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# Effect of Blood Pressure Variability on Right Ventricular Remodelling in Pregnant Women with Hypertensive disorders of pregnancy

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***Abstract***
*Background: Hypertensive disorders of pregnancy increase maternal and cardiovascular risks, impacting heart remodelling and function. Blood pressure variability can alter heart structure, yet clinical impact remains underexplored. The aim of this study was to evaluate potential impact of blood pressure variability on right ventricular remodeling in pregnant women with hypertensive disorders of pregnancy. Methods: This observational comparative study included 17 patients with preeclampsia, 23 with gestational hypertension and 20 healthy pregnant women at Aswan University hospital. Results: After three months, RV parameters remained significantly elevated in both hypertensive groups (P<0.0001), while RV strain parameters showed improvement but remained significantly lower (P<0.0001). Mean systolic and diastolic blood pressures were significantly correlated with RV dimensions and strain parameters in preeclampsia and gestational hypertension, with negative correlations observed for TAPSE and S'. In controls, mean diastolic blood pressure showed a significant negative correlation with S' (P=0.044). Maternal complications, including eclampsia (P=0.0184), preterm labor (P=0.015), and postpartum hemorrhage (P=0.0071), were significantly higher in preeclampsia. Conclusions: BP variability significantly affects RV remodeling in pregnant women with Pregnancy related hypertensive disorders. The persistence of elevated BP and RV abnormalities highlights the critical need for continued cardiovascular monitoring and management.*
***Keywords***: *Pre-Eclampsia, Gestational Hypertension, Right ventricle, remodeling, hypertensive disorder.*

## INTRODUCTION

Hypertensive disorders of pregnancy (HDP), encompassing preexisting hypertension, gestational hypertension (GH), preeclampsia (PE), and eclampsia, affect up to 10% of pregnancies and constitute a major contributor to maternal and perinatal morbidity and mortality. HDP, including pregnancy-induced hypertension, preeclampsia, and HELLP syndrome (characterized by hemolysis, elevated liver enzymes, and low platelet count), are associated with approximately 10% of gestations and represent a significant cause of maternal mortality. Preeclampsia is a multifactorial pregnancy-related disorder defined by endothelial dysfunction, systemic hypertension, and multi-organ hypoperfusion, with a reported incidence of 3–4% in Western populations **(1).**

The presence of any HDP is a recognized indicator of increased long-term cardiovascular (CV) disease risk, including cerebrovascular accidents, atherosclerotic cardiovascular events, atrial fibrillation, heart failure, and cardiovascular mortality. The risk of adverse CV outcomes is dose-dependent, with the highest risk observed in early-onset and preterm preeclampsia. Established risk factors for heart failure, such as hypertension, coronary artery disease (CAD), myocardial infarction, obesity, atrial fibrillation, and chronic kidney disease, are prevalent in individuals with a history of HDP **(2).**

Pregnancy induces profound cardiovascular adaptations to meet the metabolic demands of the mother and the developing fetus. These adaptations include increased blood volume, decreased peripheral vascular resistance (PVR), augmented placental growth, increased heart rate, and elevated cardiac output. Cardiac remodeling, including mild dilation of all cardiac chambers and increased left ventricular (LV) mass, serves as a compensatory mechanism for these hemodynamic changes **(3).** Despite increased circulating levels of renin and angiotensin II, PVR remains low during normal pregnancy, possibly due to the influence of humoral factors such as prostaglandins and progesterone. In contrast, pregnancies complicated by hypertension may exhibit aberrant pressure overload, leading to distinct patterns of cardiac remodeling compared to normotensive pregnancies **(4).**

Research has extensively characterized LV remodeling in HDP, particularly in Pregnancy related hypertensive disorders, over the past few decades. However, data on right ventricular (RV) structural, functional, and mechanical changes remain limited and inconclusive. While some studies report significant RV alterations, including increased RV diameter, elevated pulmonary pressures, and reduced RV longitudinal strain, other investigations find no significant differences in RV structure and function between normotensive pregnant women and those with HDP **(5)**

Although blood pressure (BP) measurement is a cornerstone in the diagnosis and management of hypertension and heart failure, the potential clinical relevance of blood pressure variability (BPV), either independently or in conjunction with heart rate variability (HRV), remains insufficiently explored. This gap in knowledge is primarily due to the lack of accessible, wearable, continuous BP monitoring devices. BPV is defined as fluctuations in arterial BP over a specified timeframe **(6).** The clinical implications of BPV are not yet fully elucidated, but three key aspects merit consideration: BPV introduces variability in BP assessment, particularly with isolated clinic measurements; it may enhance cardiovascular risk stratification, although its independent prognostic value requires further substantiation; and targeting BPV through therapeutic interventions may improve clinical outcomes without necessarily increasing healthcare expenditures **(7, 8).**

So, in this study we aimed to evaluate potential impact of blood pressure variability on right ventricular remodeling in pregnant women with hypertensive disorders of pregnancy.

**Patients and Methods:**

In this observational comparative study, we enrolled sixty pregnant women > 18 years old after 20 weeks of gestation collected from those admitted to labour and delivery unit or during a routine prenatal visit at Aswan university Hospital obstetrics and Gynaecology clinic and we divided them into two groups:

**Group A** (n=40): with pregnancy related hypertensive disorders and **group B** (n=20): with normal pregnancy serve as control group.

**We ruled out** patients with gestation period less than 20 weeks, congenital heart diseases, moderate to severe valvular heart diseases, cardiomyopathy whatever its cause, underlying RV dysfunction and poor image quality.

**Procedure:** All patients were subjected to complete history taking, clinical examination, routine laboratory investigations and ECG or any further research that is necessary based on the patients' clinical situation.

- **Office BP measurements:**

  In each visit systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured 2 to 3 times, taking the average of the last 2 readings, the visit-to-visit variability was determined by standard deviation (SD) or coefficient of variation (CV) of either systolic or diastolic BP from baseline, then 3 months post-partum to evaluate BP variability.

- **Non-invasive Imaging:**

**Two-dimensional transthoracic echocardiography (9):**

It was performed during pregnancy after 20 weeks of gestation and at 3 months post-partum follows up to evaluate the following parameters:

- RV Basal, mid, and longitudinal diameters.
- RV function by TAPSE and S Velocity methods.

