

PERIPARTUM CARDIOMYOPATHY IN A TERTIARY CARE HOSPITAL

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Abstract

Background: PPCM in pregnancy is a rare and potentially fatal form of heart failure presenting in the last month of pregnancy or within 5 months of delivery. It is associated with life-threatening complications necessitating management by a multi-disciplinary approach. The objective of this study is to study the clinical profile, maternal and fetal outcomes of PPCM. **Materials and Methods:** This prospective study includes all cases with PPCM confirmed in our center for a period of one year from January 2020 to December 2020. This study was conducted in the Department of OBG, Coimbatore Medical College and Hospital. **Result:** The incidence of PPCM was found to be 0.08% out of total obstetric admissions. 50% had an ejection fraction of <30%. 50% were in NYHA class III/IV. 100% required ICU admissions. Maternal mortality was 2.9% out of total maternal deaths. PPCM was identified in 25% in the antenatal period and 75% postpartum women. Poor fetal outcomes like stillbirth and prematurity were observed. **Conclusion:** The low incidence and rarity of PPCM and its presentation itself is a diagnostic dilemma. Obstetricians should take a high index of suspicion and early diagnosis and prompt management can prevent maternal morbidity and mortality.

INTRODUCTION

Peripartum cardiomyopathy (PPCM) was recently redefined by the European Society of Cardiology (ESC) as an idiopathic cardiomyopathy presenting with heart failure (HF) due to systolic dysfunction of the left ventricle (left ventricular ejection fraction [LVEF] <45%) towards the end of pregnancy or in five months following delivery, where no other cause of heart failure is found.^[1] Until specific etiologies are identified, PPCM remains a diagnosis of exclusion. The incidence is reported to be 1 in 3000 to 1 in 400 live births.^[2]

During pregnancy, blood volume and cardiac output increase, whereas after delivery, they decrease. These changes cause a transient, reversible hypertrophy of the left ventricle. The transient diastolic dysfunction during the third trimester and early postpartum period resolves shortly after birth. Nutritional disorders such as deficiencies in selenium and other micronutrients also might play a role in the pathogenesis of PPCM. Familial predisposition to PPCM has been reported.

The onset of PPCM can easily be masked because the manifestations can mimic those of mild heart

failure. Women with PPCM commonly show dyspnea, dizziness, chest pain, cough, neck vein distension, fatigue, and peripheral edema.^[1] Arrhythmias, embolic events from thrombosis of the dilated dysfunctional left ventricle and acute myocardial infarction due to decreased perfusion to coronary arteries are other less common clinical features.^[1,3-5] Blood pressure is often normal or decreased, and tachycardia is common.^[5]

An ECG should be performed in all patients with suspected PPCM as it has a high negative predictive value. There may be an absence of a specific ECG pattern for PPCM initially but the ECG is rarely normal: repolarization abnormalities, LV hypertrophy, dysrhythmias, Q-waves in the anteroseptal precordial leads, and prolonged PR and QRS intervals are common.^[5]

Several other tests should be performed: complete blood cell count and serum levels of troponin, urea, creatinine, and electrolytes,^[5] liver and thyroid function tests.

The definitive diagnosis of PPCM depends on echocardiographic identification of new-onset heart failure during a limited period around parturition.

Management of PPCM in the absence of evidence-based data is like standard treatment for other forms of heart failure.^[1,5] Timely diagnosis and delivery are crucial. The goals of heart failure treatment are to improve hemodynamic status, minimize signs and symptoms and optimize long-term outcomes. Treatment focuses on reducing preload and after load and increasing cardiac output.^[1,5]

ACE inhibitors, beta-blockers and MRAs should be initiated and continued at least till complete recovery of LV size and systolic function. Discontinuation of heart failure medications can be considered only in the case of complete recovery of ventricular function and exercise response.

Initial treatment with ivabradine aimed at controlling tachycardia even before or in parallel with beta-blockers appears to be safe and effective.^[1]

MATERIALS AND METHODS

This study was conducted in the Department of Obstetrics and Gynaecology, Coimbatore Medical College Hospital, Coimbatore from January 2020 to December 2020, accounting for a total period of 1 year.

This study was designed as a prospective study. Pregnant or postpartum women are included in the study. Development of PPCM is identified based on the definition already cited above. Women with already preexisting heart diseases, either acquired or congenital who developed signs and symptoms of cardiac failure are excluded.

Findings such as tachycardia, dyspnoea, chest-pain, pedal edema, raised JVP, PND, hemoptysis, nocturnal cough, anasarca, wet crepitations on lung fields, systolic murmur, PIH, new onset MI, hepatosplenomegaly, low saturation etc. were used as early determinants of the disease. Diagnosis was further supplemented by ECG findings. Echocardiography with ejection fraction <45% confirmed the diagnosis of PPCM. Apart from the above examination parameters, obstetrical

monitoring for maternal and fetal wellbeing like NST, ultrasound with Doppler were also carried out. The data collected were compiled in terms of age, parity etc., and the results were tabulated and analysed.

RESULTS

The incidence of PPCM was found to be 0.08% out of total obstetric admissions. During our study period total maternal deaths were 34 out of which 1 was PPCM which accounts to PPCM. In our study it is observed that peripartum cardiomyopathy is more prevalent among the age group of 25 to 30 years showing 41.66% [Table 1]. As far as parity is concerned, 50% of the cases are primi gravid. [Table 1]. 83.3 % of prevalence is among the subjects from low socioeconomic status and 16.7% from middle socio economic class. No cases were reported from high socio-economic class [Table 1].

