Original Article

Clinical Profile, Risk Factors, Echocardiographic Findings, and Six-Month Outcomes of Peripartum Cardiomyopathy: A Prospective Observational Study

Raveendra N Mudiyammanavara[1], Ranganatha M[2], K M Prabhuswamy[3], Harish Kumar K[4]

[1]Assistant Professor, Department of General Medicine, Shimoga Institute of Medical Sciences Shimoga, Karnataka, India. [2]Professor, Department of general medicine, Shimoga Institute of Medical Science shimoga, Karnataka, India. [3]Associate Professor, Department of General Medicine, Chamarajanagar Institute of Medical Sciences, Chamarajanagar, Karnataka, India. [4]Junior Resident, Department of General Medicine, Shimoga institute of medical sciences, Shimoga, Karnataka, India

Abstract

Background: Peripartum cardiomyopathy (PPCM) is a rare but potentially life-threatening form of systolic heart failure that occurs in the last month of pregnancy or within five months of delivery in women with no pre-existing structural heart disease. Despite advances in cardiovascular and obstetric care, PPCM remains a significant cause of maternal morbidity and mortality, especially in developing countries where delayed diagnosis and limited access to specialized healthcare facilities may negatively impact outcomes. The clinical presentation is often similar to physiological symptoms of pregnancy, resulting in difficulties in diagnosis and delay in intervention. The objective is to assess the clinical profile, associated risk factors, echocardiographic features, complications and six-month outcomes of patients diagnosed with peripartum cardiomyopathy in a tertiary care centre. **Material and Methods:** A prospective observational study was conducted in 40 consecutive patients diagnosed with PPCM and admitted to the Departments of Obstetrics and Gynecology, General Medicine and Cardiology at Mc Gann Teaching Hospital, Shivamogga from May 2023 to December 2024. We included patients with PPCM who fulfilled the diagnostic criteria and had a left ventricular ejection fraction (LVEF) <45%. Detailed demographic data, clinical presentation, obstetric characteristics, risk factors, electrocardiographic findings and echocardiographic parameters were recorded. Patients were treated with the standard guideline directed heart failure therapy, and followed up for 6 months serial clinical and echocardiography assessment. The primary end-points were recovery of left ventricular function, persistence of symptoms, complications and mortality. **Results:** The mean age of the participants was 28.8 ± 7.13 years. Thirty percent of the participants were in the 24–29-year age group. 65% of the study population were primigravidae. Most patients presented in the postpartum period (57.5%), especially within the first ten days after delivery. The most common presenting symptoms were dyspnea (32.5%) and orthopnea (22.5%). Raised jugular venous pressure (95%) and peripheral edema (80%) were the commonest clinical findings. Seventy-five percent of patients were in NYHA functional class III or IV at presentation. The most common associated risk factors were preeclampsia (45%) and anemia (37.5%). The most common electrocardiographic abnormality was sinus tachycardia (52.5%). The most frequent complication was acute pulmonary edema (27.5%), and thromboembolic events and cardiogenic shock were important contributors to adverse outcomes. Echocardiographic follow-up showed progressive improvement in ventricular function, with 65.7% of patients having LVEF >45% at six months. Complete recovery was observed in 62.5% of patients, partial recovery in 32.5% and mortality was 5%. **Conclusion:** Peripartum cardiomyopathy is common in young women in the peripartum period especially in primigravidae with associated pre-eclampsia and anaemia. Early diagnosis and timely initiation of evidence-based heart failure therapy along with close echocardiographic monitoring lead to significant recovery of ventricular function and improved maternal outcomes. Identification of high risk patients and multidisciplinary management are still essential to reduce the complications and mortality.

Keywords: Peripartum cardiomyopathy, pregnancy-associated heart failure, left ventricular ejection fraction, preeclampsia, echocardiography, maternal outcome.

Received: 03 April 2026 **Revised: 25 April 2026** **Accepted: 10 May 2026** **Published: 07 July 2026**

Introduction

Peripartum cardiomyopathy (PPCM) is a rare but life-threatening form of idiopathic cardiomyopathy characterized by left ventricular systolic dysfunction and the development of heart failure late in pregnancy or in the months after delivery in women without previously known structural heart disease.[1,2] PPCM is characterized by the development of heart failure in the last month of pregnancy or within 5 months of delivery, in the absence of any other identifiable cause of cardiac dysfunction.[2]

Address for correspondence: Dr. **Raveendra N Mudiyammanavara**, Assistant Professor, Department of General Medicine, Shimoga Institute of Medical Sciences Shimoga, Karnataka, India.
E-mail: raveendra.dr@gmail.com

DOI:
10.21276/amit.2026.v13.i2.811

How to cite this article: Mudiyammanavara RN, Ranganatha M, Prabhuswamy KM, Kumar KH. Clinical Profile, Risk Factors, Echocardiographic Findings, and Six-Month Outcomes of Peripartum Cardiomyopathy: A Prospective Observational Study. Acta Med Int. 2026:13(2):XX-XX.

PPCM, although rare, is an important cause of maternal morbidity and mortality worldwide.[3,4] In different geographical regions, the reported incidence ranges from approximately 1 in 100 live births in some African populations to 1 in 1000–4000 live births in Europe and North America.[5] The diagnostic techniques have been improved and awareness has increased, which has led to more recognition of PPCM during the recent decades.[6]

The precise etiology of PPCM is not well defined. Different mechanisms have been proposed, such as viral myocarditis, autoimmune activation, inflammatory responses, oxidative stress, angiogenic imbalance and genetic susceptibility.[3,7] Experimental evidence suggests that oxidative stress may promote cleavage of prolactin into a biologically active 16kDa fragment which exerts antiangiogenic and proapoptotic effects on the myocardium leading to ventricular dysfunction.[8] Other studies have shown endothelial injury, microRNA-mediated pathways and impaired cardioprotective signaling as involved in disease development.[9]

Recent genetic studies have shown that PPCM shares important molecular features with dilated cardiomyopathy. In affected women, variants in genes implicated in familial cardiomyopathy, particularly titin truncating mutations, have been detected.[10–12] The results suggest that pregnancy could be a physiologic stressor that unmasks a latent genetic susceptibility in susceptible women.

