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Original Article

Association of Peripartum Cardiomyopathy with Preeclampsia and Maternal Outcomes

Mst Sharmin Ferdous[1*], Khodeza Khatun[2], Suraiya Khatun[3], Shamsunnahar Elora[4], Afroza Sultana[5], Jannati Nuzula[6], Yasmin Akter Jony[7]

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*Corresponding author

ABSTRACT

Background: Peripartum cardiomyopathy (PPCM) is a rare but potentially life-threatening form of heart failure occurring in late pregnancy or the early postpartum period. Preeclampsia has been implicated as a risk factor for more severe cardiac dysfunction and adverse maternal outcomes in PPCM, but data on its impact in South Asian populations remain limited. **Aim of the study**: To evaluate the prevalence and characteristics of preeclampsia among women with PPCM and to investigate its association with cardiac dysfunction and maternal outcomes. **Methods & Materials**: This observational study included 150 women diagnosed with PPCM caried outat the Department of Fetomatanal Medicine, Bnagladesh Medical University, Dhaka, Bangladesh from January 2024 to December 2025. Demographic, obstetric, clinical, and echocardiographic data were collected. Preeclampsia was defined according to international criteria and categorized by severity and timing of onset. Maternal outcomes were monitored during hospitalization and at six months follow-up, with left ventricular function recovery classified as complete (LVEF ≥50%), partial (LVEF 35–49%), or persistent dysfunction (LVEF <35%). Multivariable logistic regression identified predictors of adverse maternal outcomes. **Result**: The mean maternal age was 31.8 ± 5.4 years, and 60% were multigravida. PPCM was predominantly diagnosed postpartum (63.3%) at 36.2 ± 3.1 weeks, with cesarean delivery in 58.7% of cases. Preeclampsia occurred in 33.3% of women, including 20% with severe disease. Mean LVEF at diagnosis was 36.8 ± 6.2%, with 40% presenting LVEF <35%. Patients with preeclampsia had significantly lower LVEF (34.2 ± 5.5% vs. 37.7 ± 6.1%, p=0.002) and higher NYHA class III–IV prevalence (44% vs. 28%, p=0.03). Acute heart failure was the most common maternal complication (68%), with maternal mortality of 5.3%. Multivariable analysis identified preeclampsia (AOR 2.45, p=0.024), severe preeclampsia (AOR 3.12, p=0.008), LVEF <35% (AOR 4.78, p<0.001), and NYHA class III–IV (AOR 3.65, p=0.004) as independent predictors of adverse outcomes. At six months, 45.3% achieved complete recovery, 38.7% partial recovery, and 14.7% had persistent LV dysfunction. **Conclusion**: Preeclampsia significantly exacerbates cardiac dysfunction in PPCM and is associated with increased risk of adverse maternal outcomes. Early recognition, echocardiographic assessment, and intensive management are crucial for improving maternal prognosis and left ventricular recovery.

Keywords: *Peripartum cardiomyopathy, Preeclampsia, Left ventricular ejection fraction, Maternal outcomes, Heart failure, NYHA class*

1. *Assistant Professor, Department of Fetomatanal Medicine, Bangladesh Medical University, Dhaka, Bangladesh (ORCID: 0009-0004-5218-2333)*
2. *Associate Professor, Department of Obstetrics and Gynecology, Bnagladesh Medical University, Dhaka, Bangladesh*
3. *Assistant Professor, Department of Obstetrics and Gynecology, Bnagladesh Medical University, Dhaka, Bangladesh*
4. *IMO,Department of Obstetrics and Gynecology, Shaheed Suhrawardy Medical College Hospital, Dhaka, Bangladesh*
5. *Assistant Professor, Department of Obstetrics and Gynecology, OSD,DGHS,Dhaka, Bangladesh*
6. *Resident, Department of Fetomatanal Medicine, Bangladesh Medical University, Dhaka, Bangladesh*
7. *Resident, Department of Fetomatanal Medicine, Bangladesh Medical University, Dhaka, Bangladesh*