**Speckle tracking (STE) analysis of RV:**

The examination began by positioning the patient in the left lateral decubitus position and the probe was moved across his chest to determine the RV strain (10). Image acquisition was performed using a right ventricular (RV)-focused view in the apical four-chamber (A4C) orientation to ensure optimal visualization of the RV free wall, RV apex, and tricuspid valve/annulus throughout both systolic and diastolic phases. The imaging depth was set to an intermediate level, avoiding excessive anterior tilt (wherein the left ventricular outflow tract [LVOT] would become visible) or posterior tilt (which would reveal the coronary sinus). Strain measurements were generated automatically by dedicated software with color-coding functionality. Image quality was evaluated to ensure accurate myocardial tracking, and manual adjustments were implemented as needed. Each segment's region of interest (ROI) was meticulously assessed for accurate alignment along the RV-free wall and interventricular septum. ROI

thickness was standardized at 5 mm for nonhypertrophied RV-free walls and was proportionally increased in the presence of RV hypertrophy. The outer contour placement prioritized minimizing pericardial inclusion, avoiding pericardial tracking entirely or reducing it to the lowest feasible extent. The inner contour was positioned precisely along the endocardial border, with careful exclusion of trabeculations and papillary muscles. The tracking boundary terminated at the tricuspid annulus, ensuring no extension into the right atrium (RA) or into the RV beyond the annular plane. RV global longitudinal strain (RV GLS) encompassed both the RV-free wall and the ventricular septum, with a normal reference range of 20%–25%. RV-free wall strain (RV FWS) analysis, which excluded the ventricular septum, was considered within a normal range of 23%–33%.

**Statistical analysis**

Statistical analysis was performed using SPSS version 26 (IBM Inc., Chicago, IL, USA). Quantitative data were expressed as mean ± standard deviation (SD) and compared between groups using the unpaired Student's t-test. Categorical variables were presented as frequencies and percentages (%) and analyzed using the Chi-square test or Fisher's exact test when applicable. Pearson's correlation coefficient was used to assess correlations between variables. P-value of <0.05 was considered statistically significant.

**Ethical Consideration:**

The Medical Ethic Committee of Aswan University's Faculty of Medicine granted IRB permission. Clinical trial.gov was used to prospectively register the study Clinical trial.gov ID:NCT06100484) . The study was conducted following the principles outlined in the Helsinki Declaration (11) and in accordance with CONSORT checklist for research ethics (12). Prior to the start of the study, the title and goal of the study were fully explained and informed consent from each patient was acquired. All information gathered was kept private and utilized exclusively for scientific study. Each research participant was free to leave the study at any moment without affecting the quality of the medical care they received.

## RESULTS

This observational study was performed on 60 pregnant women after 20 weeks of pregnancy and divided into two groups.

**Table (1), Figures (1-3):**

BMI was significantly higher in PE (32.25 ± 2.71) than in controls (28.9 ± 3.7, P=0.0143). Antihypertensive therapy use was significantly higher in PE (58.82%) and GH (43.48%) compared to controls (0%, P=0.0003).

Systolic and diastolic blood pressures were significantly higher in PE (152.69 ± 3.47 mmHg, 92.24 ± 2.3 mmHg) and GH (150.87 ± 3.5 mmHg, 91.9 ± 1.72 mmHg) compared to controls (120 ± 1.80 mmHg, 75 ± 3.4 mmHg, both P<0.0001).

Right ventricular (RV) parameters were significantly higher in PE and GH compared to controls. RV basal (PE: 34.82 ± 7.12 mm, GH: 32.39 ± 5.69 mm, controls: 23.6 ± 2 mm), mid (PE: 33 ± 3.55 mm, GH: 31.43 ± 3.05 mm, controls: 26.2 ± 2 mm), and longitudinal diameters (PE: 76.53 ± 5.91 mm, GH: 74.78 ± 4.16 mm, controls: 62 ± 4.9 mm) were significantly higher (P<0.0001). TAPSE was significantly lower in PE (20.76 ± 3.26 mm) compared to controls (23.9 ± 1.9 mm, P=0.0047). The systolic velocity (s') was significantly lower in PE (11.91 ± 2.18 cm/s) compared to controls (14 ± 3.3 cm/s, P=0.0284). RAVI was significantly higher in PE (25.76 ± 3.64 ml/m²) compared to GH (22.78 ± 4.37 ml/m²) and controls (17.4 ± 3.1 ml/m², P<0.0001).

RV strain parameters were significantly lower in PE and GH. Global RV strain was lower in PE (-18.41 ± 1.94) and GH (-20.56 ± 1.79) compared to controls (-23.2 ± 2.0, P<0.0001). Free wall RV strain was also significantly lower in PE (-20.83 ± 2.01) and GH (-22.42 ± 2.21) compared to controls (-27.2 ± 2.7, P<0.0001).

**Table 1: Comparison of all studied groups regarding demographic, baseline laboratory data, mean systolic, diastolic Bl P, LV, RV parameters and RV strain and description of diagnosis in cases group**

| | Preeclampsia (N = 17) | Gestational hypertension (N = 23) | Controls (N = 20) | P. Value |
|---|---|---|---|---|
| Age (Years) | 29.71 ± 4.88 | 31.48 ± 5.25 | 32.8 ± 4.1 | 0.1659 [F] |
| | P1= 0.4957, P2= 0.1412, P3= 0.6508 | | | |
| BMI (Kg/m$^2$) | 32.25 ± 2.71 | 30.29 ± 3.79 | 28.9 ± 3.7 | **0.0194*** [F] |

| | | | | |
|---|---|---|---|---|
| | P1= 0.2013, P2= 0.0143*, P3= 0.3908 | | | |
| Parity | 2.53 ± 1.24 | 2.35 ± 1.17 | 2.1 ± 1.0 | 0.5318 [F] |
| | P1= 0.8774, P2= 0.507, P3= 0.7666 | | | |
| Gestational age (weeks) | 31.18 ± 3.73 | 32.65 ± 3.89 | 32.5 ± 4.6 | 0.5085 [F] |
| | P1= 0.5125, P2= 0.625, P3= 0.9862 | | | |
| Gestational diabetes (%) | 3 (17.65%) | 1 (4.35%) | 2 (10%) | 0.3827 [X] |
| Antihypertensive therapy (%) | 10 (58.82%) | 10 (43.48%) | 0 (0%) | **0.0003*** [f] |
| Systolic blood pressure (mmHg) | 152.69 ± 3.47 | 150.87 ± 3.5 | 120 ± 1.80 | **< 0.0001*** [F] |
| | P1= 0.1674, P2< 0.0001*, P3< 0.0001* | | | |
| Diastolic blood pressure (mmHg) | 92.24 ± 2.3 | 91.9 ± 1.72 | 75 ± 3.4 | **< 0.0001*** [F] |
| | P1= 0.9122, P2< 0.0001*, P3< 0.0001* | | | |
| RV parameter | | | | |
| RV basal diameter (mm) | 34.82 ± 7.12 | 32.39 ± 5.69 | 23.6 ± 2 | **<0.0001*** [F] |
| | P1= 0.3484, P2= <0.0001*, P3= <0.0001* | | | |
| RV mid diameter (mm) | 33 ± 3.55 | 31.43 ± 3.05 | 26.2 ± 2 | **<0.0001*** [F] |
| | P1= 0.2326, P2= <0.0001*, P3= <0.0001* | | | |
| RV longitudinal diameter (mm) | 76.53 ± 5.91 | 74.78 ± 4.16 | 62 ± 4.9 | **<0.0001*** [F] |
| | P1= 0.5272, P2= <0.0001*, P3= <0.0001* | | | |
| RV thickness (mm) | 3.37 ± 0.37 | 3.27 ± 0.49 | 3.00 ± 0.4 | 0.0644 [F] |
| | P1= 0.7492, P2= 0.0634, P3= 0.2046 | | | |
| TAPSE (mm) | 20.76 ± 3.26 | 22.13 ± 3.12 | 23.9 ± 1.9 | **0.0064*** [F] |
| | P1= 0.3073, P2= 0.0047*, P3= 0.1194 | | | |
| s'(cm/s) | 11.91 ± 2.18 | 11.8 ± 2.46 | 14 ± 3.3 | **0.0203*** [F] |
| | P1= 0.9925, P2= 0.0594, P3= 0.0284* | | | |
| RAVI (ml/m$^2$) | 25.76 ± 3.64 | 22.78 ± 4.37 | 17.4 ± 3.1 | **<0.0001*** [F] |
| | P1= 0.0477*, P2= <0.0001*, P3= 0.0001* | | | |
| RV strain | | | | |
| Global RV | -18.41 ± 1.94 | -20.56 ± 1.79 | -23.2 ± 2.0 | **<0.0001*** [F] |
| | P1= 0.003*, P2= <0.0001*, P3= 0.0002* | | | |
| Free wall RV strain (%) | -20.83 ± 2.01 | -22.42 ± 2.21 | -27.2 ± 2.7 | **<0.0001*** [F] |
| | P1= 0.1005, P2= <0.0001*, P3= <0.0001* | | | |

