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Association of Peripartum Cardiomyopathy with Severe Preeclamsia

Rujuta Fuke[1], Smita Rokade[2]

[1]Associate Professor in Obstetrics and Gynecology at Government Medical College and Hospital Nagpur, India

[2]Post-Graduate Student in Obstetrics and Gynecology at Government Medical College Nagpur, India

Abstract: ***The outcome of peripartum cardiomyopathy (PPCM) is highly variable. The strong epidemiological relationship is observed between Preeclampsia (PE) and peripartum cardiomyopathy (PPCM). Echocardiography forms essential diagnostic tool to diagnose PPCM. It is useful for early diagnosis as well as prognosis of the disease when performed in suspected subjects. The aim of our study is to see the association between preeclampsia and peripartum cardiomyopathy with the help of Echocardiography which will help in early diagnosis of patients. We investigated total 260 study subjects with severe preeclampsia with the help of echocardiography and diagnosed 24 new patients with PPCM. The current study demonstrates that hypertensive disorders of pregnancy is a strong risk factor for the development of PPCM. The results of the study also support the novel concept that PE and PPCM share a standard antivascular pathobiology. Effectiveness of echocardiography obviates the need for invasive cardiac catheterization and help in prompt diagnosis.***

Keywords: Peripartum cardiomyopathy, preeclampsia, echocardiography, congestive heart failure

1. Introduction

Congestive heart failure (HF), occurring during the peripartum period, was first described in 1849.[1] However, it was not until the 1930s when it was officially recognized as a clinical entity occurring as a consequence of pregnancy.[2] In the 1970s, a 20-year experience following 27 patients who developed cardiomegaly in the puerperium was published, and the term peripartum cardiomyopathy (PPCM) was established by Demakis and Rahimtoola in 1971.[3] Since then, we have developed a greater understanding through data collection and improved diagnostic methods resulting in PPCM becoming a well-defined form of HF. Today, PPCM remains a rare yet significant cause of maternal morbidity and mortality. Peripartum cardiomyopathy (PPCM) is defined as the development of cardiac failure between the last month of pregnancy and 5 months postpartum, the absence of an identifiable cause, the absence of recognizable heart disease prior to the last month of pregnancy, and left ventricular systolic dysfunction demonstrated by classic echocardiographic criteria.

The strong epidemiological relationship observed between Preeclampsia and PPCM suggests that Preeclampsia may be part of a pathway that leads to impairments in cardiac function. Consistent with this notion, several studies have recently shown that women with preeclampsia have evidence of diastolic dysfunction, as well as impaired systolic strain despite preservation of global systolic function as assessed by ejection fraction. Gestational hypertension without preeclampsia does not cause similar cardiac dysfunction, indicating that aspects of preeclampsia other than elevation of blood pressure contribute to cardiac dysfunction. Moreover, cardiac dysfunction associated with preeclampsia persists for at least a year, despite normalization of blood pressure.

The aim of our study is to see the association between preeclampsia and peripartum cardiomyopathy with the help of Echocardiography which will help in early diagnosis of patients.

2. Literature Survey

2.1 Kathryn J. Lindley, 2017

Peripartum cardiomyopathy (PPCM) is a distinct type of heart failure that occurs within the last month of pregnancy or within 5 months following delivery. It is defined as left ventricular ejection fraction ≤45% ± left ventricular (LV) cavity dilation occurring during the peripartum period in the absence of pre-existing heart disease or other identifiable causes of heart failure.

In this study, follow-up echocardiographic analysis was performed for all women with a diagnosis of severe preeclampsia with an echo performed between 6–24 months after diagnosis. If women had an echo between 1–6 months after diagnosis that documented recovery of LV function, they were also included in the analysis.