Pregnancy presents significant cardiovascular demands on the maternal circulation. Blood volume is about 40–50% higher, cardiac output is markedly increased and systemic vascular resistance is reduced. These physiological changes are generally well tolerated, but may result in myocardial dysfunction in women prone to it.[13] Clinical heart failure may be precipitated by rapid changes in hemodynamics after delivery and hormonal fluctuations that further impair ventricular function.[3]

The clinical presentation of PPCM can be confounded with the physiological symptoms of pregnancy, which makes the diagnosis difficult. Common presenting symptoms include dyspnea, orthopnea, paroxysmal nocturnal dyspnea, pedal edema, fatigue, palpitations and decreased exercise tolerance.[14] Acute pulmonary edema, cardiogenic shock, thromboembolic complications, ventricular arrhythmias or sudden cardiac death may occur in severe cases.[1,14]

Several maternal risk factors have been associated with PPCM [Table 2]. Hypertensive disorders of pregnancy, particularly preeclampsia and eclampsia have been the most consistently reported risk factors.[15,16] Other associations include advanced maternal age, multiple gestations, chronic hypertension, diabetes mellitus, obesity and nutritional deficiencies.[5,16] The close association between PPCM and preeclampsia suggests common pathophysiological pathways, such as endothelial dysfunction and angiogenic imbalance.[15]

Echocardiography continues to be the mainstay of diagnosis and follow-up.[17] Common findings are decreased left ventricular systolic function, global hypokinesia, ventricular dilatation and functional mitral regurgitation.[18,19] The most common measurement used to assess the severity of disease and to forecast recovery is the left ventricular ejection fraction.[20] Management of PPCM is largely based on guideline-directed therapy for heart failure with reduced ejection fraction adapted for pregnancy status.[1,2] Recently promising results have been shown with prolactin inhibition with bromocriptine in selected patients.[21,22] Results are variable, with some patients achieving complete recovery despite advances in treatment, while others progress to chronic heart failure or death.[23]

Prospective data on clinical profile, risk factors, echocardiographic findings and short-term outcomes of PPCM from Indian tertiary care centres is limited. Thus, the present study was done to evaluate the demographic characteristics, clinical presentation, associated risk factors, echocardiographic abnormalities, complications and six-month outcomes in women diagnosed with PPCM.

Materials and Methods

Study Design: This prospective observational study was conducted to evaluate the clinical profile, risk factors, echocardiographic characteristics, and short-term outcomes of patients diagnosed with peripartum cardiomyopathy (PPCM). The study followed eligible participants from the time of diagnosis through a six-month follow-up period to assess clinical recovery, changes in cardiac function, and occurrence of adverse outcomes.

Study Setting: The study was carried out at Mc Gann Teaching Hospital, Shivamogga, Karnataka, India, a tertiary care referral center catering to a large urban and rural population.

Participants were recruited from:

- Department of Obstetrics and Gynecology
- Department of General Medicine
- Department of Cardiology

Women presenting with symptoms suggestive of heart failure during late pregnancy or the postpartum period were screened for eligibility and enrolled after confirmation of PPCM diagnosis.

Study Period

The study was conducted over a period of 18 months from May 2023 to December 2024.

Study Population

The study population comprised pregnant and postpartum women diagnosed with peripartum cardiomyopathy based on clinical evaluation and echocardiographic evidence of left ventricular systolic dysfunction.

All eligible patients presenting during the study period were consecutively enrolled.

Diagnostic Criteria for PPCM

Peripartum cardiomyopathy was diagnosed according to the Heart Failure Association of the European Society of Cardiology criteria, which include:

- Development of heart failure toward the end of pregnancy or within five months postpartum.
- Absence of another identifiable cause of heart failure.
- No prior history of structural heart disease.
- Echocardiographic evidence of left ventricular systolic dysfunction with LVEF <45%.

Sample Size Estimation

The sample size was calculated using the formula:

$$n = \frac{Z^2 p(1-p)}{d^2}$$

Where:
- n = required sample size
- Z = 1.96 corresponding to 95% confidence interval
- p = estimated prevalence (0.45)
- d = absolute precision (0.155)

Substituting the values:

$n = (1.96)^2 \times 0.45 \times (1 - 0.45)/(0.155)^2$

The minimum calculated sample size was 39.63, which was rounded off to 40 participants.

Accordingly, a total of 40 patients fulfilling the eligibility criteria were included in the study.

Sampling Technique

A consecutive sampling technique was employed.

All eligible patients diagnosed with PPCM during the study period and providing informed consent were included until the required sample size was achieved.

Inclusion Criteria

Women were included in the study if they met the following criteria:
- Age ≥18 years.
- Heart failure secondary to left ventricular systolic dysfunction.
- LVEF <45% on echocardiography.
- Onset of symptoms during the last month of pregnancy or within five months postpartum.
- Willingness to participate and provide informed written consent.

Exclusion Criteria

Patients were excluded if they had:
- Known pre-existing cardiac disease.
- Known thyroid disease-causing cardiac dysfunction.
- Congenital heart disease.
- Substance abuse-related cardiomyopathy.
- Other systemic illnesses causing secondary cardiomyopathy.
- Concurrent therapies likely to significantly influence cardiac function independently of PPCM.

Ethical Considerations

The study protocol was reviewed and approved by the Institutional Ethics Committee before commencement.

All participants provided written informed consent prior to enrollment.

Patient confidentiality was maintained throughout the study. Data were anonymized before analysis, and study procedures complied with the ethical principles outlined in the Declaration of Helsinki.

Study Procedure

Baseline Assessment

At enrollment, each participant underwent a comprehensive evaluation including:

Demographic Assessment
- Age
- Educational status
- Socioeconomic status
- Gravidity and parity
- Type of pregnancy (singleton or multifetal)

Clinical Assessment

A detailed history was obtained regarding:
- Dyspnea
- Orthopnea
- Paroxysmal nocturnal dyspnea
- Pedal edema
- Palpitations
- Chest pain
- Fatigue

A thorough physical examination was performed with emphasis on:
- Heart rate
- Blood pressure
- Respiratory rate
- Oxygen saturation
- Jugular venous pressure
- Presence of edema
- Cardiac murmurs
- S3 gallop rhythm
- Pulmonary crepitations

Patients were categorized according to the New York Heart Association (NYHA) functional classification.