Data are presented as mean ± SD or frequency (%). * Significant P value <0.05. F: Anova t test, $X^2$: chi-square test, f: fisher exact test.

P1: Preeclampsia vs Gestational hypertension groups, P2: Preeclampsia vs controls and P3: Gestational hypertension vs controls.

BMI: body mass index, Hb: hemoglobin, Bl P: blood pressure, RV: right ventricular, TAPSE: tricuspid annular plane systolic excursion, RAVI: right atrial volume index, PE: preeclampsia.

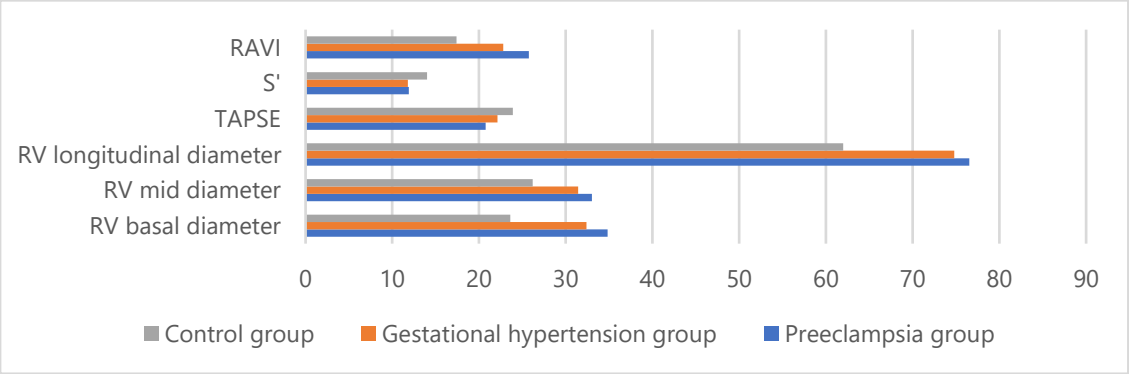


**Figure (1):** Comparison between the studied groups regarding baseline RV parameters.

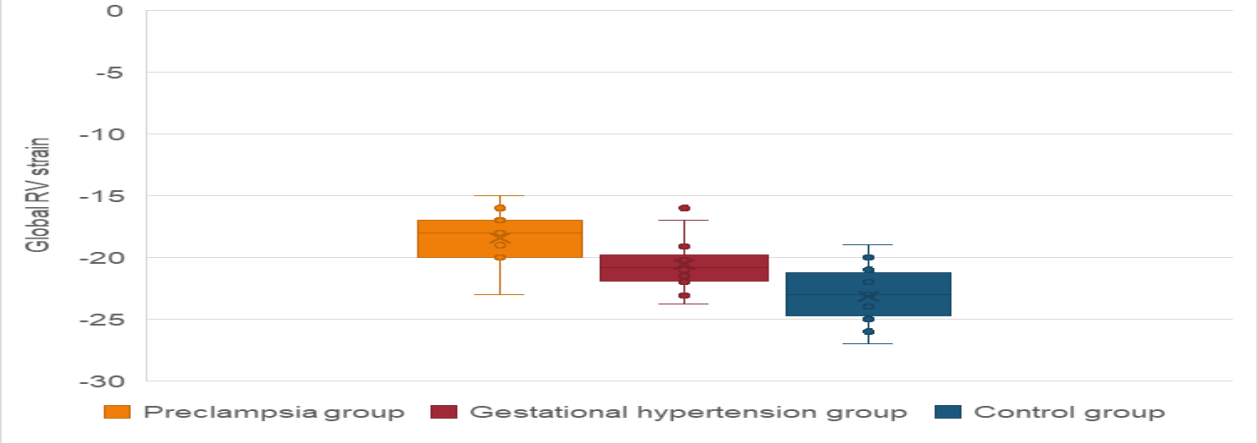


**Figure (2):** Comparison between the studied groups regarding baseline global RV strain.

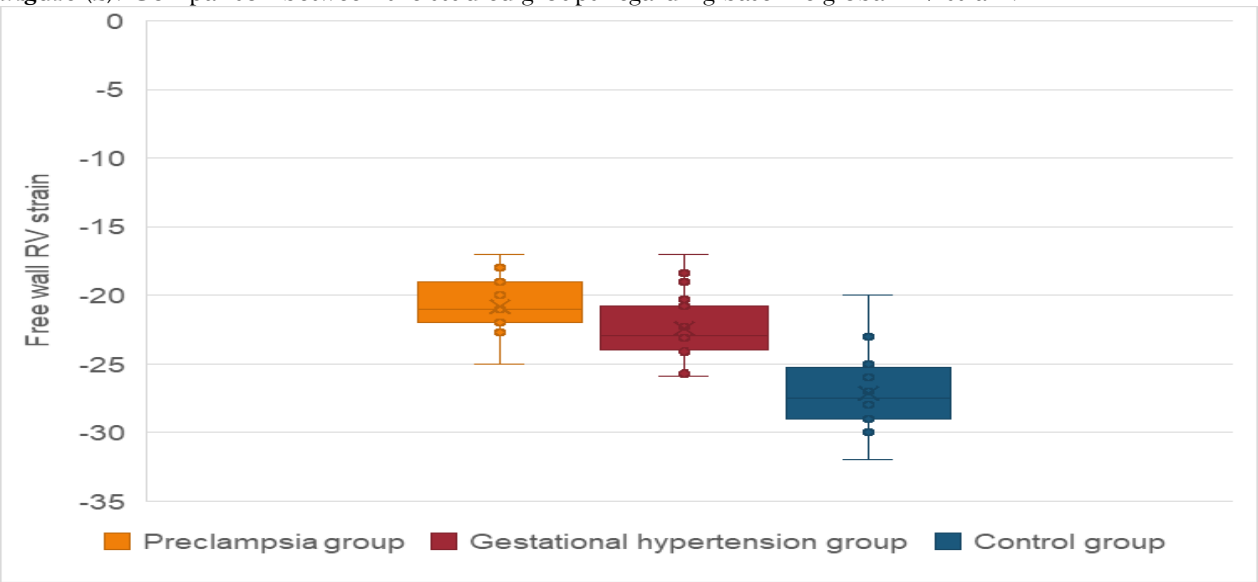


**Figure (3):** Comparison between the studied groups regarding baseline free wall RV strain.

**Table (2), Figure (4-6):**

Mean systolic blood pressure was higher in PE (128.96 ± 14.27 mmHg) and GH (122.57 ± 9.41 mmHg) than in controls (117 ± 2.3 mmHg, P<0.0001). Mean diastolic blood pressure was also higher in PE (79.22 ± 8.09 mmHg) and GH (76.56 ± 5.95 mmHg) compared to controls (71 ± 2.1 mmHg, P<0.0001).

Right ventricular (RV) parameters remained significantly higher in PE and GH groups compared to controls. RV basal diameter was greater in PE (32.12 ± 5.79 mm) and GH (30.22 ± 4.2 mm) than in controls (25 ± 1.9 mm, P<0.0001). RV mid diameter was also higher in PE (30.76 ± 3.62 mm) and GH (31.43 ± 3.47 mm) compared to controls (27.2 ± 2 mm, P=0.0001). RV longitudinal diameter remained elevated in PE (73.94 ± 5.4 mm) and GH (73.39 ± 4.12 mm) relative to controls (63.8 ± 4.1 mm, P<0.0001).

RV strain parameters showed significant improvement but remained significantly lower in PE and GH compared to controls. Global RV strain was lower in PE (-21.74 ± 2.45) and GH (-22.63 ± 2.11) than in controls (-25.3 ± 1.6, P<0.0001). Similarly, free wall RV strain was reduced in PE (-24.22 ± 2.52) and GH (-24.44 ± 2.28) compared to controls (-28.6 ± 2.3, P<0.0001).

**Table 2: Comparison of all studied groups regarding mean systolic and diastolic Bl P, RV parameters, RV strain and its delta after 3 months**

| | Preeclampsia (N = 17) | Gestational hypertension (N = 23) | Controls (N = 20) | P. Value |
|---|---|---|---|---|
| Mean Systolic blood pressure (mmHg) | 128.96 ± 14.27 | 122.57 ± 9.41 | 117±2.3 | **<0.0001*** [F] |
| | P1< 0.0001*, P2 < 0.0001*, P3= 0.01279* | | | |
| Mean Diastolic blood pressure (mmHg) | 79.22 ± 8.09 | 76.56 ± 5.95 | 71±2.1 | **<0.0001*** [F] |
| | P1= 0.34801, P2 = 0.00011*, P3= 0.0091* | | | |

| RV parameters | | | | |
|---|---|---|---|---|
| RV basal diameter (mm) | 32.12 ± 5.79 | 30.22 ± 4.2 | 25±1.9 | <0.0001* [F] |
| | P1= 0.3520, P2 < 0.0001*, P3= 0.0009* | | | |
| RV mid diameter (mm) | 30.76 ± 3.62 | 31.43 ± 3.47 | 27.2±2 | 0.0001* [F] |
| | P1= 0.7857, P2 = 0.002*, P3= 0.0009* | | | |
| RV longitudinal diameter (mm) | 73.94 ± 5.4 | 73.39 ± 4.12 | 63.8±4.1 | <0.0001* [F] |
| | P1= 0.9256, P2 < 0.0001*, P3< 0.0001* | | | |
| TAPSE (mm) | 22.59 ± 2.99 | 23.87 ± 2.31 | 24.2±1.6 | 0.107 [F] |
| | P1= 0.2170, P2 = 0.0931, P3 = 0.9006 | | | |
| s'(cm/s) | 12.22 ± 2.7 | 12.25 ± 1.98 | 14±3.2 | 0.0566 [F] |
| | P1= 0.9993, P2 = 0.0922, P3 = 0.0994 | | | |
| RV strain | | | | |
| Global RV | -21.74 ± 2.45 | -22.63 ± 2.11 | -25.3±1.6 | <0.0001* [F] |
| | P1= 0.3955, P2 < 0.0001*, P3 = 0.0005* | | | |
| Free wall RV strain (%) | -24.22 ± 2.52 | -24.44 ± 2.28 | -28.6±2.3 | <0.0001* [F] |
| | P1= 0.9546, P2 < 0.0001*, P3 < 0.0001* | | | |

Data are presented as mean ± SD.* Significant P value <0.05. F: Anova t test, Bl P: blood pressure, SD: standard deviation, RV: right ventricular, TAPSE: tricuspid annular plane systolic excursion.

