

## Echocardiography Evaluation of the Left Ventricular Mass Index (LVMI) and Geometrical Pattern in Pre-Eclampsia and Pregnancy-Induced Hypertension Patients

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### Abstract:

**Objective:** The objective of this study was to evaluate the degree of hypertrophy and its geometrical patterns in hypertensive (HTN) pregnant women, with or without proteinuria.

**Material and Methods:** Data from 88 pregnant women was analyzed., consecutively. Both clinical and echocardiographic data between normotensive and hypertensive women, with and without proteinuria, were compared.

**Results:** Hypertensive women, with and without proteinuria, have a higher LV mass indexed by height than normotensive women (108.1±64.6 g/m vs. 76.5±24.8 g/m vs. 68.7±17.1 g/m, p-value=0.001).

**Conclusion:** This research shows that women that suffer from pre-eclampsia and pregnancy-induced hypertension show significant subclinical remodelling of the left ventricle, encompassing structural and functional changes, compared to those with normotensive pregnant women.

**Keywords:** Lv function, Lv geometrical patterns, Lv mass index, pregnant women, proteinuria

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## Introduction

During the course of pregnancy, there are certain hemodynamic changes that occur in a woman's body. These changes include a gradual rise in cardiac output and a corresponding decline in systemic vascular resistance, which ultimately leads to a high-volume, low-resistance circulation. The peak of these changes is observed during the mid-third trimester, after which the cardiac output declines and the systemic vascular resistance increases towards the 40<sup>th</sup> week of gestation<sup>1,2</sup>. Studies using echocardiography to assess uncomplicated, normal pregnancies have demonstrated a notable rise in the left ventricular mass and remodeling, along with associated diastolic dysfunction, in a limited but significant percentage of women at term. However, these alterations revert to normal postpartum<sup>3,4</sup>.

Hypertension during pregnancy can be linked to several conditions. Preeclampsia-eclampsia is a pregnancy-specific syndrome that typically manifests after 20 weeks of gestation. It is characterized by an increase in blood pressure accompanied by proteinuria. Gestational blood pressure elevation, or Pregnancy-induced hypertension, is defined as a systolic blood pressure of over 140 mm Hg or a diastolic blood pressure of over 90 mm Hg in a woman that was normotensive before 20 weeks of gestation<sup>5</sup>. In addition, Pregnancy-induced hypertension and pre-eclampsia have a notable, immediate effect on the heart's structure and function, leading to visible alterations in the ventricles, decreased contractility, and diastolic dysfunction<sup>6</sup>. The adverse maternal and fetal outcomes associated with severe and early-onset preeclampsia are closely linked to reduced stroke volume, diastolic dysfunction, and left ventricular remodeling. These findings reveal that normal pregnancy places a significant strain on the maternal cardiovascular system, and worsening cardiovascular maladaptation may

lead to the recognized clinical phenotype of preeclampsia<sup>7</sup>. The current literature on hypertensive pregnancy primarily focuses on gestational hypertension, with limited data available on ventricular remodeling and functional changes. This study aims to evaluate morphofunctional cardiac abnormalities in hypertensive pregnant women with and without proteinuria, using 2D and Doppler echocardiography.

## Material and Methods

### Study design and setting

This study employed a case-control design and encompassed three distinct groups: preeclampsia, pregnancy-induced hypertension, and control groups. The research was conducted on patients referred for singleton pregnancy at the Private Medical College and Hospital in Chennai from November 1<sup>st</sup>, 2022, to the end of April 2023.

### Population and sample

This research study targeted a specific population of pregnant patients with singleton pregnancies. A total of 88 participants were recruited, adhering to the following inclusion criteria: 1) women over the age of 18 who were recruited from the outpatient clinic, and 2) those with singleton pregnancies between 28 and 41 weeks, calculated from the first day of their last menstrual cycle, and confirmed by early ultrasonography during a routine prenatal visit. Exclusion criteria consisted of 1) those with preexisting cardiovascular, pulmonary, or renal disease, gestational or pregestational diabetes mellitus, or congenital fetal malformation.

### Ethics approval and informed consent

Study approval was granted by the Institutional Ethical Committee (IEC/ACSMCH): reference number 708/IEC/ACSMCH. Participants informed consent was obtained before the commencement of the study.

**Data collection:**

The severity of PE and gestational hypertension was determined by a specialist in maternal–fetal medicine, using guidelines from the National High Blood Pressure Education Program. This study comprised of 23 Hypertensive pregnant women with proteinuria in Group–3 and 27 with no proteinuria in Group–2, compared with 38 healthy pregnant women in Group–1.

As per standard clinical protocols, A comprehensive clinical evaluation was conducted, which included measuring the height, weight, systolic and diastolic blood pressure, heart rate, and Body Mass Index (BMI). The patient's systolic and diastolic blood pressure was measured using the brachial artery in a seated position, and the data was recorded before proceeding with the echocardiogram. Additionally, information regarding the subject's menstrual status, parity, and smoking history was acquired through a questionnaire.