Laboratory Investigations

The following investigations were performed as part of routine evaluation:

Hematological Parameters
- Complete blood count
- Hemoglobin concentration
- Total leukocyte count
- Platelet count

Biochemical Parameters
- Renal function tests
- Liver function tests
- Serum electrolytes
- Blood glucose
- Thyroid function tests

These investigations were used to identify associated risk factors and exclude alternative causes of heart failure.

Electrocardiographic Evaluation

A standard 12-lead electrocardiogram (ECG) was obtained in all participants.

The ECG was evaluated for:
- Sinus tachycardia
- Atrial fibrillation
- Conduction abnormalities
- Ventricular hypertrophy
- Repolarization abnormalities
- Arrhythmias

Electrocardiographic findings were correlated with disease severity and outcomes.

Echocardiographic Assessment

Two-dimensional transthoracic echocardiography was performed in all patients at:
- Baseline (diagnosis)
- Three months follow-up
- Six months follow-up

The following parameters were assessed:

Structural Parameters
- Left ventricular end-diastolic diameter (LVEDD)
- Left ventricular end-systolic diameter (LVESD)
- Left ventricular end-diastolic volume (LVEDV)
- Left ventricular end-systolic volume (LVESV)

Functional Parameters
- Left ventricular ejection fraction (LVEF)
- Global left ventricular systolic function
- Left ventricular strain
- Presence of ventricular thrombus
- Valvular abnormalities

Echocardiography served as the primary tool for diagnosis, risk stratification, and monitoring recovery.

Assessment of Risk Factors

The following recognized risk factors for PPCM were systematically evaluated:
- Preeclampsia
- Chronic hypertension
- Anemia
- Multifetal gestation
- Diabetes mellitus
- Hypothyroidism
- Multiparity

Their association with disease severity and outcomes was analyzed.

Treatment Protocol

All patients received guideline-directed medical therapy appropriate to their pregnancy status.

Treatment modalities included:

During Pregnancy
- Diuretics
- Beta-blockers
- Vasodilators when indicated
- Oxygen therapy

Postpartum
- Diuretics
- ACE inhibitors/ARBs
- Beta-blockers
- Mineralocorticoid receptor antagonists
- Anticoagulation when clinically indicated

Treatment decisions were individualized based on hemodynamic status and severity of left ventricular dysfunction.

Follow-Up Protocol

Patients were followed monthly for six months after discharge.

During each visit:

Clinical Evaluation
- NYHA functional class
- Symptoms of heart failure
- Vital parameters
- Drug compliance
- Adverse events

Investigations
- ECG when indicated
- Echocardiography at 3 months
- Echocardiography at 6 months

Medication dosages were adjusted according to clinical response and current heart failure guidelines.

Outcome Measures

Primary Outcome

Recovery of left ventricular systolic function at six months, defined as:
- LVEF >45%
- Significant improvement in NYHA class
- Resolution of heart failure symptoms

Secondary Outcomes
- Mortality
- Cardiogenic shock
- Acute pulmonary edema
- Thromboembolic events
- Persistent left ventricular dysfunction
- Partial recovery
- Need for prolonged hospitalization

Operational Definitions

Complete Recovery

Improvement of LVEF to >45% with resolution of major heart failure symptoms.

Partial Recovery

Improvement in LVEF without normalization of ventricular function.

Persistent Dysfunction

Failure to achieve meaningful improvement in ventricular systolic function after six months.

Adverse Maternal Outcome

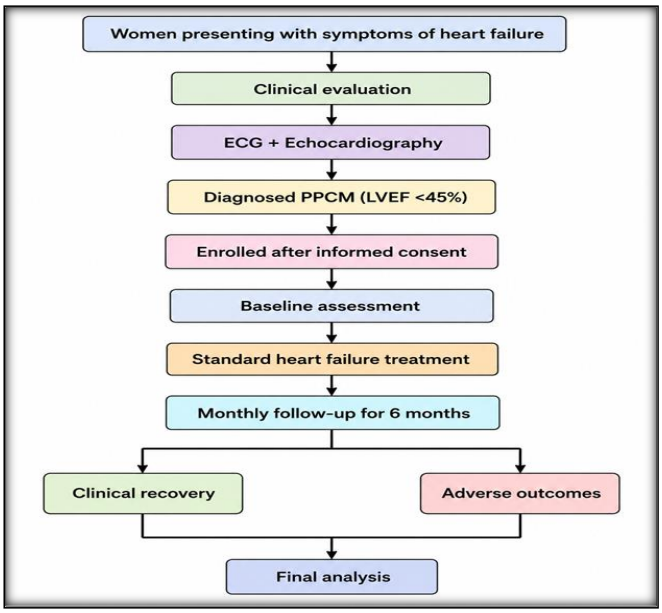
Occurrence of any of the following:
- Death
- Cardiogenic shock
- Thromboembolism
- Severe heart failure requiring intensive care

Statistical Analysis

Data were entered into Microsoft Excel and analyzed using Statistical Package for Social Sciences (SPSS) software.

Descriptive Statistics
- Mean ± standard deviation (SD) for continuous variables.
- Frequencies and percentages for categorical variables.

Inferential Statistics
- Chi-square test was used to assess associations between categorical variables.
- Repeated follow-up comparisons of echocardiographic parameters were evaluated for significance.
- A p-value <0.05 was considered statistically significant.



Results

A total of 40 patients diagnosed with peripartum cardiomyopathy (PPCM) were enrolled and prospectively followed for six months. The study evaluated demographic characteristics, obstetric factors, clinical presentation, risk factors, electrocardiographic and echocardiographic findings, complications, and outcomes.

