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PERIPARTUM CARDIOMYOPATHY: RISK FACTORS, HOSPITAL COURSE AND PROGNOSIS; EXPERIENCES AT LADY READING HOSPITAL PESHAWAR

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All the authors contributed significantly to the research that resulted in the submitted manuscript.

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ABSTRACT

Objective: To study the so-called risk factors associated with peripartum cardiomyopathy (PPCM), its hospital course, short and long term mortality and outcome of subsequent pregnancies.

Methodology: A total of 61 patients diagnosed with PPCM were enrolled in the study. Data regarding risk factors, presenting complaints, complications, pregnancy outcomes, therapeutics used and outcome at 3, 6 and 12 months were recorded.

Results: The incidence was estimated to be 1 per 933 deliveries. Mean age±SD was 30.94±6.63 years. Majority of patients 33(54.1%) were obese. The mean parity was 3.66 ±1.41. Other risk factors were chronic hypertension 19 (31.1%), pre-eclampsia 12 (19.7%) and multiple pregnancies 5(8.2%).

Forty-three patients 43(70.5%) presented in post partum period while 18 (29.5%) in antepartum period. All patients presented with dyspnea. Main ECG findings were sinus tachycardia 39 (63.9%), LV hypertrophy 42 (68.9%) and T wave inversion 28(45.9%). Ejection fraction was universally reduced on echocardiography. There were 50(82%) live births and 11(18%) perinatal deaths. Main complications were pulmonary edema 7(11.5%), cardiogenic shock 8(13.1%) and thromboembolism 13(21.3%). At hospital discharge, 9 (14.8%) patients were dead and 52(85.2%) were discharged with stable heart failure. At 12th month follow up, a total of 20(32.8%) were dead while 32(52.5%) had recovered fully and 9(14.75%) were still suffering from heart failure. During follow up, only 8(19.5%) pregnancies were detected. Five(62.5%) patients had uneventful course while 3(37.5%) developed heart failure again.

Conclusion: Peripartum cardiomyopathy is associated with multiple risk factors and carries high morbidity and mortality.

Key Words: Peripartum cardiomyopathy, Risk factors, Hospital course, Prognosis.

INTRODUCTION

Peripartum cardiomyopathy (PPCM) is a rare life-threatening cardiomyopathy of unknown cause that occurs in the peripartum period in previously healthy women.[1] It has been variably defined. The criteria for its diagnosis were first established by Demakis et al, in 1971.[2,3] Later on the National Heart, Lung, and Blood Institute and the Office of Rare Diseases workshop adopted the modified definition in 2000.[1]

In 2010, the European Society of Cardiology Working Group on Peripartum Cardiomyopathy proposed a modification to the existing definition of PPCM.[4] According to the first two definitions it must develop during the last month of pregnancy or within 5 months of delivery in absence of preexisting heart diseases with evidence of left ventricular dysfunction(i.e., left ventricular ejection fraction<45% on echocardiography) and no other identifiable cause responsible for heart failure. Its incidence is quoted as 1: 3500 to 1: 1400 for the USA and Europe, 1: 1000 for South Africa and 1 in 299 for Haiti.[5] In Pakistan its incidence is estimated to be 1 in 837 deliveries in one study.[6]

A relationship between pregnancy and dilated cardio-myopathy was first noted in 1870 when Virchow and Porak first reported autopsy evidenceof myocardial degeneration in patients who died in the puerperium.[6] In 1937 Gouley et al, demonstrated enlarged hearts with wide spread severe focal areas of necrosis and fibrosis on autopsy of pregnant women. On the basis of these atypical findings, author proposed that this heart failure was related to pregnancy and the puerperium either directly or indirectly.[7]

Risk factors for PPCM classically identified in the literature include multiparity, advanced maternal age, multifetal pregnancy, preeclampsia and gestational hypertension, obesity, malnutrition and African American race.[3,8]

A number of possible causes have been proposed for PPCM including myocarditis, abnormal immune response to pregnancy, maladaptive response to the hemodynamic stresses of pregnancy, stress-activated cytokines, cardio-tropic viruses, micronutrient or trace mineral deficiencies, genetics and prolonged tocolysis.[1,10-14] Recent evidence suggests a role for a 16 kDa prolactin derivative produced by proteolytic cleavage of prolactin secondary to unbalanced oxidative stress present during late pregnancy and early puerperium.[15]