Regarding presentation of PPCM, 100% had ECG changes, 33.3 % had pedal edema, 66% had cardiomegaly, 41.66 % had derangements of liver and renal function. 50 % had an ejection fraction between 30 to 45% and rest 50% had ejection fraction lesser <30% [Table 2].

Clinical presentation of PPCM was also variable. There was an overlap between various signs and symptoms. 83% of the subjects with PPCM either had tachycardia or dyspnoea, 66% had pedal edema and 50 % had systolic murmur [Table 3].

Out of the subjects with PPCM, 50% fell into NYHA class III, 33.3% fell into class IV, 16.7% fell into class II according to NYHA classification. 100 % were admitted in ICU, 41% of patients had pleural effusion, 41% had emboli, 33% had pericardial effusion and only 16% had fever [Table 4].

Obstetric outcomes of PPCM subjects were as follows 33% underwent LSCS and 41.66% were labour natural. As far as neonates are concerned, 66% had low birth weight (<2.5kg), 8.3% neonatal death and 83% were admitted to NICU [Table 5].

Table 1: Patient Characteristics

Age Distribution			Parity Distribution			Socioeconomic Status		
Age Group	Number	%	Parity	Number	%	SES	Number	%
18-20 YRS	3	25	0	6	50	Low	10	83.3
21-25 YRS	1	8.3	1	2	16.7	Middle	2	16.7
25 -30 YRS	5	41.7	2	1	8.3	High	0	0
>30 YRS	3	25	3 and above	3	25			

Table 2: Clinical and Laboratory Parameters and Echocardiography Findings

Parameter	Number	Percentage
Anemia	10	83.3
Cardiomegaly	8	66.6
Pulmonary Edema	4	33.3
ECG Changes	12	100
Deranged LFT	5	41.66
Deranged RFT	3	25
Electrolyte Abnormality	4	33.3
Echocardiography:		
Ejection Fraction	Number	Percentage
30-45%	6	50

<30%	6	50
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Table 3: Symptomatology

Symptoms	Frequency	Percentage
Tachycardia	10	83.3
Dyspnea / Pnd	10	83.3
Chest Pain	2	16.7
Elevated Jvp	2	16.7
Pedal Edema	8	66.6
Anaemia	4	33.3
Systolic Murmur	6	50
PIH/ECLAMPSIA	5	41.66

Table 4: Complications

Complication	Frequency	Percentage
Fever	2	16.7
ICU Admission	12	100
Ventilatory Support	5	41.66
Pericardial Effusion	4	33.3
Pleural Effusion	5	41.66
Ascites	5	41.66
Emboli	5	41.66

Table 5:

Obstetric Outcome:		
Outcome	Frequency	Percentage
Abortion	2	16.7
Preterm Delivery	1	8.3
LSCS	4	33.3
Vaginal Delivery	5	41.66
Fetal Outcome:		
Outcome	Frequency	Percentage
Prematurity	5	41.66
IUGR	6	50
Low Birth Weight	8	66.6
NICU Admission	10	83.3
Perinatal Death	1	8.3

DISCUSSION

Demakisetal⁶ defined the condition Peripartum cardiomyopathy in 1971, when he described the spectrum of the disease in 27 pregnant women with cardiomegaly and congestive heart failure. He also gave the classical diagnostic criteria for PPCM, which was then modified by the National Heart Lung and Blood Institute and Office of Rare Disease.^[8] Recent studies implicate the role of a prolactin sub fragment 16KD a which is cleaved from prolactin due to unbalanced oxidative stress. The 16KD a can destroy the endothelium and damage the micro circulation in the myocardium, reducing the cardiac function and causing ventricular dilatation.

Abnormal immune response to pregnancy is another postulated theory, in which auto antibodies are formed after delivery against uterine degradation products such as actin and myosin. These antibodies then cross react with proteins in the myocardium and cause myotoxicity. Such antibodies have been reported in many studies and are associated with increased levels of tumor necrosis factor alpha, interleukin 6c.,

Advanced maternal age >30 has been associated with PPCM. However in our study there were no association and only 25% were more than 30 years

had PPCM, which is similar to study done by Bhattacharjee. R et al. In our study, it was 50% in primigravida which does not correlate with the study done by Bhattacharjee. Retal where the incidence is more in multi gravida.^[10] The association of PPCM and GHTN in our study was 4% which correlates with the study done by Fischer et al.^[7] Most common symptoms were dyspnea and tachycardia (83%) in our study which is comparable to the study done by Elakayam & Moiolietal.^[9] A high Cesarean rate was reported by Gvordil et al, cesarean rate in our study was 33.3%.

CONCLUSION

Management of PPCM is similar to that of heart failure. A multidisciplinary approach should be undertaken, fluid and salt restriction, digoxin, vasodilators, diuretics and anticoagulants constitute the main line of treatment. In our institution, we are following BOARD therapy for management of PPCM. B-Bromocriptine, O-Oral anti failure medications, A-Anticoagulants, R-vaso Relaxants, D-Diuretics. The role of bromocriptine has been studied by many. In our study, 5 patients were given bromocriptine, after which the patients symptomatically improved. Further research in this field is imperative to enhance understanding and

enable directed treatment strategies to be incorporated into routine practice.

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