Conclusions-The study was the first to investigate the impact of preeclampsia on outcomes in women with PPCM. They observed that there was a high incidence of preeclampsia in the predominantly African American population of women diagnosed with PPCM. Despite comparable LV ejection fractions in the two groups, PPCM with preeclampsia is associated with excess early morbidity and mortality. Moreover, the patterns of LV remodeling and recovery of LV function were distinctly different in PPCM patients with preeclampsia than in PPCM patients without

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preeclampsia. Apart from the novelty of these findings, this study has a number of important clinical implications. First, while future pregnancies have been considered absolutely contraindicated only in women with PPCM with residual LV dysfunction, it is unknown if the risk of future pregnancy is equivalent for women with prior PPCM with vs. without associated preeclampsia, and whether residual diastolic dysfunction affects this risk. Their results suggest that despite complete normalization of LV ejection fraction in PPCM associated with preeclampsia, these patients remain at high risk of recurrent hospitalization and/or death. If results are replicated in a larger patient cohort, they may lead to a rethinking of recommendations for future pregnancies in PPCM with hypertensive disorders.

2.2 Feriel Azibani and Karen Sliwa: meta-analysis study

In this review, they outlined recent reports about the disease pathogenesis and management and highlighted the use of diagnosis and prognosis biomarkers.

Novel data strengthen the implication of endothelial function in PPCM pathogenesis. The first international registry showed that patient presentations were similar globally, with heterogeneity in patient management and outcome

Despite large improvement in patient management and treatment, there is still a sub-group of women who die from PPCM or who will not recover their cardiac function. Remarkable advances in the comprehension of disease incidence, pathogenesis, and prognosis could be determined with multi-center and international registries.

2.3 MICHAEL C HONIGBERG, BMJ 2019

Preeclampsia and eclampsia are associated with PPCM, which, as discussed below, may reflect shared pathophysiology. A 2013 meta-analysis of 22 studies found a 22% prevalence of preeclampsia among women with PPCM, more than four times the estimated global prevalence. Similarly, of the first 411 women in the EUR Observational Research Programme PPCM registry, 22.8% had preeclampsia. Inflammation is variably present in endomyocardial biopsies taken from women with the condition, but few patients meet histologic criteria for myocarditis. Of 40 women in the Investigations in Pregnancy-Associated Cardiomyopathy (IPAC) cohort who underwent cardiac magnetic resonance (CMR) imaging, only one had findings potentially consistent with myocarditis. Although inflammatory markers are raised in women with PPCM, the underlying driver does not seem to be infectious.

Genetic predisposition: It has long been observed that some cases of PPCM cluster in families. Analysis of pedigrees affected by the condition and registries of dilated cardiomyopathy (DCM) identified variants in genes that encode the sarcomeric proteins titin, myosin, and troponin.

Placental angiogenic factors: The high prevalence of pre-eclampsia in women with PPCM suggests a possible shared pathophysiology. Soluble fms-like tyrosine kinase receptor 1 (sFlt-1) is an anti-angiogenic protein secreted by the placenta in exponentially increasing amounts towards the end of pregnancy. sFlt-1 sequesters circulating vascular endothelial growth factor (VEGF) and placental growth factor (PlGF) and is thought to be the major driver of hypertension and endothelial dysfunction in pre-eclampsia. In addition, sFlt-1 levels correlate with global longitudinal strain and increased left ventricular mass in women with pre-eclampsia.

A diagnosis of PPCM requires echocardiographic evidence of left ventricular dysfunction with LVEF <45% and often (but not always) left ventricular dilatation; presentation peripartum or in the early postpartum period; and absence of an alternative explanation. Clinicians must remain vigilant for a diagnosis of PPCM because its symptoms overlap with those of normal pregnancy and it may be missed on initial evaluation. The differential diagnosis includes pre-existing cardiomyopathy, such as familial dilated cardiomyopathy, previous myocarditis, and drug or toxin induced cardiomyopathy; valvular disease, with mitral stenosis and aortic stenosis being the most common valvular abnormalities to be unmasked by pregnancy; congenital heart disease, such as shunt lesions; and pulmonary arterial hypertension .Conclusions: Peripartum cardiomyopathy is an uncommon but serious medical condition that affects women throughout the world. While the underlying pathophysiology remains unclear, vasculo - hormonal influences and genetic susceptibility probably play a role.

