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Original Research Article

## Peri-partum cardiomyopathy-not a rare entity, clinical profile in a rural tertiary care center in Gujarat, India

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### ABSTRACT

**Background:** Peri-partum cardiomyopathy (PPCM) in pregnancy has a potential to adversely affect both mother and fetus. Severe cases can be associated with life threatening complications unless managed promptly by multidisciplinary team. The objective was to study maternal and fetal outcome of PPCM patients.

**Methods:** An observational cohort over 4 years (2012 to 2015) was conducted in a Rural tertiary care center. Consecutive antenatal and postpartum women with PPCM were studied for medical and obstetric complications, deliveries, fetal outcome and maternal mortality.

**Results:** The incidence of PPCM was 0.3% and Most patients presented with typical signs and symptoms of heart failure. 55.5% were in NYHA 3 and 4 and 50% had ejection fraction below 30%. 83.3% required ICU admissions of varying length and Maternal mortality was in 1 patient (5.56%). PPCM occurred in 55.5% of ante partum against 44.4% of postpartum patients. Obstetric complications like PPH occurred in 11.1% while poor fetal outcome like preterm deliveries occurred in 50% while still births in 16.67%.

**Conclusions:** The low incidence and rarity of PPCM presents itself with diagnostic dilemma. Obstetrician should have high index of suspicion in patients with heart failure and high risk factors. Early diagnosis and prompt treatment can only improve the outcome.

**Keywords:** Cardiomyopathy distress, Peri-partum cardiomyopathy

### INTRODUCTION

Peri partum Cardiomyopathy (PPCM) is a rare potentially life-threatening cardiac disease occurring in the last month and within 5 months of delivery in previously healthy women. The diagnostic criteria include an echocardiography finding of ejection fraction of less than 45%, fractional shortening of less than 30% and diastolic dimension of greater than 2.7cm/m<sup>2</sup> body surface area.<sup>1</sup> The incidence is reported to be 1 in 3000 to 1 in 400 live birth.<sup>2</sup> The etiology is unknown, though some risk factors are cited such as advanced maternal age, multiparity, multiple gestations, pre-eclampsia, chronic hypertension etc.<sup>3-5</sup> The signs and symptoms are subtle and pathogenesis is poorly understood. PPCM may

remain unrecognized and the diagnosis is by exclusion of other heart diseases, sepsis, pulmonary embolism, valvular disease, idiopathic dilated cardiomyopathy and MI. This rare and fatal disease has a mortality rate of 30-60% and recurrence of 50-80% with uncommon complications such as pulmonary congestion and thromboembolic events.<sup>1</sup> A multidisciplinary approach is required to manage the heart failure during pregnancy and provide optimum outcome. This study was done with the objectives to find out incidence of PPCM among cardiovascular disease in pregnancy and to study the spectrum of PPCM along with maternal and fetal outcome in pregnancy with PPCM.

## METHODS

It is a retrospective observational database analysis conducted in the department of obstetrics and gynecology in SKH over a period of 4 years from Jan 2012 to Dec 2015. Data was collected from medical record department after ethics committee approval. Consecutive data of all pregnant women and postpartum women with PPCM were systematically studied. As it is retrospective study waiver of informed consent was sought from ethics committee. Diagnosis was based on suspicious clinical findings of heart failure such as tachycardia, dyspnoea, chest pain, pedal edema, raised JVP, PND, haemoptysis, nocturnal cough, anasarca, wet crepitations on lung fields, systolic murmur, PIH, new onset MI, hepatosplenomegaly, low spo2 etc. Diagnosis was supplemented with ECG and chest x-ray findings. Echocardiography findings of ejection fraction <45% confirmed the diagnosis of PPCM. All patients were hospitalized during the acute phase. Detailed history was taken of each patient and thorough clinical and obstetrical examination was done. NST, USG and Doppler was done where required to assess the fetal well-being. Management was done in collaboration with intensivist and cardiologist. They were given standard care for heart failure and were managed in ICU. Patients were monitored for signs and complication of thromboembolism, DIC, cardiac arrhythmia, systemic and pulmonary embolism, renal and liver failure. Fluid and salt restriction, digoxin, diuretic vasodilators and anticoagulants were instituted. They received parenteral nutrition, parenteral antibiotics and ventilator support as needed.

Obstetric complications were also looked for such as antepartum, intrapartum or postpartum haemorrhage, PROM, preterm delivery, IUFD. Patients with past history of PPCM were counseled regarding risk factors associated with continuing pregnancy.