Patients with peripartum cardiomyopathy present with typical symptoms and signs of heart failure. The majority of cases occur after delivery and in the immediate post partum period. The diagnosis requires echocardiographic information and rests on the presence of left ventricular systolic impairment. Medical treatment of peripartum cardiomyopathy is similar to treatment of congestive heart failure. Immunosuppressive therapy can be considered for women with myocarditis. Maternal mortality from peripartum cardiomyopathy in United States has been reported to be 25-50%.[16,17] Normalization of heart size and resolution of congestive heart failure within 6 months after delivery is a good prognostic sign with mortality rare among these patients.[8]

Patients with peripartum cardiomyopathy require counseling concerning the risk of a subsequent pregnancy. Patients without resolution of their cardiomyopathy are at significant risk for death or exacerbation of the disease and should be advised to avoid pregnancy.[8] However a few studies suggested that 20% of patients experienced transient exacerbation during subsequent pregnancy even after complete resolution of cardiac dysfunction.[18]

Peripartum cardiomyopathy is a rare lethal disease about which little is known.[1] It is especially true at our provincial and national level where data about it are limited. The aim of this paper was to assess the incidence, risk factors, hospital course and outcome of peripartum cardiomyopathy in our set up.

METHODOLOGY

This descriptive study was conducted from January 1st 2008 to December 30th 2011 in the Department of Cardiology, Postgraduate Medical Institute, Govt. Lady Reading Hospital Peshawar. Hospital ethical committee approved study protocol. Peripartum cardiomyopathy was diagnosed as given in the introduction.

The mortality rate mentioned in one study is 25-50%.[22] Using World Health Organization table for sample size determination with confidence level of 95%, margin of error 10% and the above response distribution, sample size of 100 to 225 patients is needed. However due the rare nature of the disease we collect only 61 patients. Patients of any age fulfilling the above diagnostic criteria were enrolled in the study.

Patients with previous history of cardiac disease including valvular heart disease, congenital heart diseases, cardiomyopathies of any cause and pulmonary artery hypertension either primary or secondary, cardiac failure due to severe preeclampsia, fluid overload and amniotic fluid embolism were excluded. Patients with normal echocardiographic findings were also excluded from the study. Informed written consent was obtained from each patient. Permanent residential address and telephone number was obtained from every patients to ensure effective follow up. Every patient is given a computer ID to ensure data retrieval.

During evaluation of patients, risk factors responsible for PPCM including age, race, parity, twin pregnancy, obesity, chronic hypertension, preeclampsia and malnutrition were

noted. Chronic hypertension was taken as elevated blood pressure of > 140/90 mm of Hg on three occasions and pre-eclampsia as blood pressure of > 140/90 with proteinuria after 20 weeks of pregnancy. Obesity was defined as Body Mass Index (BMI) of > 30. Considering an average weight gain of 8-12 Kg in pregnancy with no idea in most of the patients of pre-pregnancy weight, BMI was taken as estimates of obesity in the study patients.

Presenting clinical features including antepartum or postpartum status, dyspnea, cough, hemoptysis and fatigue were noted. ECG features like sinus tachycardia, left ventricular hypertrophy, repolarization changes, premature ventricular contractions, T wave inversion, low voltage QRS and left bundle branch block were recorded.

Echocardiographic features like chamber dilation, ejection fraction, mitral regurgitation, pulmonary artery hypertension and left ventricular thrombus were recorded. Chamber size was measured using two-dimensional (2-D) short axis view and applying M-Mode. Left ventricular diastolic dimension greater than 5.5 cm was taken as chamber dilation. Ejection Fraction was measured with Simpson's method and considered low when less than 45 and normal when greater than 55.

Patients were treated on standard lines for heart failure according to the current 2010 ESC guidelines for HF and monitored for complications including cardiogenic shock, pulmonary edema, thromboembolism, ventricular-tachycardias, atrial fibrillation and cardiopulmonary arrest. Complications were managed on standard lines and need for ICU care was assessed.

Candidacy for ICD, CRT, LV assist device and cardiac transplant was assessed using current European guidelines. Patients who died during hospitalization were recorded. Patients, once stabilized, were discharged on standard medical therapy and scheduled for follow up at 1, 3, 6 and 12 months. If patients needed re-hospitalization she was admitted with same ID to ensure correct number of hospitalization.

