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**Original Research Article**

# A prospective study on possible clinical correlation between peripartum cardiomyopathy and hypertensive disorder in pregnancy

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## ABSTRACT

**Background:** Peripartum cardiomyopathy (PPCM) and hypertensive disorder in pregnancy (HDP) are often seen together. Though their correlation is a matter of research.
**Methods:** A prospective study of 76 patients of HDP in Apollo hospital Bilaspur (C. G.) over a period of one year February 2024 to February 2025 correlation with echocardiography.
**Results:** The maximum number of people with compromised left ventricular ejection fraction (LVEF) function is 28 years. Similarly in our study most of the patients are multigravida which is 22.37%. Maximum patients are of 3rd gravida.
**Conclusions:** Although the majority of HDP women were having normal LVEF function with no signs of PPCM but HDP was associated with markedly increased risks of PPCM that increased with HDP severity. HDP in association with increasing parity and age is also one of the contributing risk factors for PPCM. HDP, severe preeclampsia in particular, probably represents an additional cardiac stress during pregnancy.

**Keywords:** Peripartum, Cardiomyopathy, Hypertensive disorder in pregnancy, Left ventricle ejection fraction

## INTRODUCTION

Peripartum cardiomyopathy (PPCM) is similar to other forms of non ischemic dilated cardiomyopathy.[1] PPCM is development of cardiac failure during the last month of pregnancy or within 5 months of delivery in absence of any identifiable cause for cardiac failure, and absence of recognizable heart disease prior to last month of pregnancy, with LVEF less than 45%.[2]

The European society of cardiology revise these criteria in 2010, stating that PPCM is an idiopathic cardiomyopathy presenting with heart failure, secondary to left ventricular systolic dysfunction towards the end of pregnancy or in the months following delivery where no other cause of heart failure is found. The left ventricle may not be dilated but the LVEF is nearly always <45%.[3]

There are common overlap with respect to factors that increases the risk of developing preeclampsia and PPCM. Both have a higher prevalence among women with prior history of the disease, multiple gestation, diabetes, obesity, and extremes of reproductive age.[4]

This overlap of risk factors suggests that there may be a pathophysiological relationship between the two disorders. According to molecular study, there may be a connection between the two entities that is inflammation and angiogenesis. Elevated circulating sFlt1 (soluble FMS-like tyrosine kinase-1) levels in preeclampsia patients, supporting the hypothesis that these anti-angiogenic molecules may contribute to myocardial dysfunction.[5] In addition, increased levels of sFlt-1/PIGF (placental growth factor) may be predictive of preeclampsia, reflecting an anti-angiogenic factor. Similarly, mouse studies implicate an anti-angiogenic process in the pathogenesis of PPCM.[6] Given the anti-angiogenic milieu that has been associated with both preeclampsia and PPCM, the two disorders may be interrelated. A recent case-control study, however, demonstrated that a low sFlt-1/PIGF ratio was linked to

PPCM, which seems to contradict the hypothesis that preeclampsia and PPCM share a similar anti-angiogenic pathophysiology. A shortcoming of this study was that sFlt-1and PIGF levels were measured post-partum after the nidus of potential anti-angiogenesis, the placenta, had been removed. The IPAC investigators demonstrated elevated levels of both sFlt1 and prolactin in 100 gravidas presenting with PPCM, with worse NYHA functional classes associated with elevated sflt1.[7]

***Cardiovascular changes in pregnancy induced hypertension***

The normal LVEF is between 55 to 70 percent. In HDP there is increase in systemic vascular resistant, which causes increase in cardiac output. The most commonly observed heart changes elevated output along with a hyperenic left ventricle afterwards subsequent decrease in cardiac output and hypertrophy of the left ventricular wall. Some studies suggest that patient with hypertensive disorders of pregnancy usually have smaller left ventricular and diastolic and systolic dimension due to left ventricular hypertrophy.[8]

## METHODS

This is a prospective study, a longitudinal hospital-based correlation study of patients of Apollo hospital Bilaspur, over a period of one year from February 2024 to February 2025 of 76 patients, presenting with pregnancy induced hypertension in last three months of pregnancy and fifth month postpartum.

Sample size calculation is done by Cochran's formula with population size being 94 (according to the average OPD visit of hypertensive disorder pregnancy patient), population proportion 50%, margin error 5% and confidence level 95%.

Final population: $n' = \frac{n}{1+\frac{Z^2 \times \hat{p}(1-\hat{p})}{\varepsilon^2 N}}$

z is the z score, ε is the margin of error, N is the population size and $\hat{p}$ is the population proportion

***Inclusion criteria***

All patients of hypertensive disorders of pregnancy in age group of 20 to 45 years at third trimester and 5 months of delivery were included in the study.

***Exclusion criteria***

Patients with prior history of cardiac disease and people refusing consent for echocardiography were from the study excluded.

Statistical analysis by Microsoft excel 2013 statistical package SPSS version 20.

After taking consent patients, at third trimester or within 5 months of delivery had undergone echocardiography for monitoring LVEF.

## RESULTS

The normal LVEF is 55% to 70%. The maximum number of people with compromised LVEF function is 26-30 years. Out of 76 patients 53 patients had normal LVEF function. Most of the patients were identified in last months of pregnancy near term or first month post-partum presenting with chronic cough and shortness of breath.