3. Problem definition

Diagnostic criteria for peripartum cardiomyopathy, as follows:

1. Development of heart failure in last month of pregnancy or 5 months postpartum
2. Absence of preexisting heart disease
3. Indeterminant cause
4. Echocardiographic findings (a, together with b or c; or all of these)
   a. Left ventricular end-diastolic dimension >2.7 cm/m²
   b. M-mode fractional shortening <30%
   c. Left ventricular ejection fraction <0.45

Adapted from Demakis JG, Rahimtoola SH. Peripartum cardiomyopathy. Circulation 1971;44(5):964-8[1]; and from Manolio TA, Baughman KL, Rodeheffer R, Pearson TA, Bristow JD, Michels VV, et al. Prevalence and etiology of idiopathic dilated cardiomyopathy (summary of a National Heart, Lung, and Blood Institute workshop). Am J Cardiol 1992;69(17):1458-66.[2]

PPCM is a rare form of dilated cardiomyopathy of unknown origin that is unique to the pregnant women of all reproductive ages. It affects previously healthy pregnant women with a low incidence of 0.1% of pregnancies but has a high morbidity and mortality rate ranging from 7% to 50%. The outcome of PPCM is highly variable.

In some patients, the clinical and echocardiographic parameters improve rapidly and may return to normal while for others it may progress and the clinical condition rapidly worsens, even with medical therapy to chronic cardiac

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failure and sudden cardiac death. PPCM affects 1 in 3,000 pregnancies, although it is more common in women of African descent.[3][4] It in as many as 1% of all pregnancies in certain "hot spots" like Haiti and Nigeria.[5],[6] The result is devastating for both mothers and infants. PPCM accounts for 5% of heart transplants in US women, an option not available in most of the world.[7] As many as 25% of women with PPCM in developing countries die within 5 years.[8] With an associated infant mortality rate as high as 50% to 75%. [9],[10] In the United States, the mortality rate for women with PPCM approximates 6% for white non-Hispanic women and is 4-fold higher in women of African descent. [11]

The etiology of PPCM remains enigmatic. Many hypotheses have been proposed, including viral myocarditis, autoimmunity, and fetal microchimerism. Hull and Hidden were first to describe a connection between postpartum heart failure and hypertensive heart disease in 1938 when they noted that >85% of cases of "'toxic' postpartal" heart conditions were associated with hypertension, which was double the frequency seen in their control group.[12] Demakis and Rahimtoola in their classic 1971 description of PPCM, reported that "toxemia," an older term for preeclampsia (PE), was detected in 22% of affected women. [13]

Symptoms of heart failure, such as dyspnea, edema and weight gain, resemble those complained about by normal peripartum women, which makes it very difficult to diagnose PPCM. At the same time, a low level of awareness and failure to routinely think of a cardiac cause for these symptoms are related to delayed diagnosis in many cases. In Japan, more than 60% of patients were initially seen by an obstetrician when they complained of heart failure symptoms, otherwise less than 10% were primarily managed by a cardiology specialist.[14] Goland et al reported that diagnosis delay was significantly associated with worse prognoses such as death or heart transplantation.[15] Increased awareness of PPCM is required for early diagnosis and better outcomes.

4. Methodology

4.1 Study Design

Prospective observational study

4.2 Study Setting

Department of Obstetrics and Gynaecology at the tertiary health care set up.

4.3 Study Period

January 2018 to november 2019 i.e. one and a half year.

4.4 Sample Size: 260 CASES

$$n = \frac{Z^2\,{}^{1-\alpha}/_{2}\,p(1-p)}{d^2}$$

Where,

- P: expected proportion (22%)
- D: absolute precision (5%)
- 1-α/2: desired confidence interval(95)

4.5 Study Population

All patients with severe preeclampsia admitted in study period

4.6 Inclusion Criteria

Women with severe preeclampsia (=/> BP 160-110)

4.7 Exclusion Criteria

- Patients with previously detected cardiac abnormalities
- Patients with severe heart failure with poor prognosis

4.8 Types of Outcome Measures

1) Burden of severe preeclampsia in study group.
2) Presence or absence of severe cardiac manifestations and its quantification in patients of severe preeclampsia.
3) Role of echocardiography in early diagnosis of cardiac derangements in patients of severe preeclampsia.
4) Prevalence of peripartum cardiomyopathy in severe preeclampsia.
5) During the study period of 18 months, 260 cases of severe preeclampsia presenting to the outpatient as well as casualty department of our center were registered in the study after taking written informed consent.
6) Study subjects were carefully selected after taking into consideration of all inclusion and exclusion criteria of study.
7) Patient is evaluated thoroughly, stabilized with careful monitoring of vital parameters.
8) Electrocardiographic and Echocardiographic evaluation of all study subjects was done.
9) The findings and data of all study subjects was analysed and we came up with following observations.

