7 Year Prospective Study of Peripartum Cardiomyopathy in a Medical College Hospital

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ABSTRACT

Peripartum cardiomyopathy is a disorder characterized by left ventricular dysfunction and heart failure in pregnant females in the absence of any other cardiac disease. PPCM is an under diagnosed entity. Severity varies from region to region and there is an ethnic disparity. PPCM is multifactorial. Virchow observed the occurrence of heart failure in pregnant females in 1890 but it was Goueley who reported the occurrence of cardiomyopathy in pregnancy in 1937. The incidence occurs one in 3000-15000 pregnancies, more incidents were reported in Africa. This condition varies in its definition, epidemiology, pathophysiology, clinical presentation, diagnosis, management and outcomes in morbidity and mortality. Future pregnancies are discouraged in women with persistently low LVEF[8]. Mortality is more in patients with low LVEF[9]. The epidemiology of PPCM is largely unknown in ASIA[4]. Mortality in USA due to PPCM has been described by Whitehead.

Peripartum cardiomyopathy has been extensively studied in 'Recommendation and review by national institute of health' & was published in JAMA, March 1, 2000-Vol 283 No.9 .

We carried out a prospective study w.e.f. 1st July 2015 to 30th June 2019 and followed 22 patients for a further 24 months for observing changes in LVEF and its impact on morbidity and mortality. The study was concluded on 30th June 2021.

Keyword: Epidemiology, Peripartum Cardiomyopathy (PPCM), Pregnancy, Left Ventricular End Diastolic Diameter, LVEF (Left Ventricular Ejection Fraction).

INTRODUCTION

Peripartum cardiomyopathy is an uncommon form of left ventricular dysfunction and congestive cardiac failure. In 1890 Virchow observed presence of heart failure without any aetiology in pregnancy, but it was Goueley who described an association between heart failure and pregnancy in females who did not have any recognizable heart disease before the last month of pregnancy. This condition occurs in 3000-15000 in pregnancy and more in AFRICA.[2,3] This condition varies in epidemiology, pathophysiology, clinical presentation, diagnosis, morbidity and mortality in different studies.[3,4,5,6]

Demakis and Rahimtoola[5] in 1971 established the criteria for diagnosis which included: a) Heart failure manifesting during the last month of pregnancy or within 5 months of delivery and the diagnosis of heart failure is without any obvious aetiology. National Heart, Lung and blood institute (NHLBI) published an article in JAMA[10] defining PPCM which include: a) Development of HF during last month of pregnancy or within five months of delivery; b) Absence of definite identifiable cause of heart failure; c) Absence of recognizable cause of heart failure during the last month of pregnancy and d) Presence of left ventricular dysfunction with LVEF less than 45% by echocardiography.

The cause and prognosis of PPCM is variable. Sarifstein et al[11,13] in his prospective study reported a LVEF of >35% at diagnosis, breast feeding and diagnosis of PPCM during post partum stage were associated with better prognosis. Repeated pregnancy in females with reduced LVEF of less than 35% had a bad prognosis[9,11]. Maternal mortality rate in USA have been estimated to as high as 25%-50% [14]. Deaths have been attributed to presence of arrhythmia and thromboembolism [15].

Epidemiology

The prevalence of PPCM at our centre was 22 per 24,464 live birth and 4 deaths in this group which was almost similar to studies at other centers. Pandit et al[18] reported 1 death per 1374 live births and 72 cases of PPCM per 100000 live births.

DISCUSSION

Risk factors: Multiple risk factors have been identified by PPCM which included advanced maternal age, multiparity, complicated pregnancy like preeclampsia, gestational hypertension, diabetes[19,20,21].

Aetiology: Aetiology in PPCM is unknown. Precise mechanism which leads to PPCM is ill understood. Recently a familial predisposition to PPCM has been reported[22,23,24]. Inhibition of prolactin activity with bromocriptine estimation might be helpful and hence prolactin might be playing a role in PPCM[31]. Recently European Society of Cardiology working group has suggested PPCM to be classified as a non-familial, non-genetic form of dilated cardiomyopathy[25].