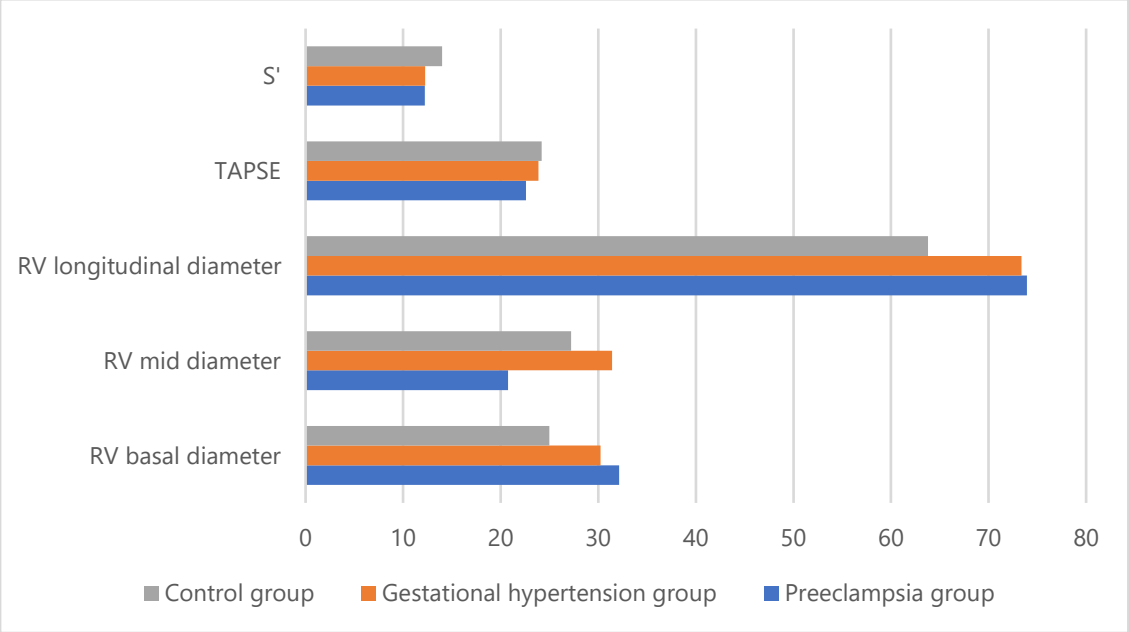


**Figure (4):** Comparison between the studied groups regarding RV parameters after 3 months.

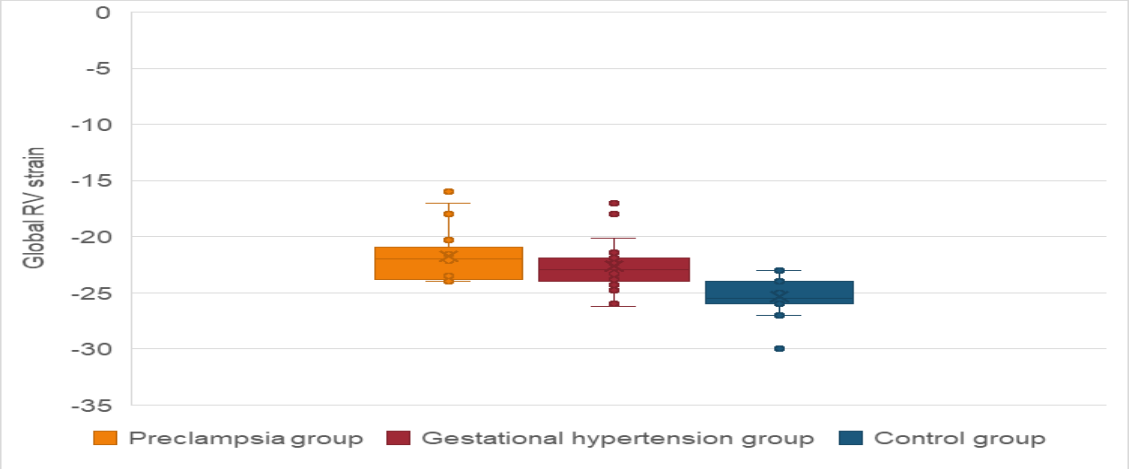


**Figure (5):** Comparison between the studied groups regarding global RV strain after 3 months.

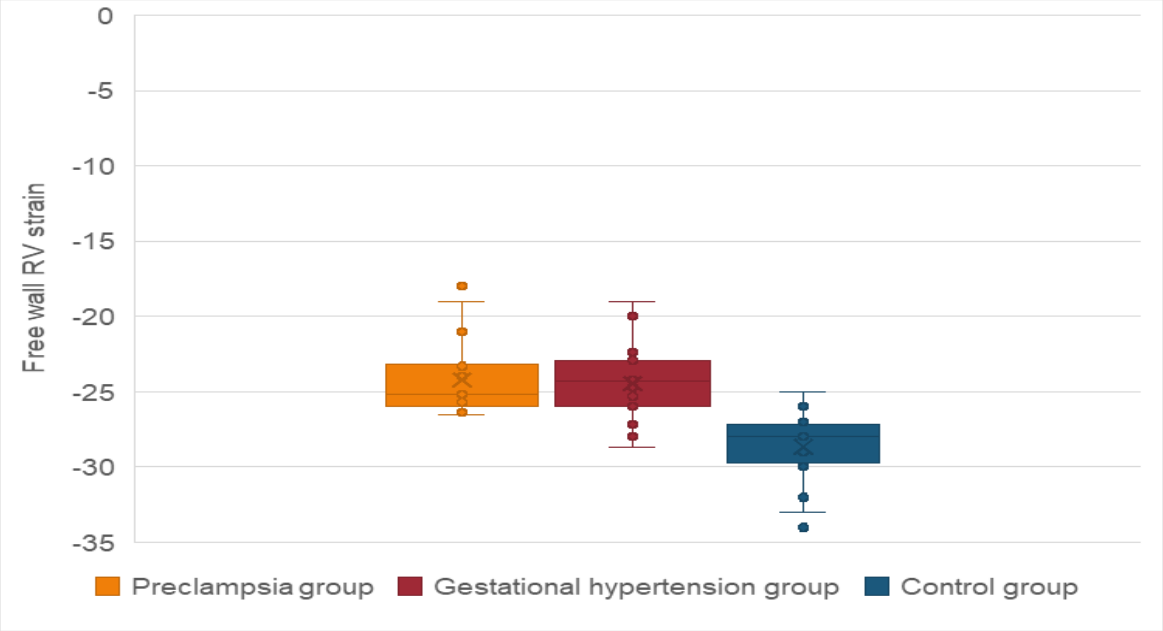


**Figure (6):** Comparison between the studied groups regarding global RV strain after 3 months.

**Table 3:**

**In the preeclampsia group:**

Mean systolic blood pressure showed significant positive correlations with RV basal diameter (r=0.568, P=0.017), RV mid diameter (r=0.688, P=0.002), RV longitudinal diameter (r=0.569, P=0.017), RV thickness (r=0.522, P=0.032), RAVI (r=0.662, P=0.004), global RV strain (r=0.682, P=0.003), and free wall RV strain (r=0.651, P=0.005).

Mean systolic blood pressure showed a negative correlation with TAPSE (r=-0.488, P=0.047).

Mean diastolic blood pressure showed significant positive correlations with RV basal diameter (r=0.523, P=0.031), RV thickness (r=0.542, P=0.025), global RV strain (r=0.562, P=0.019), and free wall RV strain (r=0.623, P=0.008).

**In gestational hypertension group:**

There was significant negative correlation between the mean systolic blood pressure an age (r = -0.572, P = 0.004).

**In the controls group:**

Mean systolic blood pressure showed significant negative correlations with age (r=-0.572, P=0.004), BMI (r=-0.547, P=0.012), and parity (r=-0.482, P=0.031).

