ORIGINAL ARTICLE

Peripartum Cardiomyopathy: Experience at a Tertiary Center

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Abstract

Background: Peripartum cardiomyopathy (PPCM) is a rare, idiopathic cardiomyopathy associated with pregnancy that can lead to heart failure and maternal mortality. This study aims to evaluate the clinical features, diagnostic and treatment approaches, and outcomes of patients diagnosed with PPCM, while also increasing awareness among obstetricians, cardiologists and emergency medicine physicians to support earlier diagnosis and intervention.

Methods: Patients diagnosed with PPCM and managed within a multidisciplinary team including the perinatology clinic between October 2019 and October 2023 were retrospectively analyzed. Risk factors, clinical presentation, and outcomes were evaluated.

Results: Twelve patients with PPCM were identified. Eight were primigravida, and two had multiple pregnancies. Four patients were diagnosed antepartum and eight postpartum. Preeclampsia was present in five cases. Dyspnea was the most common symptom. Left ventricular dysfunction was mild (EF \geq 30%) in four patients and severe (EF <30%) in eight. Two patients required intubation, and one died on the second day following diagnosis. Bromocriptine was added to standard heart failure therapy in nine patients. At 6 months, five patients had recovered LVEF \geq 50%, and at 12 months, eight patients achieved this level. Three patients were lost to follow-up.

Conclusion: Due to its rarity and nonspecific symptoms, PPCM is often diagnosed late, potentially worsening prognosis. Since delayed diagnosis negatively impacts outcomes, early recognition and timely initiation of treatment in late pregnancy or postpartum are vital for improving maternal health.

Keywords: Peripartum cardiomyopathy, pregnancy, heart failure, pregnancy-associated cardiomyopathy, echocardiography.

INTRODUCTION

Peripartum cardiomyopathy (PPCM) is an idiopathic cardiomyopathy that develops during the last months of pregnancy or in the months following delivery. It presents with symptoms of heart failure secondary to left ventricular systolic dysfunction, in the absence of any other underlying cause (1). Unlike other cardiomyopathies, PPCM can progress to end-stage heart failure within days, or may resolve spontaneously with complete recovery (1). Although its exact incidence remains unclear, it appears to be increasing in frequency due to factors such as advanced maternal age, multiple pregnancies, and greater clinical awareness of the condition (2). Various studies have also demonstrated that racial and ethnic differences influence both the incidence and prognosis of PPCM (3, 4). Furthermore, several genetic variants associated with PPCM have been identified in studies suggesting familial predisposition (5, 6).

The etiology of PPCM remains poorly understood and is considered multifactorial in nature (7). Nutritional deficiencies, autoimmune mechanisms, and viral infections are among the proposed contributing factors (2). Based on animal studies and other data, the hormonal milieu and oxidative stress of the peripartum period are thought to be major triggers for PPCM (8). There is growing evidence supporting the role of 16-kDa antiangiogenic prolactin fragments, generated as a result of oxidative stress and elevated prolactin levels, in the pathogenesis of the disease (7). In addition to oxidative stress, inflammation plays a significant role in both the pathophysiology and prognosis of PPCM (9).

Although preeclampsia and other hypertensive disorders of pregnancy are not direct causes of PPCM, they are closely related conditions (2). Preeclampsia is significantly more prevalent in PPCM patient populations, and the two entities may share several common pathophysiological mechanisms (7, 10).

There is no specific diagnostic test for PPCM; it is essentially a diagnosis of exclusion (2,11). The most critical factor is maintaining a high index of suspicion. The similarity of symptoms to those of normal peripartum physiology may delay diagnosis, leading to increased complications and poorer outcomes (1). Differential diagnoses include other cardiac conditions such as myo-

carditis, valvular disease, congenital heart disease, myocardial infarction, and acute critical conditions such as pulmonary embolism, amniotic fluid embolism, and pulmonary edema. Patients typically present with exertional dyspnea, fatigue, orthopnea, edema, and chest pain. A left ventricular ejection fraction (LVEF) of less than 45% on echocardiography is a key diagnostic criterion (1).

The aim of this study is to enhance understanding of the risk factors and clinical characteristics of PPCM and to raise awareness among obstetricians, cardiologists and emergency medicine physicians. Increased awareness may lead to earlier diagnosis, more timely and effective treatment, prevention of fatal complications, and higher rates of complete recovery.

MATERIALS AND METHODS

This retrospective, descriptive study covers a four-year period between October 2019 and October 2023. It includes patients diagnosed with peripartum cardiomy-opathy (PPCM) who were managed and treated by a multidisciplinary team including the perinatology clinic at our tertiary care center. Prior to the study, approval was obtained from the institutional ethics committee (E2-23-5173), and appropriate ethical standards were followed.