Pathophysiology: PPCM may occur from multifactorial in origin. It may be by viral myocarditis, abnormal immune response during pregnancy, maladaptive process to haemodynamic stress of pregnancy, selenium deficiencies,

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malnutrition[26,27,28]. Apoptosis, inflammation, prolonged tocolysis, development of pulmonary oedema may lead to PPCM.

Pathology: The heart is flabby, pale, hypertrophied with interstitial oedema with dilatation of all chambers [29,30].

Differential diagnosis: The differential diagnoses are AMI, amniotic fluid embolism, severe pre-eclampsia, pericarditis, pulmonary thrombo-embolism, myocarditis, sepsis, drug toxicities, metabolic disorders and aortic dissection.

MATERIAL AND METHODS

We carried out a retrospective study of all pregnant females coming to our hospital between 1st July 2015 to 30th June 2019 and carried out a thorough clinical examination, biochemical and haematological investigations.

During this period a total number of 24,464 live births were recorded in our hospital. Out of these patients, we carried out thorough cardiological examination who exhibited evidence of heart disease such as crepitations on auscultation of chest, paroxysmal nocturnal dyspnea, chest pain, cough, appearance of murmurs, raised jugular venous pulsation and evidence of pitting oedema on dependent parts of the body. We carried out ECG, 2D Echocardiography and doppler study in all these cases and criteria for diagnosis of PPCM were strictly followed,

1. Onset of heart failure in the last month of pregnancy or in the first 5 months postpartum.
2. Absence of heart failure due to causes like Rheumatic heart disease, thyroid disorders, pulmonary embolism, pulmonary hypertension; primary or secondary due to chronic obstructive pulmonary disease.
3. Absence of recognizable heart failure before last month of pregnancy.
4. Echocardiographic evidence of left ventricular dysfunction or failure within last month of pregnancy and/or 5 months postpartum. The classical echocardiographic evidence suggests LVEF less than 45%, fractional

Variable factors	No. of patients	Range	Percentage
Age at diagnosis in years	25.2±6.9	14-40	
Gravidity	2±1.2	1-4	
Parity	1.8±1.2	1-4	
Anaemia	12		=(12/22)*100
Hypertension	10		10/22*100
Delivery time	36.6±3.2	28-40	
Delivery mode			
Veginal	12		=(12/22)*100
CS	10		10/22*100
Twin Pregnancy	2		2/22*100
Time of Diagnosis			
Antepartum	4		4/22*100
Postpartum	18		18/22*100
Symptomatic	20		20/22*100
Dysproea	18		18/22*100
Fatigue	17		17/22*100
Oedema	13		13/22*100
Chest Discomfort	10		10/22*100
Palpation	6		6/22*100
NYHA Class			
1	4		4/22*100
2-3	12		12/22*100
4	6		6/22*100
Medications	22		22/22*100
	16		16/22*100
Beta Blockers	14		14/22*100
Digoxin	12		12/22*100
ACE	18		18/22*100
ARB	4		4/22*100
Anticoagulant	7		
Follow up	24 months		
Death	4/22		
Recovery	12/22		
Remained symptomatic	6/22		
Out of surviving patients, total no.18 there was a comparison between patients who recovered and became asymptomatic (GpA no.12) and patients who did not recover and remained symptomatic (GpB-No. 6) at the end of 24 months of follow up which is depicted in Table II.			

Table-1: Summary of demographic and clinical data of 22 patients who qualified to be included in our study.