**Echocardiographic measurements:**

According to current practice guidelines, standard two–dimensional and Doppler echocardiography was performed using a 3.5–MHz transducer (commercially available equipment) in the left decubitus position. Accurate measurements of LV wall thickness, LVEDD, and LVESD were obtained through M–mode recording and two–dimensional guidance from parasternal long and short–axis views to effectively evaluate the size of the left ventricle (LV). The LV mass was determined by utilizing the formula developed by Devereux et al.<sup>8</sup> The relative wall thickness (RWT) was calculated using the formula ( $2 \times$  posterior wall thickness) divided by LVEDD.

Furthermore, the LV mass index (LVMI) was calculated by normalizing the LV mass by height and BSA. LV geometry classification was determined using the LVMI value calculated with BSA. Normal LV geometry was

defined as LVMI value  $\leq 95 \text{ g/m}^2$  and RWT value  $\leq 0.42$ . Concentric remodelling was classified if LVMI value  $\leq 95 \text{ g/m}^2$  and RWT value  $> 0.42$ . LV geometry was classified as concentric hypertrophy if LVMI value  $> 95 \text{ g/m}^2$  and RWT value  $> 0.42$ , while eccentric hypertrophy was classified if LVMI value  $> 95 \text{ g/m}^2$  and RWT value  $\leq 0.42$ .

The LV EF was calculated using a modified Simpson's method based on the apical 4–chamber and 2–chamber views. Traditionally, diastolic function evaluation through echocardiography involves measuring trans–mitral flow parameters. This includes calculating the E/A ratio from an apical four–chamber view using conventional pulsed wave Doppler. The ratio compares the peak velocity of blood flow during the early diastolic phase (E wave) to the peak velocity during late diastole caused by atrial contraction (A wave). The E/A ratio is an indicator of left ventricular diastolic function.

**Results**

A total of 88 pregnant women were included in the final analysis. Moreover, the participants were categorized into three groups: normotensive (group–I), hypertensive pregnant women with (group–III) and without proteinuria (group–II); 38 women were normotensive, 27 were hypertensive pregnant women without proteinuria (PIH), and 23 were with proteinuria (PE) (Table 1). The mean gestational age in group–I was  $34.18 \pm 5.11$ , group II was  $35.44 \pm 4.31$ , and group–III was  $33.57 \pm 4.95$  weeks. The groups did not differ significantly regarding maternal age, parity, or BMI. The mean BP of group III was  $122.2 \pm 7.0$  mm Hg, which was significantly higher than group II and group I ( $118 \pm 14$  mm Hg and  $86.5 \pm 7.6$ ) ( $p$ -value  $< 0.001$ ), respectively. Compared to G1 and G2, G3 had considerably increased interventricular septal wall thickness during diastole (with  $p$ -value of less than 0.01 for all).

Hypertensive women with and without proteinuria have a higher LV mass indexed by height than normotensive women ( $108.1 \pm 64.6$  g/m vs.  $76.5 \pm 24.8$  g/m vs.  $68.7 \pm 17.1$  g/m,  $p$ -value=0.001). Additionally, hypertensive women have impaired diastolic function, as measured by the peak velocity ratio of early diastolic transmitral blood flow to early diastolic mitral annular velocity ( $1.22 \pm 0.42$  vs.  $1.17 \pm 0.38$  vs.  $1.17 \pm 0.38$ ). This change was more noticeable in hypertensive pregnant women without proteinuria (PIH) than those with proteinuria (PE) and normotensive pregnant women (Table 2). In Group-3, 13.0% of patients had eccentric hypertrophy, and 11.0% of patients in Group-2 had eccentric hypertrophy; as estimated from LVM and RWT (Figure 1).

**Table 1** Baseline characteristics

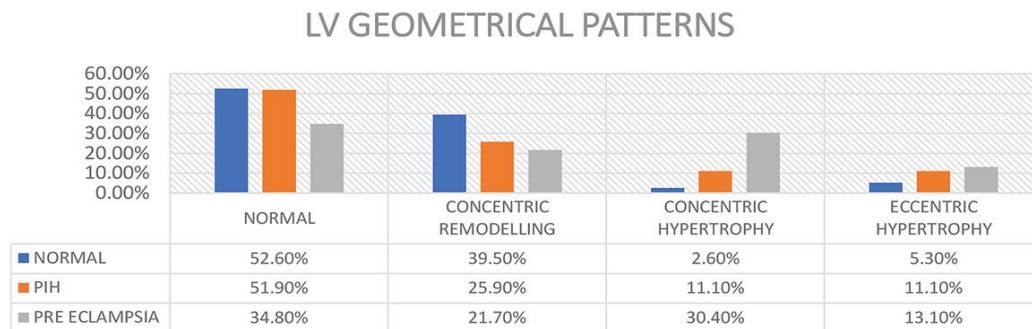
| Variable         | Normal (n=38)<br>(Group-I) | PIH (n=27)<br>(Group-II) | Pre-eclampsia (n=23)<br>(Group-III) | p-value |
|------------------|----------------------------|--------------------------|-------------------------------------|---------|
| Age (years)      | 27.2±4.8                   | 29.6±7.6                 | 30.7±6.1                            | 0.08    |
| PRIMI            | 22 (57.9%)                 | 16 (59.3%)               | 12 (52.2%)                          | 0.86    |
| Gestational week | 34.18±5.11                 | 35.44±4.31               | 33.57±4.95                          | 0.36    |
| SBP              | 115.3±11.4                 | 138.1±11.7               | 157.3±7.5                           | 0.00    |
| DBP              | 72.1±6.9                   | 84.4±7.5                 | 104.7±7.2                           | 0.00    |
| Mean BP (mmHg)   | 86.5±7.6                   | 102.4±7.6                | 122.2±7.0                           | 0.00    |
| BSA              | 1.9±0.27                   | 1.8±0.36                 | 1.7±0.42                            | 0.27    |