Demographic Characteristics

Table 1: Age Distribution of Study Participants (n = 40)

Age Group (Years)	Number	Percentage (%)
18–23	11	27.5
24–29	12	30.0
30–35	9	22.5
36–42	8	20.0
Total	40	100.0

Mean Age: 28.8 ± 7.13 years

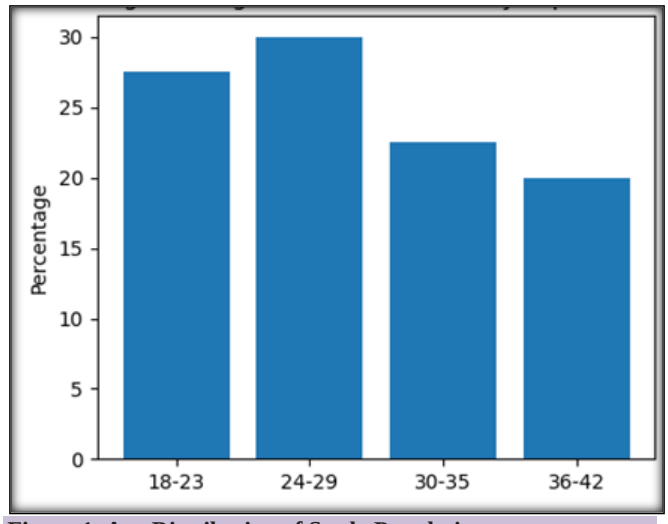


Figure 1. Age Distribution of Study Population

The majority of patients belonged to the 24–29-year age group (30%), followed closely by women aged 18–23 years (27.5%). The mean age of 28.8 years indicates that PPCM predominantly affects young women in the peak reproductive age group. This finding is consistent with previous Indian and international studies that reported mean ages ranging from 27 to 29 years. The clustering of cases in younger age groups suggests that PPCM remains an important cause of maternal cardiovascular morbidity among otherwise healthy reproductive-age women.

More than two-thirds (67.5%) of participants had only primary or secondary education. Lower educational attainment may contribute to delayed healthcare-seeking behavior, reduced awareness of warning symptoms, and inadequate antenatal surveillance. This demographic pattern emphasizes the importance of community-based education programs and early cardiovascular screening in vulnerable populations.

Table 2: Educational Status of Participants

Educational Level	Number	Percentage (%)
Primary School	15	37.5
High School	12	30.0
PUC/12th	8	20.0
Undergraduate Degree	4	10.0
Master's Degree	1	2.5
Total	40	100.0

Table 3: Socioeconomic Status of Participants

Socioeconomic Class	Number	Percentage (%)
Low	20	50.0
Middle	15	37.5
High	5	12.5
Total	40	100.0

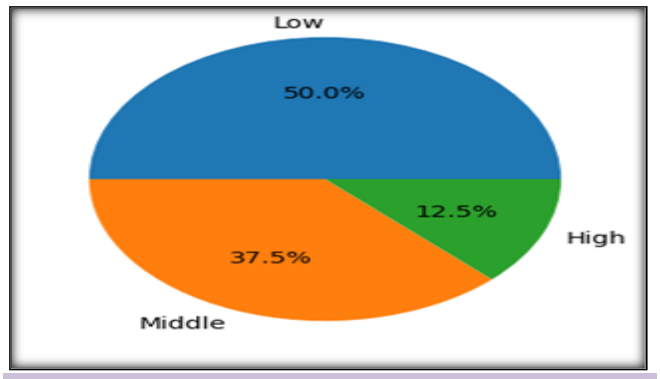


Figure 2. Socioeconomic Distribution

Half of the study population belonged to the lower socioeconomic class, while only 12.5% belonged to higher socioeconomic strata. Economic disadvantage may predispose women to nutritional deficiencies, inadequate antenatal care, untreated anemia, and delayed referral to tertiary care facilities. The findings suggest that socioeconomic factors may indirectly influence both the occurrence and prognosis of PPCM.

Obstetric Characteristics

Table 4: Distribution According to Parity

Parity	Number	Percentage (%)
Primigravida	26	65.0
Multigravida	14	35.0
Total	40	100.0

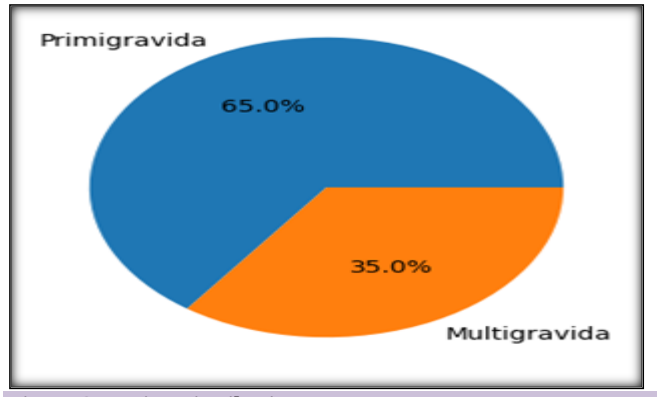


Figure 3. Parity Distribution

Primigravidae constituted nearly two-thirds of all cases. Contrary to traditional descriptions associating PPCM with multiparity, the present study demonstrates a predominance among first pregnancies. Increased cardiovascular stress during the first pregnancy and heightened susceptibility to hypertensive disorders may contribute to this finding. Similar observations have been reported in recent Indian studies.

Table 5: Distribution According to Gestation

Type of Pregnancy	Number	Percentage (%)
Singleton	37	92.5
Twin Pregnancy	3	7.5
Total	40	100.0

Twin gestation was present in 7.5% of participants. Although uncommon, all multifetal pregnancies developed PPCM, supporting previous evidence that increased hemodynamic burden in multifetal gestation may contribute to disease development.