5. Observation

We have included 260 study subjects with severe preeclampsia into study with written informed consent. We did echocardiography in all subjects and diagnosed 24 new cases of peripartum cardiomyopathy. Our observations of study are as follows,

Table 1: Incidence Of PPCM

	Frequency	Percent
Pregnancies With PPCM	24	9.2%
With Other Cardiac Lesions	14	5.38%
Pregnancies Without CARDIAC Abnormality	222	85.38%
Total	260	100%

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In our study population Out of 260 cases of severe pre eclampsia, we diagnosed 24 new cases of peripartum cardiomyopathy (9.2%) cases. This showed the high incidence of PPCM in study subjects in our study.

Out of 260 cases, 14 cases (5.38%) were having minor non specific cardiac abnormalities like mild MR, mild TR, mild AR.

Remaining 222 patients of severe preeclampsia were not having any cardiac involvement, with normal 2D echocardiographic findings.

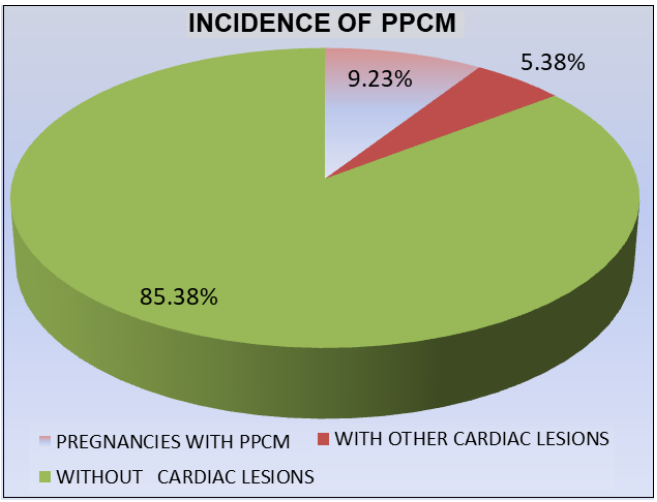

Figure 1

Table 2: Baseline haemodynamic parameters in study population

Parameter	Pregnancies With PPCM		Pregnancies Without PPCM		P-Value
	Mean	SD	Mean	SD	
Pulse Rate	116.54	3.49	92.51	9.64	<0.0001,HS
Systolic Blood Pressure	152.0	10.23	151.51	10.15	0.8232,NS
Diastolic Blood Pressure	103.41	6.05	101.75	7.46	0.2921,NS
Haemoglobin	9.68	1.40	9.64	0.97	0.8424,NS
SPO2	95.45	3.76	98.03	0.51	<0.0001,HS

All cases with PPCM are characterised by having tachycardia with mean pulse rate of 116.5±3.49. Association of tachycardia in PPCM study subjects is highly significant of the disease under study. Same applies for oxygen saturation of blood which is found to be reduced in PPCM cases as compared with study subjects without PPCM (mean value 95.45±3.76)

In contrast to these findings we have observed that mean values of systolic and diastolic blood pressure were statistically not significant to show the association with PPCM; reason being all the study subjects were having severe preeclampsia hence study subjects without PPCM were also having higher BP readings. So, It is difficult to show statistical association between PPCM and severe preeclampsia as cases with and without PPCM were having higher BP readings. But we can still prove the association based on our finding of increased incidence of PPCM among population of severe preeclampsia cases.