**Table 3: Correlation between mean systolic and diastolic BP and other studied parameters in cases and control group**

| | Mean systolic Bl P | | Mean diastolic Bl P | | Mean systolic Bl P | | Mean diastolic Bl P | | Mean systolic Bl P | | Mean diastolic Bl P | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | R | P | R | P | R | P | R | P | r | P | r | P |
| | Preeclampsia group | | | | Gestational hypertension group | | | | Control group | | | |
| Age | 0.094 | 0.719 | 0.046 | 0.861 | -0.572 | **0.004*** | -0.368 | 0.084 | 0.078 | 0.744 | 0.202 | 0.394 |
| BMI | 0.257 | 0.318 | 0.306 | 0.233 | -0.045 | 0.839 | 0.375 | 0.078 | -0.002 | 0.993 | -0.547 | **0.012*** |
| Parity | -0.193 | 0.457 | -0.395 | 0.117 | 0.121 | 0.581 | 0.322 | 0.134 | 0.004 | 0.987 | -0.482 | **0.031*** |
| Gestational age | 0.013 | 0.96 | 0.032 | 0.902 | 0.133 | 0.544 | -0.152 | 0.489 | 0.320 | 0.169 | 0.039 | 0.870 |
| RV basal diameter | 0.568 | **0.017*** | 0.523 | **0.031*** | -0.048 | 0.826 | 0.084 | 0.703 | 0.036 | 0.880 | -0.045 | 0.850 |
| RV mid diameter | 0.688 | **0.002*** | 0.43 | 0.085 | -0.004 | 0.985 | 0.067 | 0.762 | 0.069 | 0.773 | 0.087 | 0.716 |
| RV longitudinal diameter | 0.569 | **0.017*** | 0.466 | 0.06 | 0.311 | 0.148 | 0.125 | 0.571 | 0.233 | 0.322 | -0.035 | 0.884 |
| RV thickness | 0.522 | **0.032*** | 0.542 | **0.025*** | -0.009 | 0.967 | 0.098 | 0.655 | -0.109 | 0.648 | -0.333 | 0.152 |
| TAPSE | -0.488 | **0.047*** | -0.45 | 0.07 | -0.181 | 0.407 | -0.162 | 0.461 | 0.428 | 0.060 | -0.361 | 0.118 |

| | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| S' | -0.467 | 0.059 | -0.457 | 0.065 | -0.188 | 0.391 | -0.332 | 0.122 | 0.339 | 0.144 | -0.053 | 0.826 |
| RAVI | 0.662 | **0.004*** | 0.447 | 0.072 | 0.013 | 0.952 | -0.037 | 0.866 | 0.174 | 0.463 | -0.252 | 0.283 |
| Global RV | 0.682 | **0.003*** | 0.562 | **0.019*** | 0.11 | 0.618 | 0.251 | 0.248 | 0.037 | 0.876 | -0.197 | 0.406 |
| Free wall RV strain | 0.651 | **0.005*** | 0.623 | **0.008*** | 0.214 | 0.326 | 0.189 | 0.389 | -0.074 | 0.758 | 0.139 | 0.560 |

r: correlation coefficient. * Significant P value <0.05. Bl P: blood pressure, BMI: body mass index, Hb: hemoglobin, EF: ejection fraction, LVEDD: left ventricular end-diastolic diameter, LVESD: left ventricular end-systolic diameter, RV: right ventricular, TAPSE: tricuspid annular plane systolic excursion, RAVI: right atrial volume index.

**Table 4:**

**In the preeclampsia Group (After 3 Months Follow-Up):**

Mean systolic blood pressure showed significant positive correlations with RV basal diameter (r=0.650, P=0.005), RV mid diameter (r=0.669, P=0.003), RV longitudinal diameter (r=0.760, P<0.0001), global RV strain (r=0.889, P<0.0001), and free wall RV strain (r=0.836, P<0.0001).

Mean diastolic blood pressure showed significant positive correlations with RV basal diameter (r=0.653, P=0.005), RV mid diameter (r=0.759, P<0.0001), RV longitudinal diameter (r=0.799, P<0.0001), global RV strain (r=0.872, P<0.0001), and free wall RV strain (r=0.850, P<0.0001).

**In the gestational hypertension Group (After 3 Months Follow-Up):**

Mean systolic blood pressure showed significant positive correlations with RV basal diameter (r=0.838, P<0.0001), RV mid diameter (r=0.519, P=0.011), RV longitudinal diameter (r=0.606, P=0.002), global RV strain (r=0.688, P<0.0001), and free wall RV strain (r=0.622, P=0.002).

Mean systolic blood pressure showed a significant negative correlation with S' (r=-0.435, P=0.038).

Mean diastolic blood pressure showed significant positive correlations with RV basal diameter (r=0.800, P<0.0001), RV mid diameter (r=0.543, P=0.007), RV longitudinal diameter (r=0.762, P<0.0001), global RV strain (r=0.648, P=0.001), and free wall RV strain (r=0.414, P=0.049).

Mean diastolic blood pressure showed significant negative correlations with TAPSE (r=-0.430, P=0.041) and S' (r=-0.437, P=0.037).

**In the controls (After 3 Months Follow-Up):**

Mean diastolic blood pressure showed a significant negative correlation with S' (r=-0.454, P=0.044).

**Table 4: Correlation between mean systolic and diastolic BP and other studied parameters after 3 months of follow up in cases and control group**

| | Mean systolic Bl P | | Mean diastolic Bl P | |
|---|---|---|---|---|
| | **R** | **P** | **r** | **P** |
| After 3 months follow up in preeclampsia group | | | | |
| RV basal diameter | 0.650 | **0.005*** | 0.653 | **0.005*** |
| RV mid diameter | 0.669 | **0.003*** | 0.759 | **<0.0001*** |
| RV longitudinal diameter | 0.760 | **<0.0001*** | 0.799 | **<0.0001*** |
| TAPSE | -0.263 | 0.308 | -0.272 | 0.29 |
| S' | -0.436 | 0.08 | -0.4 | 0.112 |
| Global RV | 0.889 | **<0.0001*** | 0.872 | **<0.0001*** |
| Free wall RV strain | 0.836 | **<0.0001*** | 0.850 | **<0.0001*** |
| After 3 months follow up in gestational hypertension group | | | | |
| RV basal diameter | 0.838 | **<0.0001*** | 0.800 | **<0.0001*** |
| RV mid diameter | 0.519 | **0.011*** | 0.543 | **0.007*** |
| RV longitudinal diameter | 0.606 | **0.002*** | 0.762 | **<0.0001*** |
| TAPSE | -0.409 | 0.053 | -0.430 | **0.041*** |
| S' | -0.435 | **0.038*** | -0.437 | **0.037*** |
| Global RV | 0.688 | **<0.0001*** | 0.648 | **0.001*** |
| Free wall RV strain | 0.622 | **0.002*** | 0.414 | **0.049*** |

| | | | | |
|---|---|---|---|---|
| After 3 months follow up in control group | | | | |
| RV basal diameter | 0.048 | 0.841 | -0.025 | 0.918 |
| RV mid diameter | 0.038 | 0.873 | 0.098 | 0.681 |
| RV longitudinal diameter | -0.174 | 0.463 | -0.188 | 0.427 |
| TAPSE | 0.207 | 0.381 | -0.136 | 0.567 |
| S' | -0.147 | 0.537 | -0.454 | **0.044*** |
| Global RV | 0.142 | 0.549 | 0.149 | 0.530 |
| Free wall RV strain | 0.078 | 0.745 | 0.129 | 0.586 |

r: correlation coefficient. * Significant P value <0.05. Bl P: blood pressure, RV: right ventricular, TAPSE: tricuspid annular plane systolic excursion.