Clinical Presentation

Table 6: Presenting Symptoms

Symptom	Number	Percentage (%)
Dyspnea	13	32.5
Orthopnea	9	22.5
Palpitations	6	15.0
Bilateral Leg Swelling	6	15.0
PND	5	12.5
Chest Pain	1	2.5

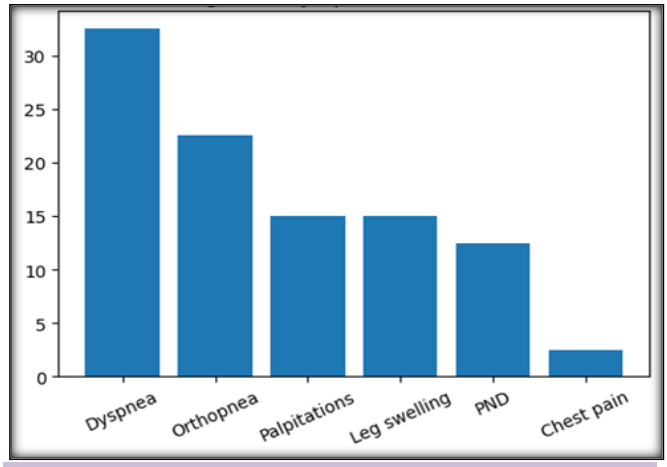


Figure 4: Symptom Distribution

Dyspnea and orthopnea were the dominant presenting symptoms, reflecting elevated left ventricular filling pressures and pulmonary congestion. The predominance of symptoms associated with heart failure underscores the importance of distinguishing PPCM from physiological breathlessness of pregnancy. Failure to recognize these symptoms early may delay diagnosis and increase the risk of severe decompensation.

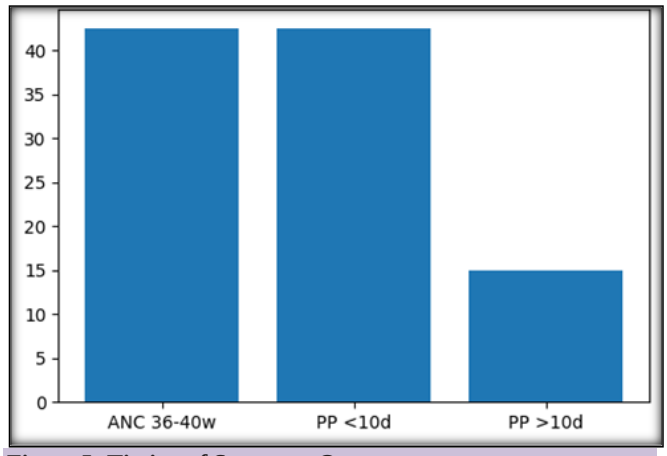


Figure 5: Timing of Symptom Onset.

Table 7: Time of Onset of Symptoms

Time of Onset	Number	Percentage (%)
ANC (36–40 weeks)	17	42.5
PNC (within 10 days)	17	42.5
PNC (11 days–5 months)	6	15.0
Total	40	100.0

More than half (57.5%) of patients developed symptoms during the postpartum period. The immediate postpartum phase is characterized by abrupt hemodynamic shifts and increased preload, which may precipitate overt heart failure in susceptible women. This observation supports the need for continued cardiovascular surveillance even after uncomplicated delivery.

Physical Examination Findings

Table 8: Signs of Heart Failure

Clinical Sign	Number	Percentage (%)
Elevated JVP	38	95.0
Edema	32	80.0
S3 Gallop	20	50.0
Systolic Murmur	18	45.0

Elevated jugular venous pressure was observed in nearly all patients, indicating systemic venous congestion secondary to advanced heart failure. Edema, S3 gallop, and systolic murmurs further support the presence of significant ventricular dysfunction and volume overload.

Functional Status

Table 9: NYHA Functional Class at Presentation

NYHA Class	Number	Percentage (%)
II	10	25.0
III	17	42.5
IV	13	32.5
Total	40	100.0

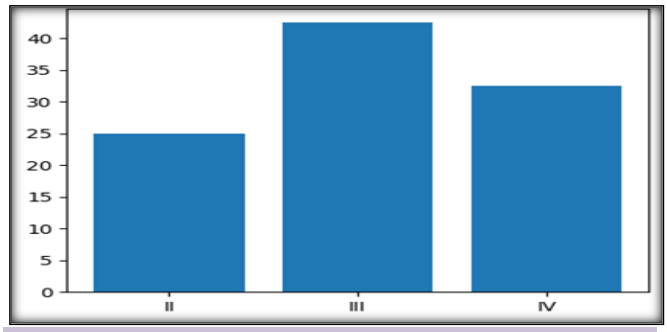


Figure 6: NYHA Functional Class Distribution

Clinical Interpretation

A striking 75% of patients presented with NYHA Class III or IV symptoms, indicating advanced heart failure at diagnosis. This finding highlights delayed recognition and referral, emphasizing the need for improved awareness among obstetricians and primary care physicians.

Table 10: NYHA Functional Class During Follow-Up

NYHA Class	Diagnosis	3 Months	6 Months
I	0	3 (7.8%)	25 (65.7%)
II	10 (25%)	18 (47.3%)	8 (21.1%)
III	17 (42.5%)	11 (28.9%)	5 (13.2%)
IV	13 (32.5%)	6 (15.7%)	0

Serial follow-up demonstrated substantial clinical recovery. By six months, nearly two-thirds of surviving patients achieved NYHA Class I status, reflecting marked symptomatic improvement with evidence-based heart failure therapy and regular monitoring.

Risk Factors

Table 11: Distribution of Risk Factors

Risk Factor	Number	Percentage (%)
Preeclampsia	18	45.0
Anemia	15	37.5
Multifetal Gestation	3	7.5
Chronic Hypertension	2	5.0
Diabetes Mellitus	1	2.5
Hypothyroidism	1	2.5

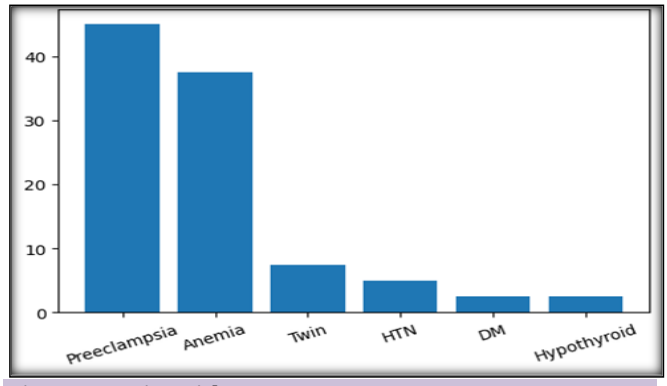


Figure 7: Major Risk Factors

Preeclampsia emerged as the most important risk factor, followed by anemia. The strong association between hypertensive disorders and PPCM supports current evidence suggesting shared antiangiogenic and endothelial dysfunction pathways. The coexistence of anemia may further increase myocardial workload and worsen cardiac reserve, thereby contributing to disease severity.