Termination of pregnancy was done in patients with IUFD, severe IUGR, non-reactive NST, PROM and severe heart failure. LSCS was done for obstetric indications and in critically ill mothers. All women with manifestations of bleeding and deranged coagulation profile were infused with fresh frozen plasma and packed red blood cells. Per rectal misoprostol (400 micro gram) was kept and/or concentrated oxytocin drip was started to prevent PPH after delivery. Patients who improved clinically or showed signs of improvement on echocardiography were discharged and followed up in OPD variables.

## RESULTS

The incidence of cardiovascular diseases in this study group during a period of 4 years was 1.1%. PPCM constituted to 0.3% of total obstetric admissions during the study. Table 1 shows patient characteristics. Most of the people in our study were in the age group of 21-23 years. Out of 18 patients 13 were second gravida. 1 patient was grand multipara. 4 patients had multiple pregnancy. Most of the patients belonged to low socioeconomic status. 10 out of 18 patients presented in their antepartum period. 8 patients were postpartum.

**Table 1: Patient characteristics.**

Age	N	%	Parity	N	%	No. of fetus	N	%	Socio economic status	N	%
18-20	3	16.67	0	1	5.5	Single	14	77.7	low	10	55.5
21-23	6	33.33	1	13	72.2	Twins	4	22.2	Middle	6	33.3
24-26	3	16.67	2	3	16.6	Triplets	0	0	High	2	11.1
27-29	3	16.67	3	0	0						
30-34	2	11.11	>3	1	5.5						
>35	1	5.56									
<b>Total</b>	<b>18</b>			<b>18</b>			<b>18</b>			<b>18</b>	

Table 2 shows the mean lab values at admission and at discharge. A decreasing trend is seen in most of the parameters.

Table 3 shows the presenting symptomatology of patients with PPCM, Tachycardia, PIH, Dyspnea, Wet lungs and Pedal edema was present in most of the patients. Very few patients in our study had orthopnea, MI or hepatosplenomegaly

55.5% patients were in NYHA 3 and 4, 44.4% patients were in NYHA grade 2. 50% patients had an ejection

fraction between {30-45} % in their echocardiographic finding, and 38.88 had ejection fraction between 20-30%.

Table 6 shows wide spectrum of maternal complications ranging from febrile illness to mortality. 56.6% patients had fever, 38.39% patients had eclampsia and 22.2% patients had developed coagulation defects. 5.56% patients had bleeding from different sites. 83.3% patients required ICU admission. The maximum ICU stay was 6 days for 1 patient while the average ICU stay was 2-4 days. 10 patients needed to be transfused blood and blood products. 2 patients developed cerebral emboli while 1 patient developed arterial emboli. 66.67% patients had

compromised heart function. 50% patients required ventilator support ranging from few hours to few days. Maternal mortality occurred in 1 patient who was a second gravida at 18 weeks' gestation with absent FHS. She had PPCM in previous pregnancy also. She presented with bilateral pulmonary edema and ejection fraction of 20% and global hypokinesia. Cause of mortality was Congestive cardiac failure.

**Table 2: Lab data and investigations.**

Investigations	On admission	On discharge
Mean Hb (gm/l)	10.1	10.65
Mean count (cells*100/l)	17.1	19.6
Mean platelets (cells*1000/l)	233	246
Mean S. Proteins	4.76	3.6
Mean S. Urea	42.5	45
Mean S. Creatinine	0.88	0.9
Mean AST	25	30
Mean ALT	27.33	26
Mean S. Billirubin	1.1 mg%	1 mg%
Mean INR	1.5	1.09
Mean Na+	137.2	135
Mean K+	4.27	3.5
Cardiomegaly	8	4
Pulmonary Edema	10	0
ECG changes T wave inversion, ST depression, multiple atrial premature beats, VPB	6	0

**Table 3: Patient symptomatology.**

Symptom/ Finding	No.	%
Tachycardia	13	72.22
Dyspnoea	12	66.67
Orthopnoea	0	0
Chest pain	4	22.22
Pedal edema	9	50
Raised JVP	0	0
PND	0	0
Nocturnal cough	2	11.11
Anasarca	4	22.22
Wet crepitations on lung	10	55.56
Systolic murmur	3	16.67
PIH	13	72.22
Eclampsia	7	38.89
Chronic Hypertension	1	5.56
New onset MI	1	5.56
Hepatosplenomegaly	2	11.11

**Table 4: NYHA grade.**

NYHA Grade	No.	%
1	0	0
2	8	44.44
3	6	33.33
4	4	22.2

**Table 5: Echocardiography.**

Ejection Fraction	30-45 %	20-30 %	<20 %
patients	9	7	2
	50 %	38.8 %	11.1 %

**Table 6: Medical complications.**

	No.	%
Fever	10	55.56
Ascites	3	16.67
Pleural effusion	2	11.11
Pericardial effusion	1	5.56
Eclampsia	7	38.89
Bleeding from different sites	1	5.56
Deranged RFT	1	5.56
Deranged LFT	1	5.56
Deranged coagulation profile	4	22.22
Cerebral emboli	2	11.11
Arterial emboli	1	5.56
Need for blood products	10	55.5
ICU admission	15	83.3
Compromised heart function	12	66.67
Patients requiring ventilatory support	9	50
Mortality	1	5.56