INTRODUCTION

Peripartum cardiomyopathy (PPCM) is a rare but serious form of heart failure characterized by new-onset left ventricular systolic dysfunction occurring in the last month of pregnancy or within months following delivery in women with no previous history of heart disease [1]. PPCM affects approximately 0.03-0.05% of all pregnancies worldwide [2,3]. In Bangladesh, the prevalence rate with reported frequencies is approximately 0.1-0.3% among obstetric admissions [4]. PPCM is closely linked to the physiological stress imposed on the maternal cardiovascular system during pregnancy. Normal pregnancy involves increased blood volume, cardiac output, and hormonal changes that require substantial myocardial adaptation [3]. Pregnancy places extra strain on the mother's heart by increasing blood volume and cardiac output, which raises the workload of the myocardium. These normal changes require the heart to adapt effectively to maintain adequate circulation during pregnancy [5]. Whereas, most women adapt successfully, a subset experiences maladaptive cardiac remodeling that leads to ventricular dilation and reduced contractility [6]. The pathogenesis of PPCM is increasingly recognized as multifactorial. Oxidative stress, systemic inflammation, endothelial injury, and vascular dysfunction contribute to progressive myocardial damage and contractile failure [7]. Despite these advances, the precise etiology remains incompletely defined, with proposed mechanisms including viral myocarditis, autoimmune activation, and fetal microchimerism [5].A strong association between PPCM and hypertensive disorders of pregnancy—particularly preeclampsia—has been consistently observed. Historical observations dating back to the 1930s identified hypertension as a frequent antecedent of postpartum heart failure [8]. Subsequent clinical studies confirmed a high prevalence of preeclampsia among women with PPCM, a finding reinforced by contemporary meta-analyses demonstrating a markedly increased risk [9]. Preeclampsia is a pregnancy-related condition marked by high blood pressure and organ problems after 20 weeks of pregnancy. It is one of the strongest risk factors for developing peripartum cardiomyopathy [10]. About 20-25% of women with PPCM also develop preeclampsia [5]. This is much higher than in the general pregnant population [11]. The two conditions likely develop through similar disease processes that affect the

blood vessels. Emerging mechanistic data implicate shared pathways involving anti-angiogenic and cardiotoxic mediators, including soluble fms-like tyrosine kinase-1 and the cleaved 16-kDa prolactin fragment, supporting a common biological link between PPCM and hypertensive disorders of pregnancy [12].These processes include damage to the vessel lining and an imbalance of factors that control blood vessel growth. This leads to impaired function of both the placenta and the heart muscle, increasing health risks for the mother and baby [13]. PPCM can have serious effects on a mother's health. Women with PPCM are more likely to have heart failure, irregular heartbeat, blood clots, longer hospital stays, and even death [14]. When PPCM and preeclampsia occur together, mothers often face more serious health problems. Babies are also more likely to be born early or have low birth weight [15]. Early diagnosis remains challenging because of symptom overlap with normal pregnancy and limited access to cardiac imaging in resource-constrained settings [14]. PPCM and preeclampsia are connected and also can improve patient care. It can help doctors find high-risk women earlier and provide better treatment. The contribution of preeclampsia to PPCM and maternal outcomes provide information about prevention and management strategies in high-burden settings. However, problems like under-reporting and differences in diagnosis still make research challenging [6]. The study aimed to evaluate the association between peripartum cardiomyopathy and preeclampsia and to assess maternal outcomes among affected women.

METHODS & MATERIALS

This hospital-based observational study was carried out at the Department of Fetomatanal Medicine, Bnagladesh Medical University, Dhaka, Bangladesh from January 2024 to December 2025. A total of 150 women fulfilling the diagnostic criteria for PPCM were consecutively enrolled.

Inclusion Criteria

- Women diagnosed with peripartum cardiomyopathy, defined as new-onset heart failure with left ventricular ejection fraction <45% occurring in the last month of pregnancy or within five months postpartum.
- Absence of known pre-existing structural heart disease before pregnancy.
- Availability of complete clinical, obstetric, and echocardiographic data.
- Willingness to provide informed consent.