At the expected date of follow up, telephone reminder was given to patients or her relatives about follow up. At follow up it was confirmed that patient is alive and her NYHA class was noted. ECG and echocardiography were performed and parameters noted. Recovery was assessed by improvement in functional class of dyspnea and ejection fraction on repeat echocardiogram.

During follow up particular attention was focused on the occurrence of subsequent pregnancies. Patients were followed for eighteen months and pregnancy screening was done with ß- HCG pregnancy test.

Statistical Analysis was performed using statistical package for social sciences (SPSS) version 19.0. Numerical variables were presented as mean±SD and categorical variables as frequency and percentages.

RESULTS

A total of 61 patients were included in the study. The mean age was 30.94±6.63 years. Most of patients 33(54.1%) were obese with mean body weight of 73.5±6.91kg. The mean parity was 3.66 ±1.41. Majority 49(80.3%) had more than three children. Other risk factors were chronic hypertension 19 (31.1%), pre-eclampsia 12 (19.7%), multiple pregnancy 5(8.2%), long term tocolysis 13 (21.3%) and anemia 21(34.4%). The incidence was estimated to be 1 per 933 deliveries. These are summarized in Table 1.

Forty-three patients 43(70.5%) presented in post partum period while 18(29.5%) in antepartum period. Majority of patients presented with dyspnea and were in NYHA class III 18(29.5%) & IV 35(57.5%). Other presenting complaints were chest pain 36(59%), palpitation 27(44.3%), cough 27(44.3%) and fatigue 30(49.2%). Mean delay in diagnosis was 6.5±3.8days.

The main ECG finding was LVH 68.9%. On echocardiography mean ejection fraction was 29.29±10.06 and it was universally reduced. ECG and Echo findings are shown in Table 2.

Thirty-six women 36(59%) had normal vaginal delivery, 12 (19.7%) had assisted vaginal delivery and 13(21.3%) required caesarean section. There were 50(82%) live births and 11(18%) perinatal deaths.

Hospital course was complicated in few patients. The main complications during hospitalization in patients of peripartum cardiomyopathy are summarized in Table 3.

During hospitalization patients were treated on standard lines for heart failure. Major therapeutics used were intravenous frusimide 61(100%), frusimide/ spirolactone combination 61(100%),metolazone 16(26.22%), angiotensin converting enzyme inhibitors (ACEI) 39(63.9%), angiotensin receptor blockers (ARBs)5(8.2), hydralazine/nitrates combination 16(26.2), beta blockers 43(70.5%), bromocriptine 23(37.7%), digoxin 26(42.6%) and warfarin 12(19.7%). Some patients needed specialized therapies for heart failure including implantable cardiac defibrillator (ICD) 5(8.2%), cardiac resynchronization therapy (CRT) 3(4.9%) and cardiac transplantation or left ventricular assist device 8(13.1%).

At the last follow up, total of 20(32.8%) were dead while 32(52.5%) had recovered fully and 9(14.75%) were still suffering from heart failure. Outcomes at hospital discharge, at 3,6,12 months are shown in Table 4.

Particular attention was focused on subsequent

Table1: Summarizing the So-Called Risk Factors Responsible for PPCM

Characteristics	No (%)	Mean ± SD
Age(Years)		30.94±6.63
18-30	28(45.9)	
>30	33(54.1)	
White Race	43(70.5)	
Black Race	18(29.5)	
Parity		
1	3(4.9)	
2	9(14.8)	
≥3	49(80.3)	
Mean parity		3.66±1.41
Multiple Pregnancy	5(8.2)	
Chronic hypertension	19(31.1)	
Pre-eclampsia	12(19.7)	
Body weight (Kg)		73.5±6.91
Obesity	33(54.1)	
Anemia	21(34.4)	
Smoking	0	
Alcoholism	0	
Long-term Tocolysis	13(21.3)	

pregnancies. We followed forty one patients. Due to effective counseling we were able to detect only 8(19.5%) pregnancies. These were patients who had recovered fully from their initial illness. Among these patients 5(62.5%) had uneventful course while 3(37.5%) developed heart failure again. No death occurred in these patients.