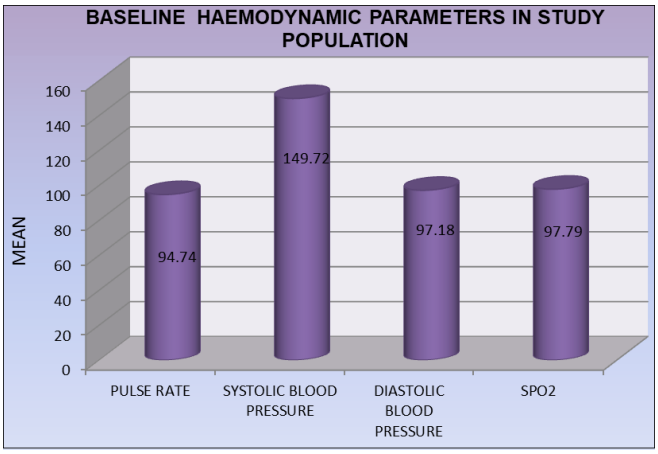

Figure 2

Table 3: Echocardiographic characteristics of PPCM

Parameter	Pregnancies With PPCM		Pregnancies Without PPCM		P-Value
LVIDd	47.62	3.94	39.75	1.05	<0.0001,HS
LVIDs	36.75	6.95	28.14	1.34	<0.0001,HS
LVEF	36.12	11.08	59.98	0.26	<0.0001,HS

Left ventricular end diastolic dimension in patients with PPCM is greatly increased as compared to patients without PPCM. Same is true for left ventricular end systolic dimensions. Whereas ejection fraction in patients with PPCM is markedly reduced as compared to patients without PPCM.(mean 36.12±11.08). These echocardiographic findings are highly significant of PPCM

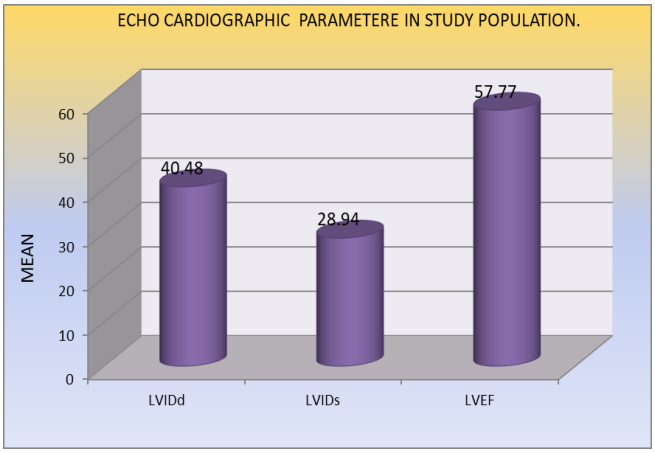

Figure 3

Table 4: Correlation of cardiogenic parameters and presence of PPCM

Ejection fraction%	Pregnancies With PPCM	Pregnancies Without PPCM	P-Value
20 – 30	10	0	Chi2=259.0 P<0.001,HS
31 – 40	6	0	
41 – 50	8	0	
>60	0	236	

Out of total 24 PPCM patients, 10 patients were having ejection fraction between 20-30%. 6 out of 24 were having ejection fraction between 31-40%. Rest 8 no of patients

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were having values of ejection fraction between 41-50%. Remaining patients without PPCM were 236 having ejection fraction of >60 %

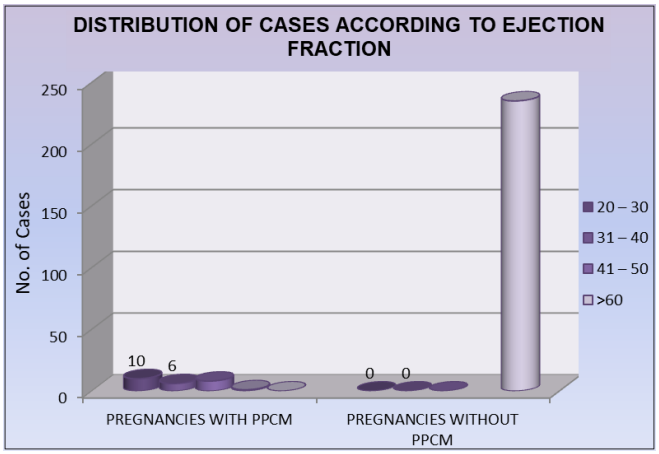

Figure 4

Table 5: Outcome of study

Outcome	No of Patients	Percentage%
Recovery	09	37.5
Residual Heart Disease	13	54.16
Mortality	02	8.33
Total(Ppcm)	24	100