Eclampsia was significantly higher in the preeclampsia group (17.65%) compared to both gestational hypertension (0%) and controls (0%) (P = 0.0184), indicating a strong association with severe hypertensive disorders. Preterm labor also showed a significant increase in preeclampsia cases (52.94%) compared to gestational hypertension (17.39%) and controls (15%) (P = 0.015), suggesting that preeclampsia poses a substantial risk for early delivery. Postpartum hemorrhage was significantly more frequent in the preeclampsia group (35.29%) compared to gestational hypertension (4.35%) and controls (5%) (P = 0.0071). **Table 5, Figures (7-8).**

**Table 5: Comparison of all studied groups regarding maternal and fetal complications**

| | Preeclampsia (N = 17) | Gestational hypertension (N = 23) | Controls (N = 20) | P. Value |
|---|---|---|---|---|
| Maternal complications | | | | |
| • Eclampsia | 3 (17.65%) | 0 (0%) | 0 (0%) | **0.0184*** [f] |
| • Maternal hemorrhage | 6 (35.29%) | 4 (17.39%) | 4 (20%) | 0.3795 [X] |
| • Pre-term labour | 9 (52.94%) | 4 (17.39%) | 3 (15%) | **0.015*** [X] |
| Fetal complications | | | | |
| • IUFD | 1 (5.88%) | 3 (13.04%) | 2 (10%) | 0.7569 [X] |
| • IUGR | 7 (41.18%) | 5 (21.74%) | 3 (15%) | 0.1678 [X] |
| Complications after 3 months | | | | |
| • Persistent HTN | 4 (23.53%) | 2 (8.7%) | 0 (0%) | 0.0572 [f] |
| • Post-partum Hemorrhage | 6 (35.29%) | 1 (4.35%) | 1 (5%) | **0.0071*** [X] |
| • Post partum fits | 2 (11.76%) | 1 (4.35%) | 0 (0%) | 0.2578 [f] |
| • Fetal deaths | 3 (17.65%) | 0 (0%) | 1 (5%) | 0.08 [f] |

Data are presented as frequency (%).f: fisher exact test, X: chi-square test, IUFD: intrauterine fetal demise, IUGR: intrauterine growth restriction, HTN: hypertension.