DISCUSSION

Very little is known about the incidence of PPCM. From the available literature, the incidence of PPCM appears to be around 1 in 2500–4000 in the USA, 1 in 1000 in South Africa, and 1 in 300 in Haiti.[17] Our institution is tertiary care referral centre receiving cases from local population as well as from Afghanistan so the exact incidence cannot be predicted due to unknown number of deliveries for these cases. We got only 30 cases from our hospital where averagely 7200 deliveries occurred annually. Using this number the estimated incidence is 1 per 960 deliveries.

Common reported risk factors for PPCM are advanced maternal age, multiparity, multiple gestations, black race, obesity, malnutrition, gestational hypertension, pre-eclampsia,poor antenatal care, alcohol and tobacco abuse, low socioeconomic conditions and long term tocolysis as found invarious studies.[18] In our study the most significant risk factors found were advancing maternal age, multiparity, obesity, chronic hypertension and pre-eclampsia and long term tocolysis. PPCM has been reported mostly in women older than30 years.[18] In our study also the mean age noted was30.94 ± 6.63 years. Thirty-three(54.1%) patients were above thirty years of age. More than half of our patients (54.1%) were obese with a mean body weight of 73.5±6.91 Kg indicating obesity as a risk factor.[18] This condition has been described in multiparous women.[18] In our study most of patients (80.3%) were multiparous. This condition is also more frequent in women with multiple gestations.[18] However in our study multiple gestations were only 8.2%. In the USA majority of such patients are of African-American origin though Asians, Hispanic and Caucasian mothers are also affected.[19]

Table 2: ECG and Echo Findings in Patients with PPCM

Investigation Findings	No (%)	Mean±SD
Presenting ECG Findings		
Sinus Tachycardia	39(63.9)	
Left ventricular hypertrophy	42(68.9)	
T wave inversion	28(45.9)	
Poor R wave progression in precordial leads	40(65.6)	
Left Bundle Branch Block	9(14.8)	
Frequent PVCs	13(21.3)	
Echocardiographic Findings		
Ejection fraction (EF %)		29.29±10.06
Chamber dilation	48(78.7)	
Moderate to Severe mitral regurgitation(MR)	15(24.6)	
Left ventricular thrombus	12(19.7)	
Pulmonary artery hypertension	15(24.6)	

Table 3: Complications during Hospitalization in Patients with PPCM

Complication	Frequency (percentage %)
Ventricular tachycardia (VT)	8(13.1)
Atrial Fibrillation (AF)	8(13.1)
Cardiopulmonary Arrest	5(8.2)
Pulmonary edema	7(11.5)
Cardiogenic Shock	8(13.1)
Pericardial effusion	5(8.2)
Thromboembolism	13(21.3)
Need for intensive care (ICU)	23(37.7)

In our study majority of the patients (70.5%) were of Pathan's ethnic origin which is white race. Pre-eclampsia and chronic hypertension has been associated with a significant number of PPCM cases invarious studies.[8] Our study showed an association of 50%. Similarly, long term tocolysis with oral salbutamol and terbutaline in women with preterm labour especially if combined with antenatal steroid administration for fetal lung maturation is a risk factor.[18] Twenty-one (21.3%) patients in our study received tocolysis and later developed cardiomyopathy. The reason for the association of PPCM with the above risk factors is not fully understood.

All patients presented with dyspnea and were in NYHA class III (29.5%) and IV (57.5%) in our study as is seen in other studies.[20] Majority of women presented in postpartum period. It is evident from other studies in which postpartum presentation was 78%.[21] The diagnosis of PPCM is often made late. The delay in reaching a correct diagnosis ranged from weeks to months in around 30% of cases.[22] In our study it was 6.5±3.86 days.