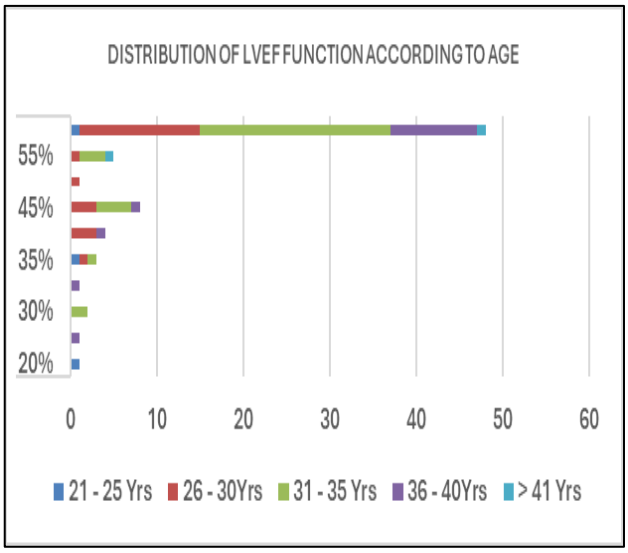


**Figure 1: Distribution of LVEF according to age.**

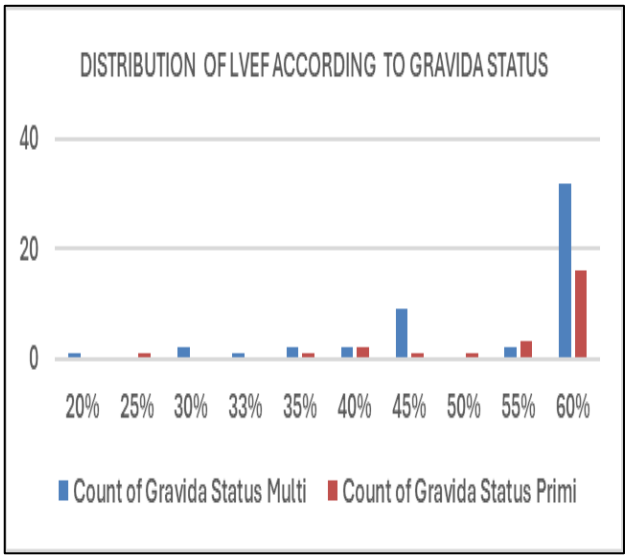


**Figure 2: Distribution of LVEF according to gravida status.**

One of the common risk factors for cardiomyopathy in patients with HDP is multigravida. Similarly in our study most of the patients are multigravida which is 22.37%. Primigravida being 9.21%.

**Table 1: Distribution of LVEF according to order of gravida.**

| Count of LVEF % / No. of gravida | 20% | 30% | 33% | 35% | 40% | 45% | 55% | 60% | Grand total |
|---|---|---|---|---|---|---|---|---|---|
| **2** | 1 | | | | 1 | 6 | 1 | 20 | 29 |
| **3** | | 2 | 1 | 2 | | 2 | 1 | 7 | 15 |
| **4** | | | | | | 1 | | 1 | 2 |
| **5** | | | | | 1 | | | 4 | 5 |
| **Grand total** | 1 | 2 | 1 | 2 | 2 | 9 | 2 | 32 | 51 |

Another risk factor for PPCM in patients with hypertensive disorders in pregnancy is increasing parity. In our study maximum patients are of 3rd gravida. This could be due to increasing urbanization and literacy leading to decreased child bearing.

## DISCUSSION

In our study the maximum number of people with compromised LVEF function is 26-30 years. Ntusi et al found that the average age of PPCM is 31.5±7 years.[8] They compared the time of onset of symptoms, clinical profile (including electrocardiographic [ECG] and echocardiographic features) and outcome of patients with hypertensive heart failure of pregnancy (n=53; age 29.6±6.6 years) and PPCM (n=30; age 31.5±7.5 years).[8]

In our study only 2 patients presented with cardiac failure out of 76 patients while in Ntusi et al study 30% percent of PPCM patient developed heart failure in first week of delivery, 46.7% after first month.[8]

**Table 2: Study comparison of Behrens et al and present study.**

| Variables | Behrens et al[9] study (n=2078822) | Our study, (n=76) |
|---|---|---|
| **Pregnancy with PPCM** | 126 | 22 |
| **Pregnancy without PPCM** | 2078 696 | 54 |
| **Twin pregnancy without PPCM** | 34 852 | 1 |
| **Twin with PPCM** | 10 | 0 |
| **Primi with PPCM** | 57 | 5 |
| **Primi without PPCM** | 1 087 840 | 20 |
| **Multigravida with PPCM** | 69 | 17 |
| **Multigravida without PPCM** | 990856 | 34 |
| **Age<30 years with PPCM** | 44 | 9 |
| **Age>30 years with PPCM** | 82 | 13 |

In our study 23 patients of PPCM were having hypertensive patients of pregnancy and were diagnosed in last month of pregnancy or first month postpartum. As quoted in the cohort of Behrens et al out of 126 women of PPCM, 39 (31.0%) in connection with an HDP-complicated pregnancy and 87 in connection with a normotensive pregnancy. Most PPCMs were diagnosed in the last month of pregnancy or the first month postpartum, both in women with (n=32, 82.1%) and without (n=71, 81.6%) HDP in the affected pregnancy.[9]

## CONCLUSION

Although the majority of HDP women were having normal LVEF function with no signs of PPCM but HDP was associated with markedly increased risks of PPCM that increased with HDP severity. HDP in association with increasing parity and age is also one of the contributing risk factors for PPCM. HDP, severe preeclampsia in particular, probably represents an additional cardiac stress during pregnancy.

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