Study Population

Patients who developed symptoms of PPCM during pregnancy or the postpartum period and were diagnosed with PPCM were included in the study. Although different diagnostic criteria have historically been used for PPCM, this study employed the definition provided by the Heart Failure Association of the European Society of Cardiology Working Group as the diagnostic standard (1). Inclusion criteria were as follows:

- No known pre-existing cardiac disease
- Onset of heart failure symptoms during late pregnancy or in the months following delivery
- No identifiable cause to explain heart failure
- LVEF < 45%

Although the ESC criteria are the most widely used at present, they are also consistent with the classic criteria proposed by Demakis et al., who first introduced the term PPCM and defined its diagnostic criteria (12). Subsequently, left ventricular systolic dysfunction was added to the diagnostic criteria of Demakis et al. (13,14).

The exclusion criteria were a history of heart disease, an identifiable alternative etiology, or an LVEF above 45%. During the study period, 24 patients with a preliminary diagnosis of PPCM were identified. Among them, three patients were found to have pulmonary thromboembolism, four had non-cardiogenic pulmonary edema, two had myocarditis, and three had valvular heart disease. After excluding these cases, a total of twelve patients with PPCM were included in the study.

The diagnosis and treatment process for each patient was managed through a multidisciplinary approach involving cardiology, intensive care, perinatology, and anesthesiology specialists. Sociodemographic characteristics, obstetric history, risk factors, clinical findings and laboratory results at the time of diagnosis, as well as treatment and follow-up data, were retrospectively collected from the patients' medical records. Echocardiographic parameters at the time of diagnosis, and at 6 and 12 months were reviewed.

Patients whose LVEF was ≥50% on follow-up echocardiography were considered to have recovered. Recovery within the first 6 months was defined as early recovery, whereas improvement occurring after 6 months was defined as delayed recovery (15). All patients received standard heart failure therapy as determined by cardiologists, in accordance with clinical guidelines. This included diuretics, angiotensin-converting enzyme inhibitors (ACE inhibitors) or angiotensin receptor blockers (ARBs), and beta-blockers (16).

Statistical Analysis

All statistical analyses were performed using SPSS v. 26 (IBM Inc., Chicago, IL, USA). Continuous variables were presented as medians and ranges, and categorical variables were expressed as counts and percentages.

RESULTS

During the planned study period, 12 patients with PPCM were identified, and their complete data were reviewed using the hospital's electronic medical records. The median age was 28.5 years (range: 22-36). The majority of patients (n=8) were primigravida. Two patients had multiple pregnancies. Four patients presented with symptoms and were diagnosed during the antepartum period, while eight were diagnosed during the postpartum period. Among postpartum cases, symptom onset occurred as early as the 1st day and as late as the 75th day. In antepartum cases, symptoms developed between the 33rd and 41st weeks of gestation. All of these patients underwent emergency cesarean delivery at the time of diagnosis due to hemodynamic instability. In total, ten out of 12 patients underwent delivery by cesarean section. Five patients had preeclampsia, and two had gestational hypertension. All patients presented with specific heart failure symptoms, with dyspnea being the most prominent and present in all cases at the time of diagnosis. The average length of intensive care unit (ICU) stay was 4 days (range: 1-8 days), and the total hospital stay averaged 9 days (range: 2-15 days). Two patients required intubation. Demographic and clinical characteristics are presented in Table 1.

As shown in Table 2, mild LV dysfunction (EF \geq 30%) was observed in four patients, while eight patients had severe LV dysfunction (EF <30%). Moderate to severe mitral regurgitation (MR) was present in six of twelve patients, and moderate to severe tricuspid regurgitation (TR) was observed in three of twelve patients. One patient experienced a thromboembolic event. All patients received standard heart failure therapy and anticoagulant treatment, including beta-blockers, ACE inhibitors, and diuretics. Bromocriptine was added to standard therapy in nine patients.

Follow-up echocardiographic evaluations were not available for three patients. Among the remaining nine patients, echocardiographic assessment revealed that five (55.6%) had a LVEF above 50% at 6 months, increasing to eight patients (88.9%) by 12 months. The echocardiographic findings at presentation, laboratory values, and outcomes are summarized in Table 2.

