hypertensives.**	**Oxygen, Diuretics .**	**ICU Care, Digoxin, Diuretics, Potassium supplements, Heparin and Inotropes**
Type of Delivery	**Emergency LSCS**	**Emergency LSCS under GA**	**Elective C-section at 36 weeks 5 days under Epidural Anesthesia**
Post op ECG/ECHO-	**Normal, LVEF-36% on Day-6.**	**Normal, LVEF-34% on post-op Day-5.**	**Post-op LVEF-42 % at 6 months post operatively.**
Post Op Stablisation	**Anti-Hypertensives, Diuretics and Beta blockers.**	**ACE Inhibitors Diuretics and Beta blockers.**	**ACE Inhibitors, Diuretics and Beta blockers.**

Table 1: Presentation and Management of three different cases of Peripartum cardiomyopathy at a tertiary care hospital.

3. DISCUSSION

3.1. Incidence

Known to be an idiopathic myocardial disease associated with pregnancy, data regarding PPCM from India are scarce. Although the actual incidence of PPCM in India is unknown, research conducted in South India revealed an incidence of one case per 1374 live births [1]. In a pooled analysis of seven Indian studies on PPCM, including 221 patients by Agarwal *et al.* the incidence of PPCM in the Indian population was 1 in 1340 live births [4].The highest incidence has been reported from Nigeria (1 in 102 deliveries) and the lowest from Japan (1 in 15,533 births)[6].In the United States, PPCM is known to occur per 4000 live births.[7]. The incidence is known to vary with race and region. However, it is difficult to evaluate ethnic or racial differences in the Indian population in the absence of robust data.

3.2. Sociodemographic Characters

Two of the patients in this case series were above 25 years of age, whilst one was 22 years. According to a study published in 2015 by Davis *et al*. the majority of individuals with PPCM are over the age of 30[8]. In the pool analysis conducted by Agarwal *et al*, the mean age of patients was around 27 years.[4] It is known that an age over 25 years is a risk factor for the development of PPCM, and a mean age of 30 years has been identified [7].
Multiparity and twin pregnancies are important risk factors. In a study conducted by Pandit *et al.* the mean parity was found to be 2.3 + 1 [9] which was comparable with a study by Mishra *et al* 2.6+ 1.[10] and Silwa *et al*. reported a mean parity ranging from 2.6 to 4.3.[11]
Sixty percent of cases had their symptom onset after delivery in the Indian population.[4]

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3.3. Medical,Obstetrical Co-morbidities&other Risk Factors.

Two out of three patients in this case series were suffering from hypothyroidism and one had history of severe preeclampsia.
A pooled analysis by Agarwal et al. identified hypertensive disorders and anaemia in almost half of the patients in the Indian subset. Associated conditions identified were multiple pregnancy in 10% of cases, hypertensive disorders in 46% of cases, anaemia in 45% of cases, gestational diabetes in 9% of cases, and hypothyroidism in around 7.8% of cases.
Obesity, smoking, excessive alcohol consumption, and malnutrition are all potentially modifiable risk factors for the disease, any previous personal or family history of heart disease should alert clinicians to a high-risk pregnancy.[12]

3.4. Clinical Presentation

All the patients in this study presented at third trimester with shortness of breath/ respiratory distress (NYHAIII-IV) lasting for a week with palpitations and light-headedness.
Most women with pre-existing cardiac disease develop symptomatic HF in the 2nd trimester, when the maximal cardiovascular changes occur. In contrast, the majority of women with PPCM typically develop symptoms in the first month postpartum, but it can occur in the third trimester or up to 6 months postpartum [3].Sixty percent of cases had their symptom onset after delivery in the Indian population [4].While most patients present with typical heart failure signs and symptoms, patients can also present with thromboembolic complications, lifethreatening arrhythmias, and even cardiac death[3].
Common symptomatology includes congestive symptoms (dyspnoea on exertion, orthopnoea, paroxysmal nocturnal dyspnoea, dry cough, or pedal oedema) or nonspecific symptoms, such as fatigue, malaise, palpitations, light-headedness, or abdominal discomfort. Diagnosis of PPCM is challenging since symptoms may mimic those encountered in a normal pregnancy. When symptoms persist or are disproportionate to what is expected for pregnancy, PPCM should be suspected and evaluation undertaken.