Exclusion Criteria

- History of cardiomyopathy or chronic heart disease prior to pregnancy.
- Presence of significant valvular or congenital heart disease.
- Heart failure attributable to identifiable secondary causes.
- Incomplete medical records or loss to follow-up at initial assessment.

Data collection and variables

Demographic, obstetric, and clinical data were collected using a structured data collection form. Baseline variables included maternal age, parity, gestational age at PPCM diagnosis, timing of diagnosis (antepartum or postpartum), mode of delivery, multiple gestation, history of hypertension, and family history of cardiomyopathy. Preeclampsia was diagnosed based on standard international criteria as new-onset hypertension after 20 weeks of gestation accompanied by proteinuria and/or end-organ dysfunction. Severity (mild or severe) and timing of onset (early <34 weeks or late ≥34 weeks) were documented. Associated complications such as HELLP syndrome and eclampsia were recorded.

Echocardiographic assessment

Transthoracic echocardiography was performed at the time of PPCM diagnosis using standardized protocols. Measurements included LVEF (Simpson's biplane method), left ventricular end-diastolic and end-systolic diameters, fractional shortening, presence of mitral regurgitation, and detection of left ventricular thrombus. Cardiac functional status was assessed using the New York Heart Association (NYHA) functional classification.

Maternal outcomes and follow-up

Maternal clinical outcomes assessed during hospitalization included acute heart failure, cardiogenic shock, need for inotropic support, mechanical ventilation, ICU admission, thromboembolic events, and maternal mortality. Patients were followed for six months after diagnosis, and left ventricular functional recovery was categorized as complete (LVEF ≥50%), partial (LVEF 35–49%), or persistent dysfunction (LVEF <35%).

Statistical analysis

Data were analyzed using SPSS version 26. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. Comparisons between women with and without preeclampsia were performed using the independent t-test for continuous variables and the chi-square or Fisher's exact test for categorical variables, as appropriate. Multivariable logistic regression analysis was conducted to identify independent predictors of adverse maternal outcomes, with results expressed as adjusted odds ratios (ORs) and 95% confidence intervals (CIs). A p-value <0.05 was considered statistically significant.

Ethical considerations

The study was approved by the institutional ethics committee, and the research was conducted in accordance with the Declaration of Helsinki. Informed consent was obtained from all participants or their legal guardians prior to inclusion in the study.

RESULT

The mean±SD maternal age was 31.8±5.4 years, with 60.0% multigravida. PPCM was the mostly diagnosed postpartum (63.3%) at 36.2±3.1 weeks. Cesarean delivery occurred the most (58.7%) with 41.33% in vaginal delivery, twin pregnancies in 3.3%, 12% had a history of hypertension, and 2.7% had a family history of cardiomyopathy (Table 1).

Table I
Baseline demographic and obstetric characteristics of the study population (*n* = 150)

Variable	Frequency (n)	Percentage (%)
Maternal age (years)		
Mean ± SD	31.8 ± 5.4	
Parity		
Primigravida	60	40.00
Multigravida	90	60.00
Gestational age at PPCM diagnosis (weeks)		
Mean ± SD	36.2 ± 3.1	
Timing of PPCM diagnosis		
Antepartum	55	36.67
Postpartum	95	63.33
Mode of delivery		
Cesarean section	88	58.67
Vaginal delivery	62	41.33
Other relevant characteristics		
Twin pregnancy	5	3.33
History of hypertension	18	12.00
Family history of cardiomyopathy	4	2.67

Preeclampsia was observed in 33.3% of cases, with 13.33% classified as mild and 20.0% as severe. Early-onset (<34 weeks) preeclampsia occurred in 10.0%, while late-onset (≥34 weeks) accounted for 23.3%. Complications included HELLP syndrome in 5.3% and eclampsia in 2.0% of patients (*Table II*).