Table 7 narrates the Obstetric outcome. Among the 18 patients, 10 were antepartum, out of which 1 had a second trimester abortion. 8 patients delivered outside. Total preterm deliveries (both vaginal and LSCS) were 6(33.33%), 16.67% had LSCS for obstetric or medical indications. 3 women had IUFD. 2 patients developed PPH which was managed conservatively. 2 of the women had conceived after developing PPCM in last pregnancy also. 2 women were discharged antenatal and they delivered live babies at or near term. All the patients were followed up in postnatal OPD after discharge and were healthy. However, 3 were lost to follow up.

The fetal outcome is presented in table 8. Both in-patient deliveries and outside deliveries were considered. 50% were preterm babies and 16.6% were still births, 77.8% babies were born live out of which 66.6% had IUGR and 44.4% had birth weight <2.5 kg. 66.7% babies had APGAR <7 at 5 minutes. 66.6% babies required NICU admission. There was no neonatal death.

**Table 7: Obstetrics outcome.**

Outcome	No.	%
Abortion	1	5.56
PROM	0	0
Preterm deliveries (vaginal and CS)	6	33.33
LSCS	3	16.67
Instrumental delivery	2	11.1
Foetal distress	1	5.56
IUFD	3	16.6
PPH	2	11.11
Induction of labour	1	5.56
Continued pregnancy and delivered at term	2	11.11
Conceived after PPCM in last pregnancy	2	11.11
Full term deliveries	3	16.67
Lost to follow up	3	16.66

**Table 8: Fetal outcome.**

Outcome	No.	%
Prematurity	9	50
IUGR	12	66.6
Birth weight <2.5 kg	8	44.4
APGAR <7 at 5 min	12	66.7
NICU admission	13	72.22
Still birth	3	16.67
Live birth	14	77.8
NND	0	0
Total perinatal deaths	3	16.67

## DISCUSSION

Descriptions of peripartum cardiomyopathy can be dated back to the 19<sup>th</sup> century. The association of the heart failure and puerperium was first established by Ritchie in 1849.<sup>6</sup> However it was only after 1937, that PPCM was recognized as a distinctive form of cardiomyopathy. In the initial years, the condition was defined by several names such as postpartum failure, postpartum myocarditis, Meadows syndrome; Zaria syndrome etc.<sup>7</sup> Demakis et al 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. These criteria include: development of cardiac failure in last month of pregnancy or within 5 months after delivery, absence of demonstrable pre-existing cardiac disease, absence of an identifiable cause of heart failure, left ventricular systolic dysfunction demonstrated by classic echocardiographic criteria: ejection fraction <45% or fractional shortening <30% or both, and end diastolic dimension >2.7 cm/m<sup>2</sup> body surface area.<sup>3-5</sup>

The incidence of PPCM is reported to be 1 in 1500 to 1:4000 live births. Mielniczuk et al reported an incidence of 1:3189 live births.<sup>8</sup> The prevalence is 1 case for 1,300 -15000 live births in USA; 1 case per 6000 live births in Japan, 1 case per 1000 live births in South Africa & 1 case per 350 – 400 live births in Haiti.<sup>2</sup> The incidence is very high in Nigeria (1 in 100), while the incidence of PPCM in our study was 4 per 1000 deliveries.<sup>9</sup> The incidence in another Indian study was 1 in 1374 live births.<sup>10</sup>

The pathogenesis of PPCM is controversial and many probable causes have been proposed. Advanced maternal age >30 has been associated with PPCM.<sup>3-5</sup> However, such an association was not found in our study. Multiparity has also been implicated. 71% of cases were reported to occur >3 pregnancies as compared with 8% in primigravidas (Denatus, as cited in Ntusi). In our study, the incidence was found to be more in second and third gravidas. More incidence was found in multifetal pregnancies in some studies, which was not so in this study. The association of PPCM and gestational hypertension was found in 43%.<sup>3-5</sup> In our study, the association was 38.8%. In many studies, the incidence of PPCM was higher in post-partum patients than antepartum patients. In a study from China, 17% of cases were diagnosed antepartum and 83% were postpartum admissions. In our study, 55.5% were antepartum and 44.4% were postpartum.