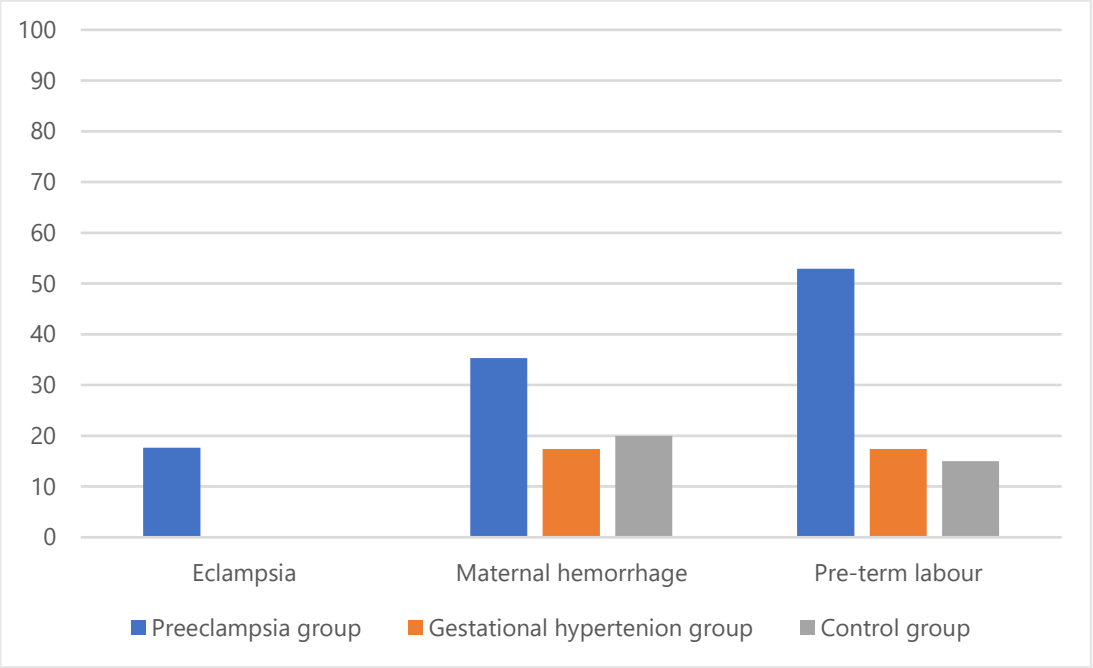


**Figure (7):** Comparison of all studied groups regarding maternal complications.

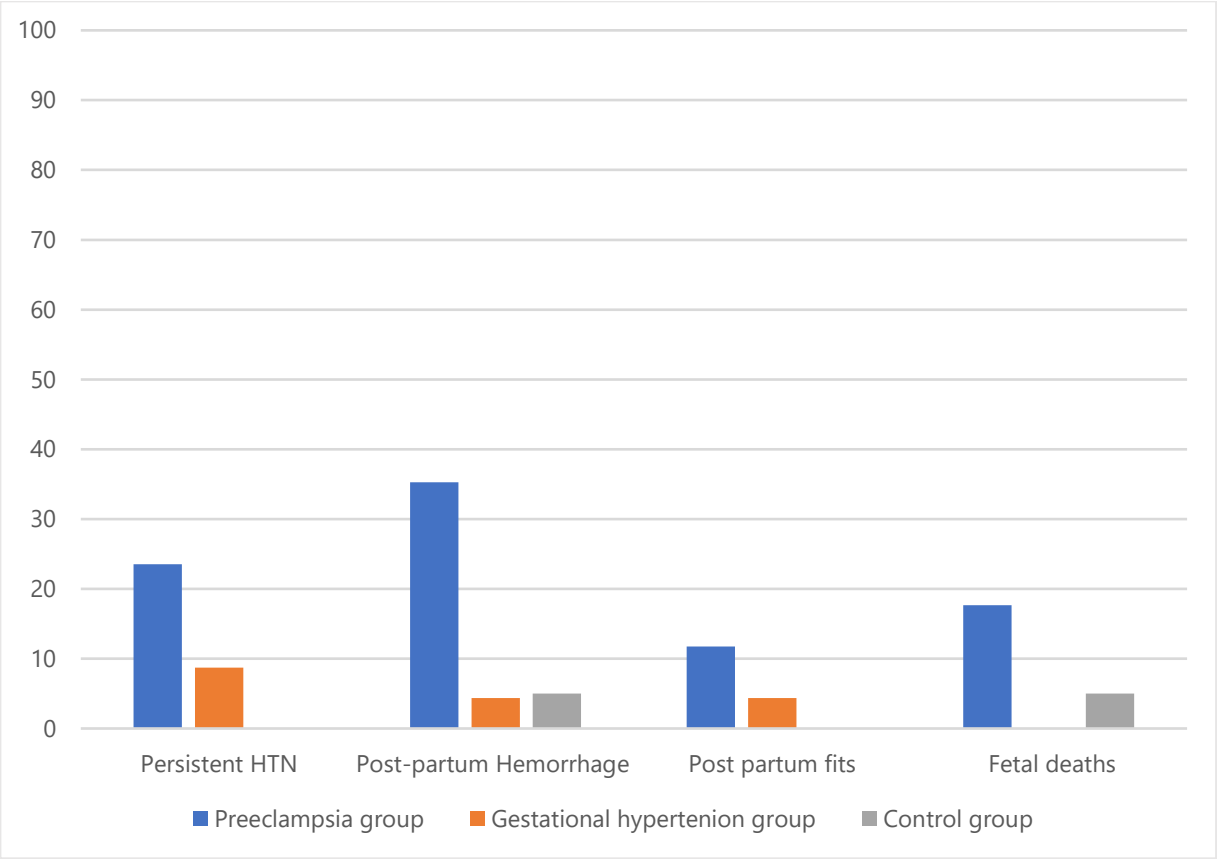


**Figure (8):** Comparison of all studied groups regarding maternal complications.

## DISCUSSION

Hypertensive disorders of pregnancy (HDP) are among the most common gestational complications, encompassing various hypertensive conditions that are consistently associated with increased morbidity and mortality, particularly in the later stages of pregnancy (10).

Regarding comparison of all studied groups as regards maternal and fetal complications. Eclampsia, preterm labor and postpartum hemorrhage were significantly more prevalent in the preeclampsia group. Our study disagrees with Masoura et al. (13) reported that there was statistically significant higher IUGR in the PE group compared to controls. Additionally, Davies et al. (14) reported that a significant positive association was found between PE and preterm birth.

In our study, cases demonstrated significantly higher mean systolic and diastolic blood pressures (BPs) compared to controls. At the 3-month follow-up, mean systolic BP remained significantly elevated relative to controls, with mean diastolic BP also significantly higher in cases. Consistent with our findings, a large study by Jieyu et al. (15) reported significantly greater BP variability among women with pregnancy-related hypertensive disorders compared to normotensive controls. Similarly, Mesquita et al. (16) observed that hypertensive pregnancy disorders were associated with higher systolic and diastolic BPs than in women whose hypertension diagnosis did not occur during pregnancy.

In concordance with our findings on right ventricular (RV) parameters and RV strain, Paudel et al. (17) who demonstrated that hypertensive patients exhibited a significantly larger left atrium, increased interventricular septal thickness, higher systolic pulmonary artery pressure, and elevated mitral E/e′ ratio compared to controls during pregnancy. Additionally, preeclamptic patients had significantly reduced RV global longitudinal strain (RV GLS) compared to controls. Similarly, Çağlar et al. (18) reported notable RV and right atrium (RA) enlargement and impaired RV systolic and diastolic function in women with pregnancy-related hypertensive disorders compared to normotensive controls.

In the preeclampsia group, mean systolic blood pressure showed a significant positive correlation with RV basal, mid, and longitudinal diameters, RV thickness, RAVI, global RV strain, and free wall RV strain, while it had a negative correlation with TAPSE. Mean diastolic blood pressure was positively correlated with RV basal diameter, RV thickness, global RV strain, and free wall RV strain. In the gestational hypertension group, mean systolic blood pressure showed a significant negative correlation with age. In the control group, mean systolic blood pressure had significant negative correlations with age, BMI, and parity.

Melchiorre et al. (19) reported that global diastolic dysfunction occurred more frequently in pregnancies complicated by preeclampsia (PE) compared to controls. Their findings suggested that increased cardiac workload and left ventricular (LV) mass indices indicated adaptive LV remodeling aimed at preserving myocardial contractility in term PE pregnancies. Furthermore Ganesh et al. (20), identified significant risk factors for hypertensive disorders in pregnancy through univariate analysis, including pre-pregnancy BMI > 25, a history of chronic hypertension (HTN), diabetes, renal disease, family history of HTN, prior PE, and multiple pregnancy..