Table II
Prevalence and characteristics of preeclampsia among patients with PPCM.

Characteristic	Frequency (n)	Percentage (%)
Any preeclampsia	50	33.33
Severity of preeclampsia		
Mild	20	13.33
Severe	30	20.00
Timing of onset		
Early-onset (<34 weeks)	15	10.00
Late-onset (≥34 weeks)	35	23.33
Complications		
HELLP syndrome	8	5.33
Eclampsia	3	2.00

Table III shows that the mean±SD LVEF was 36.8±6.2%, with 40.0% of patients presenting LVEF <35%. The value of mean±SD in LV end-diastolic and end-systolic diameters were 61.5±5.8 mm and 49.7±6.0 mm, respectively. Fractional shortening averaged 19.1±4.2%, whereas moderate-severe mitral regurgitation and LV thrombus were observed in 12.0% and 4.0% of patients, respectively.

Table III
Echocardiographic and cardiac functional parameters at diagnosis.

Parameter	Value
Left ventricular ejection fraction (LVEF, %)	36.8 ± 6.2
LVEF < 35%	60 (40.00)
Left ventricular dimensions	
LV end-diastolic diameter (mm)	61.5 ± 5.8
LV end-systolic diameter (mm)	49.7 ± 6.0
Systolic function	
Fractional shortening (%)	19.1 ± 4.2
Moderate–severe mitral regurgitation	18 (12.00)
LV thrombus	6 (4.00)

Patients with preeclampsia indicated significantly lower LVEF (34.2±5.5% and 37.7±6.1%, p=0.002) and a higher proportion with preeclampsia in LVEF < 35% (56% and 32%, p=0.008) compared to those without preeclampsia. Although, LV dilation was more frequent in the preeclampsia group (64% and 50%), but was not statistically significant (p=0.09). NYHA class III–IV occurred more often in patients with preeclampsia (44% and 28%, p=0.03) (*Table IV*).

Table IV
Association between preeclampsia and cardiac dysfunction.

Cardiac Parameter	With Preeclampsia (n = 50)	Without Preeclampsia (n = 100)	p-value
LVEF (%)	34.2 ± 5.5	37.7 ± 6.1	0.002
LVEF < 35%	28 (56.00)	32 (32.00)	0.008
LV dilation	32 (64.00)	50 (50.00)	0.09
NYHA class III–IV	22 (44.00)	28 (28.00)	0.03

Acute heart failure was the most common outcome (68%), followed by ICU admission (36.7%), need for inotropic support (24%), mechanical ventilation (14.7%), cardiogenic shock (12%), thromboembolic events (6.7%) and maternal mortality (5.3%), respectively (*Table V*).

Table V
Maternal clinical outcomes in patients with PPCM.

Outcome	Frequency (n)	Percentage (%)
Acute heart failure	102	68.00
Cardiogenic shock	18	12.00
Need for inotropic support	36	24.00
Mechanical ventilation	22	14.67
ICU admission	55	36.67
Thromboembolic events	10	6.67
Maternal mortality	8	5.33

Table VI presents adverse maternal outcomes in preeclampsia (AOR 2.45, p=0.024), severe preeclampsia (AOR 3.12, p=0.008), LVEF < 35% (AOR 4.78, p<0.001), and NYHA class III–IV (AOR 3.65, p=0.004), respectively. Whereas, postpartum diagnosis was not significant (AOR 1.22, p=0.61).

Table VI
Multivariable logistic regression analysis for predictors of adverse maternal outcomes.

Predictor	Adjusted OR	95% CI	p-value
Preeclampsia	2.45	1.12–5.36	0.024
Severe preeclampsia	3.12	1.35–7.21	0.008
LVEF < 35%	4.78	2.10–10.88	<0.001
NYHA class III–IV	3.65	1.52–8.76	0.004
Postpartum diagnosis	1.22	0.57–2.63	0.61

At 6 months, 45.33% of patients achieved complete recovery (LVEF ≥ 50%), 38.67% had partial recovery (LVEF 35-49%), 14.7% showed persistent LV dysfunction (LVEF < 35%), and 1.3% were lost to follow-up (*Table VII*).