Echocardiographic Outcomes

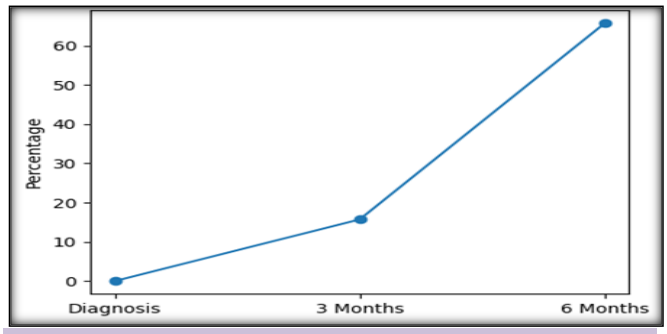


Figure 8: Improvement in LVEF.

Table 12. Left Ventricular Ejection Fraction (LVEF) During Follow-Up

LVEF (%)	Diagnosis	3 Months	6 Months
<30	3 (7.5%)	1	0
30–35	12 (30%)	5	0
36–40	16 (40%)	14	4
40–45	9 (22.5%)	12	9
>45	0	6 (15.7%)	25 (65.7%)

A progressive improvement in left ventricular systolic function was observed throughout follow-up. While only 15.7% achieved recovery by three months, nearly two-thirds recovered normal ventricular function by six months. These findings emphasize the reversible nature of PPCM when diagnosed early and treated appropriately.

Complications and Outcomes

Table 13: Major Complications

Complication	Percentage (%)
Acute Pulmonary Edema	27.5
Cardiogenic Shock	15.0
Thromboembolic Events	30.0

Acute pulmonary edema represented the most common immediate complication. The high incidence of thromboembolic phenomena reflects the combined effects of pregnancy-related hypercoagulability and severe left ventricular dysfunction. These complications significantly influenced maternal morbidity and necessitated intensive monitoring.

Table 14: Final Maternal Outcomes at Six Months

Outcome	Number	Percentage (%)
Complete Recovery	25	62.5
Partial Recovery	13	32.5
Death	2	5.0
Total	40	100.0

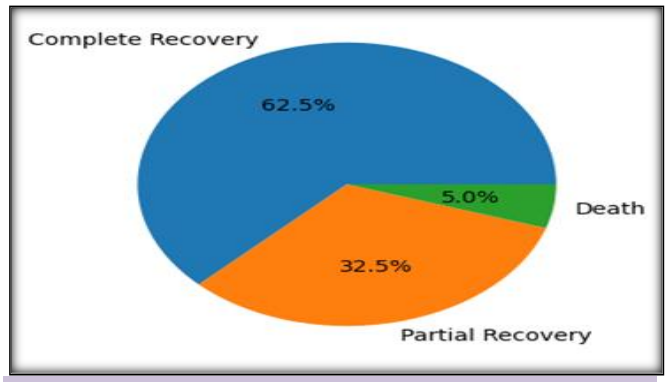


Figure 9: Six-Month Outcomes

The recovery rate of 62.5% demonstrates favorable outcomes with timely diagnosis and standard heart failure therapy. Nevertheless, mortality remained significant at 5%, underscoring the potentially fatal nature of PPCM. Patients presenting with severe LV dysfunction, cardiogenic shock, or thromboembolic complications were more likely to experience adverse outcomes.

Discussion

Present prospective observational study was to evaluate the clinical profile, risk factors, echocardiographic findings, complications and six months outcomes of women diagnosed with PPCM. The findings offer important insight into disease patterns and outcomes in a tertiary care setting.

The mean age of patients in this study was 28.8 ± 7.13 years and most patients were in the 24–29-year age group. The age distribution was comparable to that reported in previous studies from developed and developing countries.[23,24] The findings indicate that PPCM primarily targets women in their prime reproductive years when pregnancy-related cardiovascular stress is at its peak.

65% of the study population were primigravidae. Multiparity has been traditionally considered as a risk factor but recent studies have increasingly reported a large proportion of cases occurring during first pregnancies.[24,25] This finding underscores the need for clinical vigilance irrespective of parity status.

In the postpartum period, symptoms developed in over half of the patients. International registries and observational studies have also described similar observations, identifying the immediate postpartum period as the time of greatest vulnerability.[6,24] Changes in hemodynamics after delivery can unmask subclinical ventricular dysfunction and lead to overt heart failure.

Dyspnea and orthopnea were the most common presenting symptoms in this study. These findings are in line with earlier reports which identified symptoms of congestive heart failure as the main clinical presentation of PPCM.[14,25] Additionally, three-quarters of the patients had NYHA Class III or IV symptoms, meaning that they were diagnosed with advanced disease.

The most relevant associated risk factor was preeclampsia in 45% of the patients. This finding is consistent with systematic reviews and registry data that have demonstrated a strong association between hypertensive disorders of pregnancy and PPCM.[15,16] This association can be explained by common mechanisms such as endothelial dysfunction, angiogenic imbalance, oxidative stress and inflammatory activation.[15] The second most frequent risk factor was anemia, which may have contributed to increased myocardial workload and worsening cardiac reserve.

The most common electrocardiographic abnormality was sinus tachycardia. Similar findings have been published in registry-based studies where tachycardia was associated with the severity of the disease and ventricular dysfunction.[26] ECG findings are often nonspecific. Persistent tachycardia in a pregnant or postpartum woman requires a detailed cardiovascular work-up.

Serial echocardiographic assessment revealed significant improvement of ventricular systolic function during follow-up. At six months, the majority of patients had recovered LVEF above 45 %. Similar rates of recovery have been reported in the IPAC study and other multicenter registries.[20,23] These findings support the concept that PPCM is a potentially reversible cardiomyopathy if recognized early and treated appropriately.