3.5. Investigations

PPCM is a diagnosis of exclusion. The mainstay of diagnosing and prognosticating PPCM is echocardiographic analysis with demonstration of LV systolic dysfunction after excluding other causes of HF, like valvular heart disease, restrictive and hypertrophic cardiomyopathy. Echocardiogram does not only confirm the diagnosis but also evaluates for other causes of heart failure (valvular disorder or any other structural abnormalities), assesses for complications of PPCM (e.g., LV thrombus), and it also provides prognostic data.
Electrocardiography mostly shows non-specific features such as sinus tachycardia, non-specific ST-T changes and evidence of left atrial or ventricular enlargement, but is useful for detecting and correcting aggravating factors such as arrhythmias.
Chest radiography may show cardiomegaly or signs of congestion (vascular redistribution, pleural effusions, interstitial oedema).
Although echocardiographic finding of LVEF less than 45% is necessary to make the diagnosis of PPCM, other findings can also be present, including left ventricular dilatation, four-chamber enlargement, mitral or tricuspid regurgitation, elevated pulmonary artery pressures, and right ventricular enlargement. A left-ventricular end-systolic diameter of <5.5 cms portends better cure rates and shorter recovery times [3].Conventionally used in

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postpartum period due to foetal toxicity of gadolinium, cardiac magnetic resonance imaging may provide a more precise measurement of chamber volumes and ventricular function than echocardiogram.

3.6. Management

All our cases were managed with a multi-disciplinary team approach, which included an obstetrician, cardiologist, anaesthetist and paediatrician. After ruling out other causes of cardiomyopathies, patients were initially stabilised with oxygen, non -invasive/ invasive ventilation, diuretics, inotropes, anti-hypertensives which were all safe in pregnancy. After initial stabilisation of the patient, Elective C-section was planned for them, however due to obstetrical emergencies, two of them underwent emergency C-section.Post partum they were started on ARBs and medications were titrated gradually. ECG and ECHO were repeated which showed significant improvement in their cardiac status and they were discharged after 2 weeks.

The exact management strategy for PPCM depends on the individual clinical case but is essentially based on the stability of the patient. Acute heart failure and stable heart failure are managed quite differently[12].A multidisciplinary approach involving cardiologist, obstetrician, intensivist, and paediatrician is crucial for management of PPCM patients [3]. Initial management relies on the general guidelines of other forms of nonischaemic cardiomyopathy, but a special attention is required when choosing agents that have a safe profile in the settings of pregnancy and lactation.

Acute heart failure in the peripartum period is managed in just the same way as acute heart failure at any other time of life, with pulmonary oedema and hypoxia treated rapidly with oxygen, non-invasive ventilation if required, and diuretics or nitrates in the volume overloaded. Inotropic support should be used in those with evidence of shock. For patients who continue to deteriorate despite optimal medical management, mechanical circulatory support such as a ventricular assist device (VAD) should be considered. The patients can be supported by either a short- or long-term VAD until recovery of ventricular function has been established. Management strategies for those with stable heart failure in PPCM are dictated by whether the patient is postpartum or still pregnant, with consideration being given to the acceptability of the drugs used to treat heart failure during pregnancy. The drugs used are largely the same as those used for any other form of heart failure. ACE-inhibitors and angiotensin receptor blockers are toxic to the foetus and should be avoided in pregnancy, as should certain diuretics due to the possibility of reduced blood flow to the placenta. Modifications can be made to current regimes to make drugs choices and combinations safe and effective during pregnancy; in particular, beta-blockers have not been proven to be teratogenic. Caution should be exercised when considering drugs for which there is limited data available in pregnancy.[12]

3.7. Delivery

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In women with PPCM with advanced heart failure, prompt delivery can be considered for maternal cardiovascular indications or hemodynamic instability. Planned caesarean delivery is preferred for women with advanced HF requiring inotropic therapy or mechanical circulatory support. Early delivery is not required if the maternal and foetal conditions are stable and most pregnant patients can safely be delivered vaginally [3].