Table VII
Left ventricular function recovery at 6-month follow-up.

Recovery Category	Frequency (n)	Percentage (%)
Complete recovery (LVEF ≥ 50%)	68	45.33
Partial recovery (LVEF 35–49%)	58	38.67
Persistent LV dysfunction (LVEF < 35%)	22	14.67
Lost to follow-up	2	1.33

DISCUSSION

Peripartum cardiomyopathy (PPCM) is a rare, de novo form of cardiomyopathy that is potentially reversible and typically manifests in the late third trimester of pregnancy or within the early postpartum period. Several maternal characteristics have been implicated as potential risk factors for PPCM, including advanced maternal age, ethnicity or racial background, multiple gestations, multiparity, and hypertensive disorders of pregnancy, particularly preeclampsia [16].In the present study, the mean maternal age was 31.8 ± 5.4 years, which is consistent with the findings of Lee et al., who reported that women with peripartum cardiomyopathy tend to be older compared with control populations [17]. Additionally, multiparous women constituted the majority of our study population (60%), a pattern that aligns with observations reported by Abbas et al., suggesting a higher prevalence of PPCM among multiparous mothers [18]. In the present study, a clear predominance of postpartum diagnosis was observed, with 63.3% of peripartum cardiomyopathy (PPCM) cases identified after delivery. This finding is consistent with previous large observational and registry-based studies, which have shown that the majority of PPCM cases are diagnosed in the early postpartum period rather than during pregnancy [19]. This delayed presentation is thought to reflect the substantial hemodynamic stress of labor and delivery, together with abrupt postpartum cardiovascular and hormonal changes, which may unmask previously subclinical left ventricular dysfunction [20]. Hypertensive disorders of pregnancy were also highly prevalent in our cohort. Preeclampsia was documented in 33.3% of patients, with severe preeclampsia accounting for 20% and early-onset disease for 10%. These figures are notably higher than those observed in the general obstetric population and align with prior evidence demonstrating a strong association between PPCM and preeclampsia. A systematic