Out of 24 PPCM patients, 09(37.5%) patients were recovered completely without any residual cardiac lesion. 13(54.16%) patients were having some residual cardiac lesions, not recovered completely. During study period 02(8.33%) mortalities were observed

6. Discussion

The connection between postpartum heart failure and hypertension was first described in 1938 by Hull and Hidden.[12] They observed that postpartum heart disease was associated with hypertension and occurred twice as often in women with hypertension during pregnancy than in the control population . This study showed that history of hypertension in current pregnancy is the most influential risk factor for PPCM. In our study, we found 24 (9.23%) patients with PPCM out of 260 cases of severe preeclampsia. 14 patients were having minor valvular heart lesions like mild TR, mild MR, trivial TR. These patients were asymptomatic and the findings were incidental on echocardiography.

A recent epidemiology report out of North Carolina shows that out of 79 PPCM patients, 51 (65%) had some form of hypertension. Eleven, (13.9%) had preeclampsia, 18 (22.8%) had gestational hypertension, 10 (12.7%) had chronic hypertension, 10 (12.7%) had chronic hypertension and preeclampsia, 1 had eclampsia. [16]

All the Earlier studies explained 41.2% of patients with PPCM in Japan have hypertension in pregnancy, with 42.8% being pre-eclampsia. Also, some other studies show the strong relationship between PPCM and hypertension in pregnancy.[14] Data in the US showed 45% of patients with PPCM have hypertension in pregnancy, 13.9% of which have pre-eclampsia.[17] Literature indicates nearly half of patients with PPCM had hypertension in pregnancy. A meta-analysis by N Bello et al suggests the prevalence of pre-eclampsia and other forms of hypertension in pregnancies is significantly higher in PPCM patients than the general population.[18] This suggests a strong relationship between hypertension in pregnancy and PPCM and therefore supports some theories that point out the similarities of pathogenesis between hypertension in pregnancy, especially pre-eclampsia, with PPCM.

To explore the association between Hypertensive disorders of pregnancy (HDP) and PPCM, a cohort study was done in Denmark by Behrens et al.[19] It identified 126 women with PPCM, 39 of whom had hypertensive disorders of pregnancy whereas 87 were normotensive. Most cases (82% vs. 81.6%) were diagnosed either during the final month of pregnancy or the first month of postpartum. Hypertensive disorders of pregnancy were found to be strongly associated with PPCM, and the magnitude of this association was proportional to the degree of hypertension.

Bello et al. found that the overall prevalence of Preeclampsia among patients with PPCM ranged from 0-78% in the included studies [18] They found the average prevalence to be 22%, which was more than four times the average worldwide prevalence of 3-5% [probability value (p): <0.001].

In another study by Kamiya et al., which looked at the characteristics of PPCM, 42 patients were found to be complicated with hypertensive disorders of pregnancy (HD+ group), and 60 patients were without the complication (HD- group).[20] In the article by Ersbøll et al., almost half of the women with PPCM had preeclampsia.[21]

Table 6: Echocardiographic characteristics and mortality

Author	Mc Namara [22]	Ntusi [23]	Biteker [24]	Cooper 2011	Sliwa [25]	Mishra [26]	Fett [27]	Current
LVEF	35 ± 10	24 ± 8	26 ± 6	27 ± 7	30 ± 9	31 ± 7	24	36 ± 7
LVEDD	5.5 ± 0.07	7.4 1.1	6.6 ± 0.6	—	5.66 ± 0.6	6.4 ± 1.25	5.9	4.7
Mortality	4	17	24	0	28	23	15	2

From above table we can observe that, there is relation between mean ejection fraction value and mortality. Studies having mean ejection fraction value of 20-30% has higher mortality rate, whereas current study and study by McNamara with ejection fraction >30% has mortality of 4 and 2 respectively.

