

ORIGINAL ARTICLE

Potential Impact of Blood Pressure Variability on Right Ventricular Remodeling in Pregnant Women with Pre-Eclampsia or Gestational Hypertension

Aml Mohammed Soliman*, Mahmoud Elsayed Abdellatif, Hossam El-Dein Mohammed, Amr Hanafy Mahmoud

Department of Cardiology, Faculty of Medicine, Aswan University, Aswan, Egypt.

Abstract

Keyword: Blood Pressure, Right Ventricular Remodeling, Pregnancy, Pre-Eclampsia, Gestational Hypertension.

* Corresponding author Mahmoud Elsayed Abdellatif Mobile: 01021728001 E-mail: mahmoud.abdellatief124@gm

ail.com

Background: Hypertensive disorders of pregnancy complicate as much as ten percent of pregnancies and represent a significant cause of maternal and perinatal morbidity and death. **Objective** of this research was to assess right ventricular (RV) and blood pressure (BP) variability remodeling in females had gestational HTN (GH) and PE, in addition to their correlation. Methodology: This observational comparative investigation involved 40 female patients with pregnancy-related hypertensive disorders (Group A) and 20 healthy pregnant women as a control group (Group B) at Aswan University hospital. Results: At three-month follow-up, systolic/diastolic BP, RV dimensions, and RV strain remained significantly higher in the cases group, whereas SD of systolic BP and S' were lower (P<0.05). RV structure and function were similar between preeclampsia and gestational hypertension groups, except for higher RAVI, global RV, and free wall RV strain in preeclampsia (P<0.05). Maternal and fetal complications showed no significant difference between groups. Conclusions: BP variability significantly impacts RV remodeling in pregnant women with GH and PE. Despite similar maternal and fetal complication rates between groups, the persistence of elevated BP and RV abnormalities postpartum underscores the need for ongoing cardiovascular monitoring and management.

INTRODUCTION:

Hypertensive disorders of pregnancy, involving preexisting and gestational hypertension (GH), pre-eclampsia (PE), and eclampsia, complicate approximately ten percent of gestations and represent a significant etiology of maternal and perinatal death and morbidity ^[1].

Any hypertensive disorder during pregnancy might lead to PE. It presents in as much as thirty-five percent of females with GH and as much as twenty-five percent of those with chronic hypertension (HTN) ^[2].

Preeclampsia is correlated with two percent to eight percent globally pregnancy-related complications. It leads to nine percent to twenty-six percent of maternal mortalities in low-income countries and sixteen percent in high-income countries. The variables for the initial diagnosis of preeclampsia are specifically described as a systolic blood pressure of 140 millimeters of mercury or greater or a diastolic blood pressure of 90 millimeters of mercury or greater on 2 occasions a minimum of four hours apart; or shorter interval timing a systolic blood pressure of 160 millimeters of mercury or above or a diastolic blood pressure of 110 millimeters of mercury or above may be recorded within a shorter interval, all of that must occur following twenty weeks of pregnancy ^[3].

The initial presentation of PE usually occurs in near-term pregnancies. Additional significant results that might or might not be present in the clinical presentation involve proteinuria, signs of endorgan damage like thrombocytopenia, compromised hepatic function, severe persistent pain in the



right upper quadrant or epigastric, excluding all alternative diagnoses, new-onset headache unresponsive to all treatment forms, kidney insufficiency, or pulmonary edema with abnormal lab values [4].

GH, formerly referred to as pregnancy-induced hypertension, is an onset of hypertension following twenty weeks of gestation. The diagnosis needs that the patient demonstrates ^[5] high blood pressure (systolic not less than 140 or diastolic not less than 90 millimeters of mercury, with the latter assessed via the 5th Korotkoff sound), previous history of normal blood pressures, absence of protein in the urine, and absence of manifestations of PE or eclampsia. Additionally referred to as HTN, GH is identified retrospectively when the case doesn't develop to preeclampsia and if blood pressure returns to normal by the twelve-week postpartum visit. 50% of females identified with GH among twenty-four and thirty-five weeks develop PE. The diagnosis of GH mandates raised monitoring. Women who progress to severe GH due to elevated blood pressure demonstrate worse perinatal results compared to those with mild preeclampsia and need treatment like to that of severe preeclampsia ^[6].