PRIMI=primigravida, SBP=systolic blood pressure, DBP=diastolic blood pressure, BSA=body surface area, PIH=pregnancy induced hypertension

**Table 2** Echocardiographic parameters of the studied groups

| Variables                | Normal (n=38)<br>(Group-I) | PIH (n=27)<br>(Group-II) | Pre-eclampsia (n=23)<br>(Group-III) | p-value |
|--------------------------|----------------------------|--------------------------|-------------------------------------|---------|
| LVIDd (cm)               | 42.4±4.99                  | 42.9±5.39                | 43.8±5.11                           | 0.56    |
| IVSd (cm)                | 0.99±0.32                  | 1±0.0                    | 1.3±0.77                            | 0.00    |
| PWD (cm)                 | 0.99±0.03                  | 1.0±0.00                 | 0.95±0.20                           | 0.30    |
| EF%                      | 67.1±3.42                  | 65.7±4.94                | 63.3±2.77                           | 0.00    |
| LVM                      | 118.0±24.47                | 123.3±35.10              | 183±121.13                          | 0.00    |
| LVMI (g/m <sup>2</sup> ) | 68.7±17.19                 | 76.5±24.85               | 108.1±64.63                         | 0.00    |
| E/A                      | 1.17±0.37                  | 1.22±0.42                | 1.17±0.38                           | 0.80    |

LVIDd=left ventricular internal dimension at end diastole, IVSd=interventricular septal end diastole, PWD=left ventricular posterior wall thickness at end diastole, EF=ejection fraction, LVM=left ventricular mass, LVMI=left ventricular mass index, E=early diastolic mitral inflow velocity, A=mitral peak velocity of late filling



**Figure 1** Geometrical patterns

## Discussion

During pregnancy, the human body is subject to several changes in blood flow. These changes include increased blood and stroke volumes and heart rate. Furthermore, peripheral resistance and mean arterial pressure tend to decrease. As a result of such modifications, the woman’s heart undergoes structural remodeling throughout pregnancy; including an increase in end-diastolic volume and functional changes in the left ventricle after the first half of the pregnancy<sup>9,10</sup>. Pregnancy-induced hypertension is a rapidly developing condition that affects a previously healthy cardiovascular system and lasts for a short duration. Due to the sudden onset, the body does not have enough time to adapt to the increased pressure<sup>11</sup>. Published studies on left ventricular structure and function in PIH lack consensus. While Sanchez et al<sup>12</sup>. and Thompson et al<sup>13</sup>. failed to observe significant changes in left ventricular mass, Vazquez Blanco et al<sup>14</sup>. found evidence of increased left ventricular mass and RWT, along with modified geometric patterns of the left ventricle, in PIH patients. The present research indicates a remarkable rise in the left ventricular mass of individuals with PIH, with particular emphasis on the thickness of the septal wall. The assumption is that the left ventricular mass was within the normal range before pregnancy, as there was no history of hypertension.

Pre-eclampsia, commonly referred to as PE, is a severe condition that can negatively impact the mother and fetus. It leads to significant hemodynamic changes in the cardiovascular system. This study’s findings indicate that LV eccentric hypertrophy with a higher LVMI is more prevalent among PE cases, with a greater degree of LV remodeling observed in women with PE. The observed alterations in LV shape are undoubtedly a consequence of the afterload-triggered compensatory mechanism, leading to a shift from an ellipsoid configuration to a more spherical one<sup>15</sup>. Analysis, using mean±2 standard deviations, established that normotensive pregnant women’s upper normal limits for LVMI are 103.08 g/m<sup>2</sup>, respectively. These findings differ slightly from Ganau et al<sup>16</sup>. 106 g/m<sup>2</sup> for left ventricular mass. Meanwhile, Soh et al<sup>17</sup>. and Park’s studies<sup>18</sup>. On Korean populations have identified upper limits of 100.5 and 103.6 g/m<sup>2</sup>, respectively. These slight variations in results could be due to population differences, which should be taken into consideration.

## Conclusion

To our knowledge, this present investigation is the first to evaluate the left ventricular mass and ventricular geometrical patterns in hypertensive pregnant women, with or without proteinuria, in the Indian population. This research shows that women who suffer from pre-eclampsia

and pregnancy-induced hypertension show significant subclinical remodelling of the left ventricle, encompassing structural and functional changes, compared to those with normotensive pregnant women.

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## Conflict of interest

All authors have no conflicts of interest to disclose.

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