The incidence is particularly high in some African countries, probably due to certain cultural practices followed during the puerperium. These include consuming lake salt or rock salt (Kanva) or heating of the body on a clay bed with fire beneath to keep warm.<sup>15</sup>

Recent studies implicate the role of a prolactin sub fragment 16KDa which is cleaved from prolactin due to unbalanced oxidative stress. The 16KDa can destroy the endothelium and damage the micro circulation in the myocardium, reducing the cardiac function and causing ventricular dilatation.<sup>12</sup>

Myocarditis has also been proposed as a cause of PPCM because of presence of fibrosis and necrosis in dilated hearts. Gouley et al, Melvin et al, Conoc, Ntusi and many others tried to show the association.

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 6 etc.<sup>13,14</sup>

Another phenomenon is fetal chimerism in which fetal cells are present in maternal circulation during and after delivery, which after delivery are recognized by maternal

system as foreign antigens and may trigger an immune response.<sup>13-15</sup> Abnormal response to haemodynamic stress of pregnancy is a hypothesis which suggests that some alterations in the haemodynamic setup results in cardiac hypertrophy.

According to another theory, increased myocyte apoptosis may be a cause, wherein, there is an imbalance between cellular elimination and cellular regeneration due to over expression of certain genes. Recently the proapoptotic gene Nix or Bnip3 have been demonstrated to play an important role (Diwan, as cited in Hilfiker-Kleiner)

Patients with PPCM present with classical symptoms of systolic heart failure. The most common symptoms are dyspnea and fatigue (90%), tachycardia (62%), peripheral edema (60%). (Elkayam et al, Muioli et al, reported increased incidence of persistent nocturnal dry cough, orthopnea and paroxysmal nocturnal dyspnea.<sup>16</sup> Tachycardia or supra ventricular tachyarrhythmias were reported by Bertrand.<sup>17</sup>

In our study, tachycardia (72.2%), Dyspnea (66.6%), chest pain (22.2%) and pedal edema (50%) were the main presenting symptoms.

Unusual presentations like acute cyanosis (cole et al), multiple thromboembolic events (Carlson et al), liver failure (Fussel et al), systemic and pulmonary embolic episodes (Bennani; Box; Jha) have also been described.<sup>18,19</sup>

The mainstay of investigations includes chest x-ray, ECG and echocardiography, besides an array of blood tests to detect hematological, renal and liver function abnormalities. Cardiac markers, such as Troponin T is suggested to have prognostic implication. B-type natriuretic peptide (BNP) and N terminal pro – BNP (NT-pro BNP) are recommended by the Heart Failure Association of the ESC Working Group on PPCM.<sup>20</sup>

Antenatal testing such as non-stress test and serial ultrasonography should be done to monitor fetal well-being.

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. Seriously ill patients are given supportive care in the ICU. The role of bromocriptine has also been studied by many. It can inhibit prolactin secretion and reduce activity of 16kDa proapoptotic form of prolactin.<sup>21,22</sup>

Mechanical Ventricular support and cardiac transplantation or intraaortic balloon pump counter pulsation may be needed in patients not responding to medical treatment.<sup>7</sup>

Patients who are hemodynamically stable can be allowed to go to term. However, for patients near term labour should be induced. Vaginal deliveries have many advantages such as minimal blood loss, greater hemodynamic stability, less surgical stress, and pulmonary complications. Effective pain management should be instituted. Second stage should be cut short with forceps or vacuum. Caesarean section should be reserved for obstetric indications or critically ill patients.<sup>1</sup> A high caesarean section rate was reported by H.V Gvordil et al. The c/s rate in our study was 16.67%. Regional anaesthesia has benefits over general anaesthesia. Careful haemodynamic monitoring and fluid balance is obligatory.<sup>1</sup>

Obstetric and fetal outcome have not been extensively studied. Low birth weight, premature delivery, IUGR and fetal death have been reported by some authors.<sup>23</sup> 50% of the babies born were preterm in our study. The incidence of still births was 16.67%

PPCM may recur in subsequent pregnancies. Fett et al described a recurrence rate of 53%. A study from Haiti reported 53.3% of recurrence in next pregnancies.<sup>24</sup> In a study by Silva et al from South Africa, out of 6 women with recurrent PPCM, 2 died. Elkayan et al in the United States reported 44 women with PPCM in subsequent pregnancies. He recorded a high mortality rate (19%) in patients who had ejection fraction less than 50% in the pre-pregnant state. In our study the recurrence rate was 11.1% out of which 1 patient had mortality.

According to Silva et al, pregnancy should be avoided if LV ejection fraction <25% at diagnosis or with incompletely recovered left heart function.<sup>20</sup> Contraceptive counseling is imperative. The combined oral contraceptives are contraindicated. Intrauterine systems or sterilization can be considered.<sup>20</sup>

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