Dramatic cardio-vascular (CV) changes occur during pregnancy to meet the maternal and growing fetal metabolic requirements. Blood volume increases, and PVR decreases along with progressive placental growth. Increase also in heart rate and cardiac output during pregnancy ^[7]. Cardiac remodeling, involving progressive mild dilation of all cardiac chambers and rise in LV mass, occurs as a compensatory response for such changes. PVR throughout normal gestation keeps low despite raised renin and angiotensin II blood concentrations. This lack of vascular response to activated renin-angiotensin system may be related to the humoral factors such as prostaglandin and progesterone. Abnormal pressure overloading in pregnancy complicated by HTN would result in variant cardiac remodeling compared with that of normal gestation ^[8].

Whereas BP measurements are widely accepted as essential for diagnosing and managing heart failure and HTN, the possible importance of blood pressure variability (BPV), alone or in tandem with heart rate variability (HRV), remains unexamined because of the lack of wearable, convenient, continuous blood pressure monitors ^[9].

Blood pressure variability is characterized as an alteration in arterial blood pressure over a described duration. The complex physiology of blood pressure variability is dependent on the interactions among hemodynamic, humoral, neuronal, in addition to behavioral factors (lifestyle, anxiety, as well as postural alters), environmental factors (atmospheric pressure and climate). and the interaction of aortic compliance and systemic capacitance and is complicated by concurrent antihypertensive and heart failure medical treatments [10].

The potential clinical significance of blood pressure variability is not yet fully established; nevertheless, three aspects must be deemed. Blood pressure variability introduces uncertainty in evaluating a subject's blood pressure state, particularly when utilizing spot clinic assessments. The evaluation of BPV may enhance cardiovascular risk stratification, though the size of its independent contribution in this regard still to be superior recorded. Elevated blood pressure variability may serve as a therapy target, with the objective of enhancing results, probably without generating further costs. The option of long-acting medications, in particular dihydropyridine calcium antagonists and combinations of long-lasting compounds, may be indicated for people with raised blood pressure variability; though, the potential clinical advantages of this method remain inadequately demonstrated [111]. So, in this study we aimed to evaluate RV and BPV remodeling in females with PE and GH, in addition to their correlation.

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 GH or PE and **group B** (n=20): with normal pregnancy serve as control group.

We ruled out patients with gestation period below twenty weeks, congenital heart diseases, moderate to severe valvular heart diseases, cardiomyopathy whatever its reason, underlying RV dysfunction and poor image quality.

Procedure: All cases have been subjected to complete history taking, clinical examination, laboratory investigations [complete blood count (CBC), coagulation profile, platelet count (PLT), international normalization ratio (INR), activated partial thromboplastin time (aPTT), prothrombin time (PT), blood sugar, alanine transaminase (ALT), aspartate aminotransferase (AST), serum creatinine, blood urea, urine albumin (dipstick) and albumin/creatinine ratio in urine] and ECG or any additional research that is needed based on the patients' clinical situation.

• Office BP measurements:

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

Non-invasive Imaging:

Two-dimensional transthoracic echocardiography [12]:

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

- o RV Basal, mid, and longitudinal diameters.
- o RV function by TAPSE and S Velocity methods.
- Speckle tracking (STE) analysis of RV:

The examination began by positioning the case in the left lateral decubitus position and the probe was moved across his chest to determine the RV strain [13]. Image acquisition has been conducted from an RV-focused view at the apical four-chamber view to fully visualize the right ventricular free wall, right ventricular apex, and tricuspid valve/annulus within the imaging sector throughout systole and diastole. At a suitable intermediate depth (intermediate depth), any excessive anterior tilt (left ventricular outflow tract not observed) or posterior tilt (coronary sinus not observed) has been avoided. All strain measurements have been automatically given via the software with color coding. The image has been evaluated for tracking quality. Manual corrections have been done when needed. Each segment region of interest (ROI) was carefully discovered for proper placement along the RV-free wall and ventricular septum. region of interest thickness has been set at five millimeters in a non-hypertrophied right ventricular free wall and has been raised in a Pericardial tracking was completely prevented or, at best, minimized to the hypertrophied RV. lowest extent possible for the outer contour. The inner contour was along the endocardial border, excluding trabeculations and papillary muscle. The tracking stopped at the tricuspid annulus and wasn't into the right ventricular or the right ventricular, and away from the tricuspid annulus. right ventricular global longitudinal strain (RV GLS): Involved the RV-free wall and the ventricular septum. Normal range: 20%-25%. right ventricular -free wall strain (RV FWS): excluded the ventricular septum. Normal range: 23%-33%.