review of 22 studies reported a pooled prevalence of preeclampsia of approximately 22% among women with PPCM, compared with a global background prevalence of around 5% in pregnancy [5]. Moreover, contemporary nationwide registry data from Denmark have shown that hypertensive disorders, particularly severe preeclampsia, markedly increase the risk of PPCM, with severe disease conferring up to a 20-fold higher risk compared with normotensive pregnancies [21].Echocardiographic evaluation of patients with peripartum cardiomyopathy (PPCM) in our cohort revealed marked baseline cardiac dysfunction, with a mean left ventricular ejection fraction (LVEF) of 36.8 ± 6.2% and 40% of patients exhibiting severely reduced LV function (LVEF <35%). Left ventricular dilation and moderate-to-severe mitral regurgitation were also frequently observed. Notably, patients with preeclampsia demonstrated significantly lower LVEF (34.2 ± 5.5% vs 37.7 ± 6.1%, p=0.002) and a higher prevalence of severely reduced LV function (56% vs 32%, p=0.008), alongside more advanced NYHA class III–IV symptoms (44% vs 28%, p=0.03). These findings are consistent with previous reports showing substantial baseline systolic dysfunction in PPCM, with mean LVEF typically in the mid-30s% range and frequent LV dilation [1]. The observed association of preeclampsia with more severe left ventricular dysfunction in our study aligns with findings from Lindley *et al.* (2017), who reported that peripartum cardiomyopathy complicated by preeclampsia is characterized by distinct patterns of left ventricular remodeling and adverse clinical outcomes compared to PPCM without preeclampsia, including differences in LV dimensions and event-free survival, underscoring the complex interplay between hypertensive disorders of pregnancy and myocardial function in this population [22].Furthermore, comprehensive meta-analyses indicate that hypertensive disorders can influence both baseline ventricular function and the likelihood of LVEF recovery, though mortality differences may not be significant in the short term [23].In our study, acute heart failure occurred in 68% of women, ICU admission in 36.7%, and maternal mortality was 5.3%. These outcomes are consistent with literature showing that peripartum cardiomyopathy (PPCM) presents with high rates of symptomatic heart failure and is frequently associated with hypertensive disorders such as preeclampsia, which occurs at significantly higher prevalence in PPCM than in the general pregnant population [5]. Preeclampsia has been identified as a risk factor for more severe clinical courses in PPCM, including increased morbidity and worse event-free survival at 1 year, and is associated with distinct ventricular remodeling patterns [22]. Large database analyses indicate that preeclampsia is independently associated with a greater likelihood of ICU admission among PPCM patients, underscoring the severity of combined cardiac and hypertensive pathology [24]. Overall, our results align with broader evidence that PPCM co-existing with preeclampsia leads to substantial maternal cardiac morbidity and significant use of intensive care resources, and may contribute to elevated short-term mortality [25]. Multivariable logistic regression analysis revealed that preeclampsia, severe preeclampsia, markedly reduced left ventricular ejection fraction (LVEF <35%), and NYHA functional class III–IV independently predicted adverse maternal outcomes in peripartum cardiomyopathy (PPCM). Among these, severe left ventricular dysfunction conferred the greatest risk (OR 4.78, 95% CI 2.10–10.88, p<0.001) [1]. Epidemiological data indicate that the prevalence of preeclampsia in women with PPCM substantially exceeds that in the general obstetric population, with pooled estimates suggesting that approximately 25–36% of PPCM cases coexist with preeclampsia or other hypertensive disorders of pregnancy, highlighting overlapping pathophysiological mechanisms [2,3]. Consistent with this, a systematic analysis by Huang et al. demonstrated that all hypertensive disorders significantly elevate the risk of PPCM, with severe preeclampsia conferring the highest odds (OR ~13.33, 95% CI 5.95–29.83), followed by superimposed preeclampsia and chronic hypertension, suggesting a severity-dependent gradient of risk [4]. Nationwide cohort data further indicate that severe preeclampsia markedly increases PPCM incidence (RR 21.2; 95% CI 12.0–37.4), whereas moderate forms show more modest associations [5]. Clinically, preeclampsia-associated PPCM exhibits worse early outcomes but may demonstrate higher rates of LVEF recovery among survivors [6].Finally, we found at 6 months, complete recovery (LVEF ≥50%) was 45.30%, partial recovery 38.70%, and persistent dysfunction 14.70%, with minimal loss to follow up. This recovery profile aligns well with a study which represented approximately 40.0%–50.0% of PPCM patients achieve LVEF ≥50% by 6–12 months [14].

LIMITATIONS

This study did not assess long-term maternal and neonatal outcomes beyond six months, limiting evaluation of sustained cardiac recovery and late complications. Biomarkers and advanced imaging such as cardiac MRI were not incorporated, which may have provided deeper insight into myocardial remodeling. Additionally, residual confounding from unmeasured factors, including socio-economic status, nutritional status, and genetic predisposition, cannot be excluded, potentially affecting the observed associations with adverse maternal outcomes.

CONCLUSION & RECOMMENDATIONS

Preeclampsia significantly worsens cardiac function in women with PPCM, manifesting as lower LVEF, higher NYHA functional class, and increased incidence of adverse maternal outcomes. Severe left ventricular dysfunction (LVEF <35%) and advanced heart failure (NYHA III–IV) independently predict poor prognosis, underscoring the need for early identification and aggressive management. Despite substantial recovery in many patients, a notable proportion experience persistent LV dysfunction, highlighting the critical importance of close monitoring, timely intervention, and tailored postpartum follow-up to optimize maternal cardiovascular outcomes in this high-risk population.

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CONFLICT OF INTEREST

None declared

ETHICAL APPROVAL

The study was approved by the Institutional Ethics Committee.

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