We can say that In addition to diagnosis, echocardiography imaging provides valuable prognostic information on PPCM. Generally, PPCM has a favorable prognosis with slightly over half of the patients having a spontaneous recovering of Left ventricular function six months post-partum . However, delayed diagnosis, NYHA functional class and baseline LVEF ≤ 25% indicated a higher risk for

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mortality. Consistent with previous studies [29] LV functional parameters defined using echocardiography imaging modality is an accurate predictor of PPCM prognosis. Higher LVEF > 30%, LV fractional shortening, LVEDD and LVESD have a statistically significant correlation with subsequent improvement in LV function [28],[29]. Although in general PPCM has a low mortality rate whereas patients African ancestry, very low LVEF < 30%, and higher NYHA functional class suggests a greater risk of death [30],[31].

7. Conclusions

PPCM is multifactorial origin. Preeclampsia, Lower socioeconomic status, multiparty, multiple gestation all contribute for disease pathogenesis.
Echocardiography is essential for diagnosis as well as formulating prognosis for recovery, and following the course of the disease. Its effectiveness obviates the need for invasive cardiac catheterization. Prompt echocardiography in all symptomatic pregnant patients can help to diagnose patients with peripartum cardiomyopathy earlier in the course of the disease, thereby leading to earlier institution of care and to improvement of outcomes. Subsequent pregnancy in women who have been diagnosed with peripartum cardiomyopathy should be approached with great caution.

The current study demonstrates that hypertensive disorders of pregnancy are strongest risk factor for the development of PPCM throughout the world. The results of the study also support the novel concept that preeclampsia and PPCM share a common antivascular pathobiology contributing to significant cardiovascular morbidity and mortality in women of reproductive age. Our analysis highlights the need for better understanding of the mechanistic overlap between preeclampsia and PPCM, and a need to include patients with both these conditions in the same clinical studies. The prognosis of peripartum cardiomyopathy is best when diagnosed and treated early.

8. Future Scope

Despite the strong association between Hypertensive disorders of pregnancy and PPCM, most cases of PPCM (69%) occur in healthy normotensive pregnant females. [37] Certain susceptible women are unable to handle the hemodynamic stresses of pregnancy. A recent study showed that 18-28% of healthy nulliparous women had some degree of cardiac dysfunction at term [38].

The focus of most research work to date has been on Preeclampsia and PPCM as shown in the meta-analysis performed by Bello et al. examining the relationship between the two [39]

However, there is a growing need to design and perform more studies looking at the etiology and treatment of various pathologies involved in the causation of PPCM, especially in patients with preeclampsia. This can provide concrete evidence for the underlying mechanism as well as the association between the two conditions.

Table 7: Comparision Between Different Studies

1st Author [Ref. #]	Study Period	No. of Patients	Diagnosis Criteria	LV Recovery (LVEF > 50%) (%)	Predictors of LV Recovery	Mortality (%)
Fett, *et al.*[10]	2000-2005	92	LVEF< 45%	32	LVEF ≥ 30%; FS ≥ 20%	15.3
Duran, *et al.*[30]	1995-2007	33	LVEF< 45%	24	LVEF > 27%; LVEDD ≥ 5.5 cm	10.0
Sliwa, *et al.*[31]	2003-2005	100	LVEF< 40%	23	LVEF ≥ 30%, LVEDD	15.0
Amos, *et al.*[29]	1990-2003	55	LVEF< 45%	45	LVEDD > 5.6 cm; LVEF ≥ 30%; No LV thrombus	0.0
Modi, *et al.*[32]	1992-2003	44	LVEF< 45%	35	LVEF ≥ 30%	15.9
Sliwa, *et al.*[33]	2005-2006	80	LVEF< 30%	--	LVEF ≥ 30%	10.0
Goland, *et al.* [34]	2009-2011	182	LVEF< 45%	49	LVEF ≥ 25%	7.1
Hasan, *et al.* [35]	2003-2007	32	LVEF< 45%	63	--	9.3
Safirstein,*et al.* [36]	2005-2007	55	LVEF< 45%	--	LVEF ≥ 35%; gHTN, breastfeeding; postpartum diagnosis	--
Current study	2017-2019	**24**	LVEF<45%	--	LVEF >40%	**2**

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Author Profile

Dr. Rujuta Fuke, Associate professor, Department of Obstetrics and Gynaecology Government college and hospital, Nagpur

Dr. Smita Rokade, Post graduate student in, Department of Obstetrics and Gynaecology Government college and hospital, Nagpur

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