Table 1. Demographic and clinical characterist	tics of the patients	
Variable		n/N (%) or Median (Min–Max)
Age, years		28.5 (22–36)
	Gestational hypertension 2/12 (16.7%)	
Hypertensive disorders of pregnancy	Preeclampsia	5/12 (41.7%)
Primigravida		8/12 (66.7%)
Multigravida		4/12 (33.3%)
Multiple pregnancy		2/12 (16.7%)
	Dyspnea	9/12 (75%)
Symptoms	etoms Edema 4/1	4/12 (33.3%)
	Palpitations	7/12 (58.3%)
	Peripheral edema $4/12 (33.3\%)$ Pulmonary edema $9/12 (75\%)$	4/12 (33.3%)
Findings		9/12 (75%)
	Pleural effusion	3/12 (25%)
	Antepartum 4/12 (33.3%)	4/12 (33.3%)
Onset of symptoms	Postpartum	8/12 (66.7%)
Gestational age at diagnosis, weeks*		37 (33–41)
Gestational age at delivery, weeks*		37 (28–41)
Mode of delivery, caesarean section		10/12 (83.3%)
Postpartum symptom onset, days†		7 (1–75)
Need for intubation		2/12 (16.7%)
ICU stay, days		4 (1–8)
Total hospital stay, days		9 (2–15)
Standard heart failure treatment		12/12 (100%)
Additional bromocriptine therapy		9/12 (75%)
Abbreviations: ICU, intensive care unit; PPCM, peripartum ca* Calculated for antepartum patients. † Calculated for postpartum patients.	ardiomyopathy.	

Variable		n/N (%) or Median (Min–Max
	Mild (Grade 1)	10/12 (83.3%)
Mitral regurgitation	Moderate (Grade 2)	4/12 (33.3%)
	Severe (Grade 3)	1/12 (8.3%)
	Mild (Grade 1)	8/12 (66.7%)
Tricuspid regurgitation	Moderate (Grade 2)	2/12 (16.7%)
	Severe (Grade 3)	1/12 (8.3%)
Oxygen saturation (SpO2), %		88 (80–98)
D-dimer, μg/L		6.8 (3–35)
рН		7.36 (7.0–7.46)
Bicarbonate (HCO₃), mmol/L		20 (15–24)
PASP, mmHg		29.5 (8–55)
BNP, pg/mL	5443.5 (422–35000)	
Troponin, ng/L	92.5 (3.46–9254)	
LVEF, %		25 (15–45)
LVEDD, mm	50 (44–64)	
LVEF ≥ 30% at diagnosis		4/12 (33.3%)
LVEF < 30% at diagnosis		8/12 (66.7%)
Lost follow-up		3/12 (25%)
Outcome	Exitus	1/9 (11.1%)
	Early recovery (0-6 months)	5/9 (55.6%)
	Delayed recovery (6-12 months)	3/9 (33.3%)
	Total recovery	8/9 (88.9%)

DISCUSSION

In our study population, 58% (7/12) of the patients had preeclampsia or gestational hypertension. One-third were diagnosed during the antepartum period, while two-thirds were diagnosed postpartum. The mean LVEF at the time of diagnosis was 25% (range: 15–45%), and the mean LVEDD was 50 mm (range: 44–64 mm). Three patients were lost to follow-up. One patient died within the first week after diagnosis, early recovery was observed in five of nine patients, and delayed recovery in three of nine patients.

As in all forms of cardiomyopathy, plasma BNP levels are markedly elevated (11). Chest X-ray may reveal pulmonary venous congestion. Electrocardiography (ECG) may show nonspecific changes, although a normal ECG does not exclude PPCM (17). If additional information regarding cardiac function is required, cardiac magnetic resonance imaging may be used as a supportive tool (18). Some patients may experience cardiac arrest due to ventricular fibrillation (18). In certain cases, advanced mechanical circulatory support and urgent cardiac transplantation may be required (18). Management requires a multidisciplinary approach (19). At diagnosis, LVEF, left ventricule fractional shortening (LVFS), and LVEDD are the most reliable prognostic indicators (20,21). Left ventricular dilatation, presence of LV thrombus, and right ventricular systolic dysfunction are all markers of poor prognosis. The interval between symptom onset and diagnosis has also been shown to be a crucial prognostic factor (4).

Consistent with previous literature and the first national PPCM registry in Turkey—A RegisTry of pEripartuM cardIomyopathy in Turkish patientS (ARTEMIS)—two-thirds of our patients were diagnosed in the postpartum period, predominantly within the first month (22, 23). A notable finding from the ARTEMIS study was that several patients who presented with PPCM in the study had been previously diagnosed with pregnancy-associated cardiomyopathy during earlier postpartum periods. The authors interpreted this as previously undiagnosed PPCM in earlier pregnancies (22).

Studies have shown that approximately 45-50% of PPCM patients recover LV systolic function (LVEF \geq 50%), generally within six months following diagnosis (1, 24, 25). In a study from Pakistan involving 45 patients, early recovery was reported in 70% of cases (26). In contrast, Biteker et al. observed predominantly

delayed recovery (i.e., improvement after six months) in their cohort (15). A study from the United States reported 25% full recovery and 19% partial recovery, with delayed recovery seen in 83% of patients (27). In the multicenter study by the European Society of Cardiology Study Group, which included data from 49 countries, the 6-month mortality was 6%, and myocardial recovery was 46% (23). In our study, early recovery within 6 months was observed five out of nine patients (55.6%), and overall improvement in LVEF was observed in eight out of nine patients (88.9%) by the end of 12 months.