Various studies had described ECG findings in peripartum cardiomyopathies. According to those studies sinus-tachycardia (68.4%), cavity hypertrophy (78.8%) and T wave inversion (47.3%) were more frequent findings.[17] In our

Table 4: Outcomes at Hospital Discharge, at 3 Month, 6 Month and 12 Months in Patients with PPCM

Outcomes at Hospital	n (%)	Mean±SD
Hospital death at first admission	9(14.8)	
Discharge with stable heart failure	52(85.2)	
Hospital stay(days)		6.62±2.31
At 3 Months		
Ejection Fraction (EF %) at 3 months		48.04±13.84
Deaths at 3 months	6(9.8)	
Recovery at 3 months	33(54.1)	
Heart failure at 3 months	13(21.3)	
At 6 Months		
Ejection Fraction (EF %) at 6 months		50.34±14.44
Deaths at 6 months	3(4.9)	
Recovery at 6 months	32(52.5)	
Heart failure at 6 months	11(18.03)	
At 12 Months		
Ejection Fraction (EF %) at 12months		53.37±11.56
Deaths at 12 months	2(3.3)	
Recovery at 12 months	32(52.5)	
Heart failure at 12 months	9(14.75)	
Total		
Recurrent Hospitalization		2.88±2.09
Total recovery	32(52.5)	
Patients left with disabling heart failure	9(14.75)	
Total death since diagnosis	20(32.8)	

study sinus tachycardia was 63.9% while LV hypertrophy and T wave inversion were 68.9% and 45.9% respectively. Similarly various studies have reported echo-cardiographic features of peripartum cardiomyopathies. According to those reduced ejection fraction, chamber dilation, moderate to severe mitral regurgitation, left ventricular thrombus and raised pulmonary artery pressure were the frequent findings. The findings in our study are consistent with those studies.[23]

Various complications have been described in patients with peripartum cardiomyopathy. These include pulmonary edema,cardiogenic shock; thromboembolic events, ventricular tachycardia, cardiopulmonary arrest and atrial fibrillation.[5] In our study these were also the major complications emerged.

Various studies have described mode of delivery and neonatal outcome in such patients. In study by Jahan Ara Hasan et al, 68.75% of patients had normal vaginal delivery, 6.2 % assisted vaginal delivery and 31.6% caesarean section.[5] In our study 59% of patients had normal vaginal delivery,19.7% assisted vaginal delivery and 21.3% required caesarean section mainly due to obstetric reasons. Regarding neonatal outcome 82% babies were live born, and 18% perinatal deaths occurred which is also in accordance with published literature.[5] Main cause of perinatal deaths were prematurity, IUGR and associated congestive cardiac failure in mothers.

The prognosis for women with PPCM appears to depend on the normalization of left ventricular size and function within 6

months after delivery. In one study, approximately half of 27 women studied had persistent left ventricular dysfunction. In this group, the cardiac mortality rate was 85% over 5 years, compared with the group in whom cardiac size returned to normal, who experienced no reported cardiac mortality in the same time interval.[2] A more recent study corroborates these results: 50%(7/14) of patients had dramatic improvement soon after delivery, but 6 of the 7 remaining patients died. Survivors were found to have a higher mean ejection fraction (23%vs.11%) and smaller mean left ventricular cavity size (5.8vs.6.9cm) at diagnosis.[23] In our study recovery as evidenced by improvement in clinical features and normalization of echocardiographic findings at 12 months was observed in 52.5% of patients. Mean hospital stay was 6.62±2.31 days. Prognosis is related to left ventricular dysfunction at presentation and recovery as shown in various studies. Recovery mostly occurs in first 2 months but it can take 6-12 months as is evident from our study also.[24]

Family-planning counseling is very important as women with PPCM are usually in the middle of family building. Only a few studies have reported on subsequent pregnancies of women with a history of PPCM. In a retrospective investigation, Elkayam et al, studied 44 women with PPCM and a subsequent pregnancy and found that LVEF increased after the index pregnancy but decreased again during the subsequent pregnancy, irrespective of earlier values.[17] Development of HF symptoms were more frequent in the group where LVEF had not normalized before the subsequent pregnancy (44 vs. 21%). In addition, three of the women with a persistently low LVEF entering the subsequent pregnancy died, whereas none with normalized LVEF died. In our study we detected only eight pregnancies in which five were asymptomatic and three developed heart failure. These were those patients who had recovered completely from their initial heart failure.

CONCLUSION

Peripartum cardiomyopathy (PPCM) is a rare but potentially life-threatening illness. All women having clinical features suggestive of PPCM should be evaluated using echo-cardiography and modern diagnostic criteria. Standard management of cardiac failure using a multi disciplinary approach should be started. Patients should be followed for recovery and those with persistent ventricular dysfunction should be properly counseled for contraception and avoidance of pregnancy. Subsequent future pregnancies if occurs, then should be managed in multi disciplinary units.

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