3.8. Post partum period

Women who are clinically stable should not be discouraged from breastfeeding as long as it is compatible with their heart failure medications.
In breast feeding women, beta blockers, enalapril, and spironolactone is compatible with breast feeding. ARBs, Neprilysin inhibitors, and Ivabradine do not have enough information and should be avoided during pregnancy and lactation. Captopril and enalapril were found in clinically insignificant amounts in the breast milk and are deemed to be compatible with breast feeding according to the American Academy of Paediatrics [3]

3.9. Prognosis

In previous reports, baseline LVEDD and LVESD were frequently described as predictors of adverse outcomes in PPCM [14].In the IMAC-2 study, baseline LVEDD was the strongest predictor of LV recovery at 6 months. A smaller LV size at baseline is thought to be associated with a more reversible cardiac pathology.[15]
More than 50% of women recover without complication, with left ventricular systolic function at rest returning to normal [13]. The risk of recurrence in subsequent pregnancy is high, especially if left ventricular function has not returned to normal, and is usually more serious in these women [13]. Women with PPCM with persistent left ventricular (LV) dysfunction or LV ejection fraction (LVEF)$\leq$ 25% at diagnosis are at high risk of recurrent PPCM and should avoid future pregnancy[3]. Some reduction in left ventricular ejection fraction seems inevitable in all patients, though it may be subclinical.
The Task Force on the Management of Cardiovascular Diseases during Pregnancy of the European Society of Cardiology discourages subsequent pregnancy when the LVEF has not returned to the values from before pregnancy. Continuation of guideline-directed medical therapy (GDMT) is recommended for the long term in these patients.[3].There is also a risk of future HF and LV dysfunction even without pregnancy. These patients must remain under regular cardiac care. If subsequent pregnancies are planned, echocardiography should be performed, and dobutamine stress testing may be helpful to determine the contractile reserve and further risk-stratify the potential for recurrence [3]

3.10. Recent Advances

3.10.1 Genetic Study-A genome-wide study has shown a strong association between a gene on chromosome 12 and PPCM [16]
3.10.2 Disorder of Prolactin Cleavage-It has been shown in mice that a product of protein cleavage causes impairment of cardiac myocyte function resulting in PPCM due to its antiangiogenic and proapoptotic properties. This effect was successfully abrogated in these mice by administration of bromocriptine and by inhibiting prolactin secretion and may hold therapeutic promise in humans [12].

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3.10.3 Disordered Immune Response. -Antibodies directed against cardiac tissue have been found in PPCM patients, though it remains unclear whether or not autoimmune disease is causative or if the antibodies are raised when the epitopes are unmasked following cardiac myocyte damage of another
mechanism [17]

3.10.4 Disorder of Cytokine Imbalance- Inflammatory markers including TNF-α, IFN-Y, IL-6, and CRP have been shown to be higher in PPCM than in healthy controls [8], though it is unclear if raised levels of these mediators are causative or reactive. Restoration of cytokine balance may, therefore, be an area that holds some promise for therapy.[18]

4. CONCLUSION

PPCM should be taken into consideration in women who come with characteristics compatible with left ventricular failure because of its rarity, unpredictability in presentation, and possibility for mortality; nevertheless, it should be noted that PPCM can mimic the typical physiological changes associated with pregnancy. Furthermore, cautious cardiac monitoring is necessary once an individual is diagnosed with PPCM.

5. DECLARATION

The authors have no conflict of interest.

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