	Group A		Group B		Group C		Fatality
	LVEF	LVEDD	LVEF	LVEDD	LVEF	LVEDD	
At diagnosis	28.6 (24-42.4)	59.4 (56-70)	22.1 (16.2-32.4)	63.2 (54-69)	18.2 (16-32.4)	64.8(59-64)	Nil
At 6 months	36.4 (28.2-44.4)		26.2 (19.2-38.4)	59 (50-64.4)	(16.4-34.2)	62.6(58.2-63)	1
At 12 months	41.3 (32.4-49.6)		29.2 (22.4-42.6)	54 (51.3-60.3)	(17.2-35.2)	59.8(57.2-64)	1
At 18 months	44.8 (38.3-56.3)		35.6 (29.3-46.8)	49.8 (50-59.3)	16%	57	1
At 24 months	47.8 (40.4-60.2)		38.4 (32.4-50.4)	44.2 (48.4-54.3)	-	-	-

Table-2: Comparison of 3 groups (A,B &C) for recovered/asymptomatic Gp A, N-12 symptomatic Gp B No-6 and fatality Gp C No.4

shortening less than 30% or left ventricular end diastolic diameter of less than 2.7 cm^2/sq meter of body surface. [10]

Observations

Out of the 24,464 live births, 22 females fulfilled the criteria for PPCM in our study. (0.89% of total live births)

We followed up these 22 cases for a further 24 months for mortality, recovery or patients who remained symptomatic during this period in spite of treatment. Treatment was carried out with diuretics, digoxin, beta blockers, ACE inhibitors, ARBs or anticoagulants.

All the patients (N:22) underwent 2D Echocardiography which is the most important diagnostic tool for diagnosis of PPCM[16].

The mean LVEF was 28.9% $\pm$ 8.5% (Range 15%-42%)

MR cardiac magnetic resonance can be used to measure global & segmental myocardial contraction & inflammation of cardiac muscles[17]. After the initial 2D Echo examinations serial 2D echo was done at every 3 months interval.

RESULTS

Out of 24,464 live births in our hospital during the study period, 22 females(0.89%) qualified to be included in our study after fulfilling the criteria laid down by national heart Lung and Blood Institute and office of rare diseases (National Institute of Health).[10]

The median age of these females were 18-44. Gravidity was 3$\pm$ 1.6. All these females exhibited the signs and symptom of congestive heart failure. 20 patients were symptomatic, dyspnoea was present in 18 patients, fatigue in 17 patients, oedema in 13 patients and chest discomfort in 10 patients. Palpitation was complained by 6 patients. We lost 4 patients during this 24 months follow up and 18 patients remained out of which 12 were asymptomatic (Gp A) and 6 patients remained symptomatic (Gp B). 4 patients died during follow up period (Gp C).

2 patients died of refractory congestive heart failure as a consequence of sepsis. 1 patient died because of atrial fibrillation (Flash AF) with large embolism to the brain and 1 patient died due to hypokalaemia and ventricular fibrillation. Management was similar to that of treatment for other dilated cardiomyopathies. Aim was to minimize signs & symptoms of oedema to improve haemodynamics. Na^+ restriction, diuretic, vasodilators and low dose beta blockers were helpful.

Ventilatory assistance was given where there was persistent hypoxia below 90%. Majority deaths occurred within first 3 months. Aggressive treatment was required during this period. About 50% females continued to have symptoms of heart failure and cardiomegaly beyond 6 months. These patients were counselled against having subsequent pregnancy as mortality in subsequent pregnancy was likely to be high. Factors associated with poor prognosis were large left ventricular end diastolic diameter, low left ventricular ejection fraction, 5 months after delivery, advanced maternal age, multiparity.

Prevention of pregnancy in women in child bearing age is likely to be traumatic psychologically in women and hence counseling should be done with utmost care and sympathy. In spite of counseling authors have encountered two episodes of pregnancy in 2 females at 30th week for which elective CS was carried out at 36th week in both the females. The risk of maternal death was 19% higher[32] in previous studies, but we were lucky that both the females and infants survived in our study.

CONCLUSION

PPCM is a rare form of cardiomyopathy and high index of suspension, exclusion of pre-existing cardiac disease and other causes of cardiac ailment and findings of classical echocardiographic picture clinch the diagnosis[10]. The disease is multifactorial in its origin, careful assessment of the possible risk factors encountered during clinical examinations and investigations may be helpful in prevention of PPCM[33].

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