Statistical analysis

Statistical analysis has been carried out by SPSS v26 (IBM Inc., Chicago, IL, United States of America). Quantitative parameters have been presented as mean and standard deviation (SD) and compared among the 2 groups applying unpaired Student's t-test. Qualitative parameters have been presented as percentage (%) and frequency and examined utilizing the Chi-square or Fisher's exact



test when suitable. Association among numerous parameters has been conducted utilizing Pearson moment correlation equation. A two-tailed P value under 0.05 has been deemed statistically significant.

Ethical Consideration:

The Medical Ethic Committee of Aswan University's Faculty of Medicine granted IRB permission (IRB?????). Clinical trial.gov was used to prospectively register the study Clinical trial.gov ID:NCT06100484). The research has been performed following the principles outlined in the Helsinki Declaration [14] and in accordance with CONSORT checklist for research ethics [15]. Prior to the beginning of the research, the title and goal of the study were completely clarified 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 research 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.

Age, parity, gestational age and diabetes, creatinine, Hb, SD of systolic, diastolic Bl P and EF were insignificantly different between both groups. Base line BMI, 24-hrs urine protein level, mean systolic, diastolic Bl P, LVEDD, LVESD, RV basal, mid and longitudinal diameter, RV thickness, RAVI, global RV and free wall RV strain were significantly greater in cases group in comparison with control group (P below 0.05). TAPSE and S' were significantly decrease in cases group compared to control group (P below 0.05). Cases group includes 17 patients (42.5%) with PE and 23 patients (57.5%) with gestational diabetes. **Table 1, Figures (1-4).**

Table 1: Comparison of all examined groups according to demographic, baseline laboratory data, mean systolic, diastolic Bl P, LV, RV parameters and RV strain and description of

diagnosis in cases group

ulagnosis in cases group	Cases group	Control group	Test	P				
	(n=40)	(n=20)						
Age (years)	30.7±5.2	32.8±4.1	T = -1.550	0.126				
BMI (kg/m ²)	31±3.6	28.9±3.7	T=2.294	0.025*				
Parity	2.4 ± 1.2	2.1±1	T= 1.026	0.309				
Gestational age	32 ± 3.9	32.5±4.6	T = -0.372	0.711				
Gestational diabetes	4(10.0%)	2(10.0%)	$X^2 = 0$	1				
Antihypertensive therapy	20(50.0%)	0(0.0%)	$X^2 = 15$	<0.001*				
	Laboratory data							
Creatinine (mg/dL)	86.1±15	79.8±11.4	T=1.646	0.105				
Hb (g/dl)	11.2±1.4	11.6±1.3	T=-1.094	0.279				
24-hrs urine protein level (mg/day)	894±939	91.4±19.9	T=3.809	<0.001*				
Mean systolic Bl P	152±3.6	120±1.8	T=36.341	<0.001*				
SD of systolic Bl P	3.6±1.8	3±1.8	T=1.137	0.260				
Mean diastolic Bl P	92±2	75±3.4	T=24.756	<0.001*				
SD of diastolic Bl P	3±1.7	2.6±1.4	T=0.845	0.402				
	LV param	neters						
EF (%)	60.7±3.5	62.1±2.5	T=-1.582	0.119				
LVEDD (mm)	47.9±2.7	45.8±3.8	T=2.462	0.017*				
LVESD (mm)	30.8±3.8	26±3.7	T=4.649	<0.001*				
	RV param	neters						