The main complications observed were, acute pulmonary edema, thromboembolic events and cardiogenic shock. These complications have been consistently reported as important determinants of adverse maternal outcomes in PPCM.[22,27] Pregnancy associated hypercoagulability with severe ventricular dysfunction markedly increases the risk of thromboembolic events and contributes to maternal morbidity.

The overall prognosis observed in this study was favorable, with complete recovery observed in almost two-thirds of patients and mortality being limited to 5%. These results are more favorable than those reported in previous studies with higher mortality rates.[23–25] The positive results in this cohort were likely due to early diagnosis, guideline-driven therapy, multidisciplinary approach, and regular follow-up.

In summary, the findings of the current study underline the importance of a high index of suspicion for PPCM in pregnant and postpartum women who present with symptoms of unexplained heart failure. Early echocardiographic evaluation, early introduction of optimal treatment and structured follow up are essential for the optimization of maternal outcome and recovery of ventricular function.

Conclusion

Peripartum cardiomyopathy is an uncommon but potentially fatal cardiac disease that continues to pose important diagnostic and therapeutic problems in maternal health care. The present prospective observational study proved that PPCM is mainly seen in the young women of the reproductive age group especially in primigravidae and is strongly associated with hypertensive disorders of pregnancy and anemia.

Dyspnea and orthopnea were the most frequent presenting symptoms, while elevated jugular venous pressure and peripheral edema were the most common clinical signs. Most patients presented with symptoms of advanced heart failure, which underscores the need for increased clinical suspicion among health care providers. Preeclampsia was the most important associated risk factor, followed by anemia, emphasizing the need for close cardiovascular monitoring in these high-risk groups.

Serial echocardiographic evaluation revealed a significant improvement in left ventricular systolic function during follow-up. More than 60% of the patients had complete recovery within 6 months indicating that PPCM is potentially reversible if diagnosed early and treated properly. However, the incidence of acute pulmonary edema, thromboembolic complications, cardiogenic shock and mortality underlines the severity of the disease.

The findings of this study reinforce the significance of early diagnosis, multidisciplinary management, guideline-directed heart failure therapy, and structured follow-up to improve maternal outcomes. Increased awareness among obstetricians, physicians and cardiologists may help in timely diagnosis and management and thus reduce PPCM related morbidity and mortality.

Strengths of the Study

1. This study was conducted prospectively, allowing systematic collection of clinical and echocardiographic data.
2. Serial follow-up over six months enabled assessment of dynamic changes in cardiac function and clinical recovery.
3. Comprehensive evaluation of demographic, clinical, obstetric, electrocardiographic, and echocardiographic parameters provided a holistic understanding of PPCM.
4. The study generated valuable data from an Indian tertiary care setting, contributing to the limited literature available from this region.

5. Uniform diagnostic criteria and standardized echocardiographic assessment enhanced the reliability of findings.

Limitations of the study

1. The study was conducted at a single tertiary care center, which may limit generalizability of the findings.
2. The sample size was relatively small (n=40), reducing statistical power for subgroup analyses.
3. Follow-up was limited to six months; therefore, long-term outcomes and recurrence rates could not be evaluated.
4. Genetic testing and biomarker analysis were not performed, preventing assessment of underlying molecular mechanisms.
5. Fetal and neonatal outcomes were not included in the study.
6. Advanced imaging modalities such as cardiac magnetic resonance imaging were not routinely available for all patients.

Clinical Implications

1. Healthcare providers should maintain a high index of suspicion for PPCM in pregnant and postpartum women presenting with unexplained dyspnea, orthopnea, edema, or persistent tachycardia.
2. Women with preeclampsia, anemia, multifetal gestation, or other recognized risk factors require closer cardiovascular surveillance during late pregnancy and the postpartum period.
3. Early echocardiographic evaluation should be performed whenever PPCM is suspected, as timely diagnosis significantly influences prognosis.
4. Guideline-directed heart failure therapy and multidisciplinary management involving obstetricians, cardiologists, intensivists, and physicians are essential for improving outcomes.
5. Structured follow-up with serial assessment of ventricular function is crucial for monitoring recovery and guiding future pregnancy counseling.
6. Effective contraception and preconception counseling should be emphasized, particularly in women with persistent left ventricular dysfunction.

Future Recommendations

1. Large multicentric prospective studies are required to better define prognostic factors and regional disease patterns.
2. Longer follow-up periods are necessary to evaluate long-term ventricular recovery, recurrence, and survival.
3. Genetic studies should be incorporated to identify hereditary predisposition and potential molecular targets.
4. Research exploring novel biomarkers and advanced imaging modalities may improve risk stratification and early diagnosis.
5. Future studies should include fetal and neonatal outcomes to provide a comprehensive understanding of disease impact.