After three months of follow-up in the preeclampsia group, mean systolic and diastolic blood pressures showed significant positive correlations with RV basal, mid, and longitudinal diameters, global RV strain, and free wall RV strain. In the gestational hypertension group, both mean systolic and diastolic blood pressures were positively correlated with RV basal, mid, and longitudinal diameters, global RV strain, and free wall RV strain. Additionally, systolic blood pressure had a significant negative correlation with S', while diastolic blood pressure showed negative correlations with TAPSE and S'. In the control group, mean diastolic blood pressure exhibited a significant negative correlation with S'. A study by Countouris et al. (21) found that women with a history of HDP were more likely to present with current HTN compared to those with normotensive pregnancies. After adjusting for age, race, MVM lesions, BMI, and current HTN, women with HDP history demonstrated increased interventricular septal thickness and relative wall thickness.

In agreement with our result about PE and gestational HTN patients regarding RV parameters and RV strain and after 3 months in cases group, Tadic et al. (10) reported that 24-hour, daytime, and nighttime systolic and diastolic BPs, as well as visit-to-visit systolic and diastolic BPs, were significantly higher in women with Pregnancy related hypertensive disorders compared to controls. Parameters of both short- and long-term BP variability progressively increased from controls to women with PE and were highest in those with GH. Additionally, RV diameter, E/e′ ratio, and PAP were significantly elevated in women with Pregnancy related hypertensive disorders relative to controls.

**Limitations of the study:**

- The relatively small sample size.

- The single center design of the study.
- The short periods of follow up.
- The observational nature of the study means that causality cannot be definitively established between BP variability and RV remodeling. Variations in individual treatment regimens and adherence to antihypertensive therapy may have influenced the outcomes, potentially introducing variability in the results.

## CONCLUSIONS

BP variability significantly influences RV remodeling in pregnant women with pregnancy-related hypertensive disorders. Elevated BP during pregnancy is associated with notable RV structural changes and functional impairment, as indicated by increased RV dimensions, reduced TAPSE, and altered RV strain values. Although maternal and fetal complication rates were comparable between groups, the persistence of elevated BP and RV abnormalities postpartum emphasizes the necessity for continuous CV monitoring and management. These findings underscore the importance of early intervention and comprehensive postpartum care to mitigate long-term CV risks associated with HDP.

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**Author Contribution:**
**Mahmoud Elsayed Abdellatif (MEA);** concept, design, literature search, clinical studies, statistical analysis, manuscript preparation. **Hossam Eldein Mohammed Mohammed (HMM);** design, literature search, manuscript preparation and review**. Amr Hanafy Mahmoud (AHM);** design, literature search, clinical studies, final draft review. **Aml M. Soliman (AMS):** literature search, clinical studies, manuscript editing and final draft preparation and review**.**

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