RV basal diameter (mm)	33.4±6.5	23.6±2	T=6.578	<0.001*			
RV mid diameter (mm)	32.1±3.4	26.2±2	T=7.227	<0.001*			
RV longitudinal diameter	75.5±5.1	62±4.9	T=9.797	<0.001*			
(mm)							
RV thickness (mm)	3.3±0.4	3±0.4	T=2.296	0.025*			
TAPSE (mm)	21.6±3.3	23.9±1.9	T=-2.947	0.005*			
S' (cm/s)	11.8±2.4	14±3.3	T=-2.913	0.005*			
RAVI (ml/m ²)	24.1±4.4	17.4±3.1	T=6.068	<0.001*			
	RV stra	in					
Global RV	-19.6±2.2	-23.2±2	T=6.025	<0.001*			
Free wall RV strain (%)	-21.7±2.3	-27.2±2.7	T=8.063	<0.001*			
	Cases group (n= 40)						
Diagnosis							
PE	17(4						
Gestational HTN	23(5	7.5%)					

Data are presented as mean \pm SD or frequency (%). * Significant P value below 0.05. T: independent sample t test, X^2 : chi-square test, BMI: body mass index, Hb: hemoglobin, Bl P: blood pressure, SD: standard deviation, EF: ejection fraction, LVEDD: left ventricular end-diastolic diameter, RV: right ventricular, TAPSE: tricuspid annular plane systolic excursion, RAVI: right atrial volume index, LVESD: left ventricular end-systolic diameter, PE: preeclampsia.

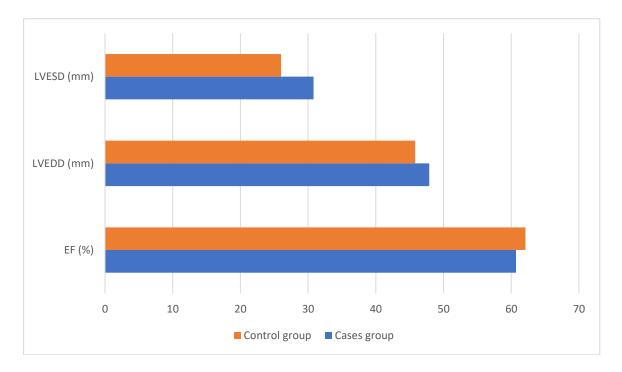


Figure (1): Comparison among the cases and controls according to the baseline mean values of LV parameters.



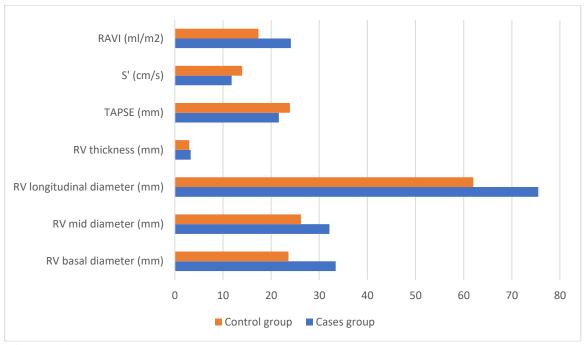


Figure (2): Comparison among the cases and controls as regards the baseline mean values of RV parameters.

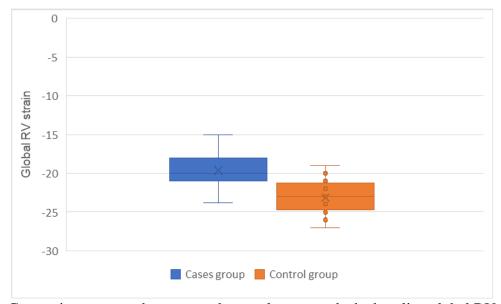


Figure (3): Comparison among the cases and controls as regards the baseline global RV strain.



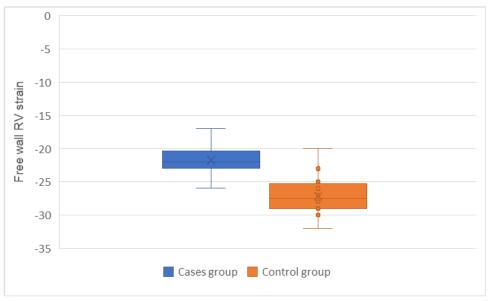


Figure (4): Comparison between the cases and controls regarding the baseline free wall RV strain.