In the study by Blauwet et al., younger maternal age and smaller LVEDD at diagnosis were identified as the best predictors of recovery, while LVEF was not found to be predictive (28). However, other studies have reported that LVEF at diagnosis is one of the most important prognostic indicators (20). Due to the small sample size in our study population, we were unable to assess the correlation between recovery and potential prognostic factors, including baseline LVEF.

In our case series, 58% of patients had hypertensive disorders of pregnancy. This aligns with existing literature, which reports preeclampsia or gestational hypertension in 20–50% of PPCM patients (20,29). While these are well-known risk factors, they are thought to share common pathophysiological mechanisms with PPCM rather than act as direct causes (29,30). In PPCM cases accompanied by preeclampsia, symptoms tend to be more severe; however, baseline LVEF has been reported to be higher, baseline LVEDD smaller, and the likelihood of LV recovery greater (31).

All patients in our study received standard heart failure therapy administered by the cardiology team in accordance with current clinical guidelines. Suppression of prolactin release with bromocriptine is increasingly considered a key component of treatment. However, there is still no consensus on the routine use of bromocriptine, including which patients should receive it, the optimal dose, and duration of therapy. In recent years, bromocriptine has been shown to improve LVEF and result in better maternal outcomes (8, 32-34). Furthermore, a recent meta-analysis has shown that bromocriptine use is associated with a statistically significant improvement in survival among patients with peripartum cardiomyopathy (35). Current studies do not provide clear recommendations regarding which patients should receive bromocriptine, and the decision is generally left to the discretion of the treating clinician. When bromocriptine therapy is initiated, the recommended regimen is 2.5 mg twice daily for two weeks, followed by 2.5 mg once daily for an additional six weeks (34). In studies evaluating bromocriptine efficacy, only 10–20% of patients have received the drug (32-34). In contrast, nine out of 12 patients (75%) in our study received bromocriptine, a rate notably higher than that reported in the literature. The lack of a standardized protocol for bromocriptine administration, combined with emerging evidence supporting its potential role in left ventricular functional recovery, likely accounts for its high utilization rate in our cohort.

Although the importance of early diagnosis has been emphasized in many studies, delays in diagnosis may still occur due to the overlap with pregnancy-related symptoms and the rarity of the condition. It has been suggested that the cardiomyopathic process begins long before the diagnosis is established (36). In our study population, the timing of diagnosis was known; however, the interval between symptom onset and diagnosis could not be determined. The earlier the diagnosis is made, the better systolic function is preserved, and with appropriate treatment, the likelihood of full recovery is considerably higher (20,36).

The limitations of this study include its small sample size and retrospective design. Due to the retrospective nature of the study, there were missing data in echocardiographic findings, follow-up intervals, and control echocardiograms. As a tertiary referral center, most patients were referred to our hospital postpartum after receiving a PPCM diagnosis or requiring intensive care, limiting access to complete obstetric data. Due to the small number of patients and single-center nature, the generalizability of our findings is limited. The relationship between patients' recovery status and prognostic factors could not be evaluated. However, given the rarity of the condition, the study may still contribute to the growing body of literature despite these limitations.

Although rare, PPCM is a condition that warrants attention due to its high potential for recovery with early diagnosis. It is crucial not only for obstetricians but also for cardiologists and emergency medicine physicians to maintain a high level of suspicion for PPCM in pregnant or recently postpartum women presenting with symptoms of heart failure. Increasing awareness and ensuring timely intervention can improve maternal outcomes and save lives.

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Abbreviations list

ECG: Electrocardiography

EF: Ejection fraction

ICU: Intensive care unit

LV: Left ventricule

LVEDD: Left ventricular end-diastolic diameter

LVEF: Left ventricular ejection fraction

LVFS: Left ventricule fractional shortening

MR: Mitral regurgitation

PLVD: Persistent left ventricular dysfunction

PPCM: Peripartum cardiomyopathy

TR: Tricuspid regurgitation

Ethics approval and consent to participate

Prior to the study, approval was obtained from the Ankara Bilkent City Hospital Ethical Commitee (Date: 11.10.2023, No: E2-23-5173), and the study was conducted in accordance with the Helsinki Declaration of Human Rights.

Consent for publication

Written informed consent was obtained from all participants.

Availability of data and materials

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no conflict of interest.

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Authors' contributions

Idea/Concept: EB Design: EB, AS, AT Data Collection And/Or Processing: EB, BAA, AS Analysis And/Or Interpretation: EB, AT, CY Literature Review: EB, BAA, AS Writing The Article: EB, BAA Supervising: AT, CY, DŞ Critical Review: AT, CY, DŞ.

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