Acknowledgements

The authors express their sincere gratitude to the Department of General Medicine, Department of Cardiology, and Department of Obstetrics and Gynecology, Mc Gann Teaching Hospital, Shivamogga, for their invaluable support and cooperation throughout the study. The authors also thank all participating patients and their families for their willingness to contribute to this research.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Bauersachs J, Arrigo M, Hilfiker-Kleiner D, Veltmann C, Coats AJS, Crespo-Leiro MG, et al. Current management of patients with severe acute peripartum cardiomyopathy: practical guidance from the Heart Failure Association of the European Society of Cardiology Study Group on peripartum cardiomyopathy. Eur J Heart Fail. 2016;18(9):1096-105.
2. Sliwa K, Hilfiker-Kleiner D, Petrie MC, Mebazaa A, Pieske B, Buchmann E, et al. Current state of knowledge on aetiology, diagnosis, management and therapy of peripartum cardiomyopathy: a position statement from the Heart Failure Association of the European Society of Cardiology Working Group on peripartum cardiomyopathy. Eur J Heart Fail. 2010;12(8):767-78.
3. Hilfiker-Kleiner D, Haghikia A, Nonhoff J, Bauersachs J. Peripartum cardiomyopathy: current management and future perspectives. Eur Heart J. 2015;36(18):1090-7.
4. Abboud J, Murad Y, Chen-Scarabelli C, Saravolatz L, Scarabelli TM. Peripartum cardiomyopathy: a comprehensive review. Int J Cardiol. 2007;118(3):295-303.
5. Johnson-Coyle L, Jensen L, Sobey A. Peripartum cardiomyopathy: review and practice guidelines. Am J Crit Care. 2012;21(2):89-98.
6. Sliwa K, Mebazaa A, Hilfiker-Kleiner D, Petrie MC, Maggioni AP, Laroche C, et al. Clinical characteristics of patients from the worldwide registry on peripartum cardiomyopathy (PPCM): EURObservational Research Programme in conjunction with the Heart Failure Association of the European Society of Cardiology Study Group on PPCM. Eur J Heart Fail. 2017;19(9):1131-41.
7. Isogai T, Kamiya CA. Worldwide incidence of peripartum cardiomyopathy and overall maternal mortality. Int Heart J. 2019;60(3):503-11.
8. Afana M, Brinjikji W, Kao D, Jackson E, Maddox TM, Childers D, et al. Characteristics and in-hospital outcomes of peripartum cardiomyopathy diagnosed during delivery in the United States from the Nationwide Inpatient Sample database. J Card Fail. 2016;22(7):512-9.
9. Ijaz SH, Jamal S, Minhas AMK, Sheikh AB, Nazir S, Khan MS, et al. Trends in characteristics and outcomes of peripartum cardiomyopathy hospitalizations in the United States between 2004 and 2018. Am J Cardiol. 2022;168:142-50.
10. Bello N, Arany Z. Molecular mechanisms of peripartum cardiomyopathy: a vascular/hormonal hypothesis. Trends Cardiovasc Med. 2015;25(6):499-504.
11. Hilfiker-Kleiner D, Kaminski K, Podewski E, Bonda T, Schaefer A, Sliwa K, et al. A cathepsin D-cleaved 16-kDa form of prolactin mediates postpartum cardiomyopathy. Cell. 2007;128(3):589-600.
12. Ricke-Hoch M, Pfeffer TJ, Hilfiker-Kleiner D. Peripartum cardiomyopathy: basic mechanisms and hope for new therapies. Cardiovasc Res. 2020;116(3):520-31.
13. Bauersachs J, König T, van der Meer P, Petrie MC, Hilfiker-Kleiner D, Mbakwem A, et al. Pathophysiology, diagnosis and

management of peripartum cardiomyopathy: a position statement from the Heart Failure Association of the European Society of Cardiology Study Group on Peripartum Cardiomyopathy. Eur J Heart Fail. 2019;21(7):827-43.
14. Pearson GD, Veille JC, Rahimtoola S, Hsia J, Oakley CM, Hosenpud JD, et al. Peripartum cardiomyopathy: National Heart, Lung, and Blood Institute and Office of Rare Diseases workshop recommendations and review. JAMA. 2000;283(9):1183-8.
15. Bello N, Rendon ISH, Arany Z. The relationship between pre-eclampsia and peripartum cardiomyopathy: a systematic review and meta-analysis. J Am Coll Cardiol. 2013;62(18):1715-23.
16. Jackson AM, Petrie MC, Frogoudaki A, Laroche C, Gustafsson F, Ibrahim B, et al. Hypertensive disorders in women with peripartum cardiomyopathy: insights from the ESC EORP PPCM Registry. Eur J Heart Fail. 2021;23(12):2058-69.
17. Ware JS, Li J, Mazaika E, Yasso CM, Souza T, Cappola TP, et al. Shared genetic predisposition in peripartum and dilated cardiomyopathies. N Engl J Med. 2016;374(3):233-41.
18. Goli R, Li J, Brandimarto J, Levine LD, Riis V, McAfee Q, et al. Genetic and phenotypic landscape of peripartum cardiomyopathy. Circulation. 2021;143(19):1852-62.
19. Spracklen TF, Chakafana G, Schwartz PJ, Kotta MC, Shaboodien G, Ntusi NAB, et al. Genetics of peripartum cardiomyopathy: current knowledge, future directions and clinical implications. Genes (Basel). 2021;12(1):103.
20. Avila WS, Alexandre ERG, Castro ML, Lucena AJG, Marques-Santos C, Freire CMV, et al. Brazilian Cardiology Society statement for management of pregnancy and family planning in women with heart disease – 2020. Arq Bras Cardiol. 2020;114(5):849-942.
21. Sugahara M, Kagiyama N, Hasselberg NE, Blauwet LA, Briller J, Cooper LT, et al. Global left ventricular strain at presentation is associated with subsequent recovery in patients with peripartum cardiomyopathy. J Am Soc Echocardiogr. 2019;32(12):1565-73.
22. Hilfiker-Kleiner D, Haghikia A, Berliner D, Vogel-Claussen J, Schwab J, Franke A, et al. Bromocriptine for the treatment of peripartum cardiomyopathy: a multicentre randomized study. Eur Heart J. 2017;38(35):2671-79.
23. Hoevelmann J, Engel ME, Muller E, Hohlfeld A, Böhm M, Sliwa K, et al. A global perspective on the management and outcomes of peripartum cardiomyopathy: a systematic review and meta-analysis. Eur J Heart Fail. 2022;24(9):1719-36.
24. McNamara DM, Elkayam U, Alharethi R, Damp J, Hsich E, Ewald G, et al. Clinical outcomes for peripartum cardiomyopathy in North America: results of the IPAC Study. J Am Coll Cardiol. 2015;66(8):905-14.
25. Binu AJ, Rajan SJ, Rathore S, Beck M, Regi A, Thomson VS, Sathyendra S. Peripartum cardiomyopathy: an analysis of clinical profiles and outcomes from a tertiary care centre in southern India. Obstet Med. 2020;13(4):179-84.
26. Mumtaz A, Kela D, Rai S, Agrawal V. Peripartum cardiomyopathy: an analysis of clinical profiles and outcomes from a tertiary care center in Northern India. J Indian Coll Cardiol. 2024;14(3):92-96.
27. Mbakwem AC, Bauersachs J, Viljoen C, Hoevelmann J, van der Meer P, Petrie MC, et al. Electrocardiographic features and their echocardiographic correlates in peripartum cardiomyopathy: results from the ESC EORP PPCM Registry. ESC Heart Fail. 2021;8(2):879-89.