Three months follow up mean systolic and diastolic Bl P, RV basal, mid and longitudinal diameter, global RV and free wall RV strain were significantly greater in cases group compared to control group (P below 0.05). SD of systolic Bl P and S' were significantly reduce in cases group in comparison with control group (P below 0.05). SD of diastolic Bl P, TAPSE, Δ global RV and Δ free wall RV strain were insignificantly variant among both groups. **Table 2, Figure (5-7).**

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

.,	Strain and its delta ar	Cases	Controls	T test	P			
		(n=40)	(n=20)					
	After 3 months							
Mean	systolic Bl P	125±12.3	117 ± 2.3	3.082	0.003 *			
SD of	systolic Bl P	3.1±1.4	4.2±1.7	-2.700	0.009 *			
Mean	diastolic Bl P	78±7.2	71 ± 2.1	4.229	<0.001*			
SD of	diastolic Bl P	3.9±1.9	3.1±1.3	1.709	0.093			
RV	RV basal diameter	31±5.1	25±1.9	5.136	<0.001*			
parameters	(mm)							
	RV mid diameter	31.2±3.6	27.2 ± 2	4.628	<0.001*			
	(mm)							
	RV longitudinal	73.6±4.8	63.8 ± 4.1	7.853	<0.001*			
	diameter (mm)							
	TAPSE (mm)	23.3±2.7	24.2±1.6	-1.324	0.191			
	S'(cm/s)	12.2±2.3	14±3.2	-2.479	0.016*			
RV strain	Global RV	-22.3±2.3	-25.3±1.6	5.291	<0.001*			
	Free wall RV	-24.3±2.4	-28.6 ± 2.3	6.602	<0.001*			
	strain (%)							
Δ RV strain	∆ Global RV	-13.98±13	-10±9.3	-1.21	0.18			
	Δ Free wall RV	-12.7±12.1	-6.8 ± 16.4	-1.58	0.12			
	strain							

 Δ : delta.



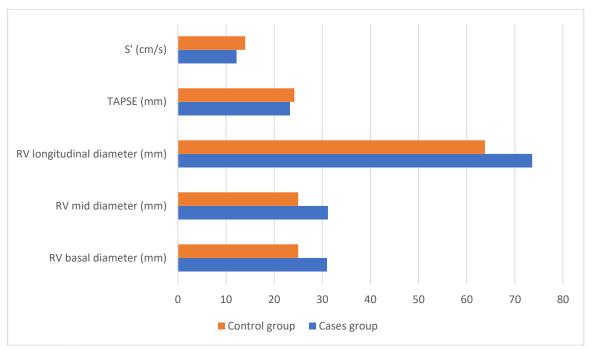


Figure (5): Comparison among the cases and controls according to the mean values of RV parameters after 3 months.

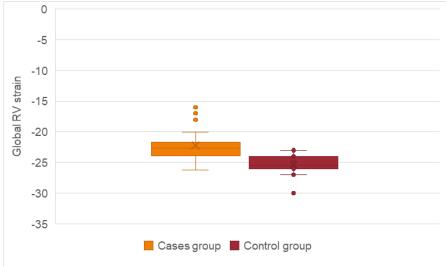


Figure (6): Comparison among the cases and controls as regards the global RV strain after 3 months



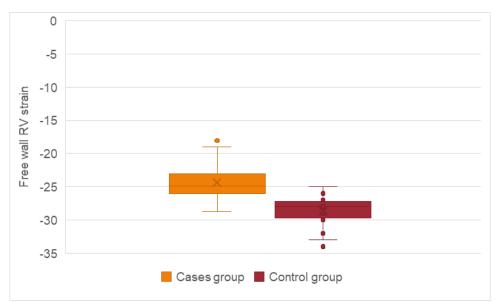


Figure (7): Comparison among the cases and controls as regards the free wall RV strain following three months

There was a significant negative association among mean systolic blood pressure and age and TAPSE in cases group (P below 0.05). There was a significant positive association among mean systolic blood pressure and creatinine, RV mid and longitudinal diameter, RAVI, global RV and free wall RV in cases group (P below 0.05). There was a significant positive association among mean diastolic BP and BMI, creatinine, RV basal and longitudinal diameter, global RV and free wall RV in cases group. There was a significant negative association among mean diastolic BP and TAPSE and S'. There were no significant association among mean systolic and diastolic BP and other parameters in cases and control group. There was a significant negative association among mean diastolic BP and BMI and parity in control group (P below 0.05). **Table 3**

Table 3: Relation among mean systolic and diastolic BP and other studied parameters in cases and control group

and control group								
	Mean sy	stolic Bl	Mean dia	stolic Bl	Mean s	ystolic	Mean dia	stolic Bl
]	P	P		Bl	P	P	
	R	P	r	P	r	P	r	P
Cases group					Contr	ol group		
Age	-0.330	0.037*	-0.178	0.273	0.078	0.744	0.202	0.394
\mathbf{BMI}	0.123	0.449	0.340	0.032*	-0.002	0.993	-0.547	0.012*
Parity	0.002	0.988	-0.039	0.812	0.004	0.987	-0.482	0.031*
Gestational age	0.033	0.841	-0.077	0.639	0.320	0.169	0.039	0.870
Creatinine	0.325	0.041*	0.316	0.047*	-0.228	0.334	-0.069	0.773
Hb	-0.179	0.268	-0.296	0.064	-0.018	0.938	-0.304	0.193
24h urine protein	0.235	0.145	0.118	0.469	0.317	0.173	-0.053	0.823
EF	-0.002	0.990	-0.096	0.556	-0.070	0.769	0.245	0.298
LVEDD	0.094	0.564	0.067	0.679	0.266	0.257	-0.166	0.485
LVESD	0.124	0.447	0.139	0.393	-0.122	0.607	-0.194	0.412
RV basal diameter	0.280	0.081	0.336	0.034*	0.036	0.880	-0.045	0.850
RV mid diameter	0.353	0.026*	0.272	0.089	0.069	0.773	0.087	0.716
RV longitudinal diameter	0.459	0.003*	0.332	0.036*	0.233	0.322	-0.035	0.884
RV thickness	0.200	0.217	0.283	0.076	-0.109	0.648	-0.333	0.152



TAPSE	-0.349	0.027	-0.316	0.047*	0.428	0.060	-0.361	0.118
S'	-0.283	0.077	-0.379	0.016*	0.339	0.144	-0.053	0.826
RAVI	0.320	0.044*	0.194	0.231	0.174	0.463	-0.252	0.283
Global RV	0.429	0.006*	0.396	0.011*	0.037	0.876	-0.197	0.406
Free wall RV strain	0.440	0.005*	0.391	0.013*	-0.074	0.758	0.139	0.560

r: correlation coefficient.

There was a significant positive relation among mean systolic and diastolic BP and right ventricular basal, mid and longitudinal diameter, global RV and free wall RV strain in cases group after three months (P below 0.05). There was a significant negative association among mean systolic and diastolic BP and TAPSE, S' (P below 0.05). There was insignificant relation among mean diastolic and systolic BP and other parameters in control group after 3 months. There was a significant negative association among mean diastolic BP and S' in control group after 3 months (P below 0.05). **Table 4**

Table 4: Relation among mean systolic and diastolic BP and other studied parameters following three months of monitoring in cases and control group

Tonowing three months of mon		stolic Bl P		stolic Bl P			
	R	P	r	P			
After 3 months follow up in cases group							
RV basal diameter	0.735	<0.001*	0.724	<0.001*			
RV mid diameter	0.542	<0.001*	0.617	<0.001*			
RV longitudinal diameter	0.686	<0.001*	0.779	<0.001*			
TAPSE	-0.363	0.021*	-0.369	0.019*			
S'	-0.422	0.007*	-0.410	0.009*			
Global RV	0.803	<0.001*	0.775	<0.001*			
Free wall RV strain	0.717	<0.001*	0.637	<0.001*			
After	3 months follow	w up in control gr	oup				
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			

RV basal, mid and longitudinal diameter, right ventricular thickness, TAPSE and S' were insignificantly different among both groups. RAVI, global right ventricular and free wall RV strain were significantly greater in pre-eclampsia group in comparison with gestational HTN group (P below 0.05). RV parameters and RV strain were insignificantly variant among both groups after 3 months. **Table 5**

Table 5: Comparison of PE and gestational HTN patients regarding RV parameters and RV strain and after 3 months in cases group

		PE (n= 17)	Gestational HTN (n= 23)	T test	P
RV	RV basal diameter (mm)	34.8±7.3	32.4±5.8	1.1	0.25
parameters	RV mid diameter (mm)	33±3.7	31.4±3.1	1.5	0.15
	RV longitudinal diameter (mm)	76.5±6.1	74.8±4.3	1.1	0.3
	RV thickness (mm)	3.4±0.4	3.3 ± 0.5	0.7	0.49
	TAPSE (mm)	20.8±3.4	22.1±3.2	-1.3	0.2



	S'(cm/s)	11.9±2.2	11.8±2.5	0.13	0.9		
	RAVI (ml/m²)	25.8±3.8	22.8±4.5	2.22	0.032*		
RV strain	Global RV	-18.4±2	-20.6±1.8	2.5	0.001*		
	Free wall RV strain (%)	-20.8±2.1	-22.4±2.3	2.3	0.028*		
After 3 months							
RV	RV basal diameter (mm)	32.1±6	30.2±4.3	1.2	0.25		
parameters	RV mid diameter (mm)	30.8±3.7	31.4±3.6	-0.58	0.57		
	RV longitudinal diameter (mm)	73.9±5.6	73.4±4.2	0.36	0.72		
	TAPSE (mm)	22.6±3.1	23.9±2.4	-1.5	0.15		
	S'(cm/s)	12.2±2.8	12.2±2	-0.04	0.97		
RV strain	Global RV	-21.7±2.5	-22.6±2.2	1.2	0.24		
	Free wall RV strain (%)	-24.2±2.6	-24.4±2.3	0.28	0.78		

Maternal and foetal complications were insignificantly variant between both groups. **Table 6**, **Figures (8-9).**

Table 6: Comparison of all studied groups regarding maternal and fetal complications

	Ü	Cases group (n= 40)	Control group (n= 20)	\mathbf{X}^2	P
Maternal	Eclampsia	3(7.5%)	0(0.0%)	1.58	0.21
complications	Haemorrhage	10(25.0%)	4(20.0%)	0.186	0.67
	Pre-term labor	13(32.5%)	3(15.0%)	2.1	0.15
Fetal	IUFD	4(10.0%)	2(10.0%)	0	1
complications	IUGR	12(30.0%)	3(15.0%)	1.6	0.21
	Compl	ications after 3 mo	nths		
Persiste	nt HTN	6(15.0%)	0(0.0%)	3.33	0.068
Post-partum haemorrhage		7(17.5%)	1(5.0%)	1.8	0.179
Post partum fits		3(7.5%)	0(0.0%)	1.58	0.21
Fetal (deaths	3(7.5%)	1(5.0%)	0.134	0.714

IUGR: intrauterine growth restriction, IUFD: intrauterine fetal demise, HTN: hypertension.

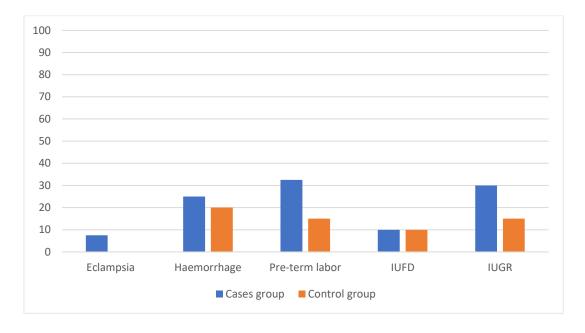


Figure (8): Comparison between cases and controls regarding complications.



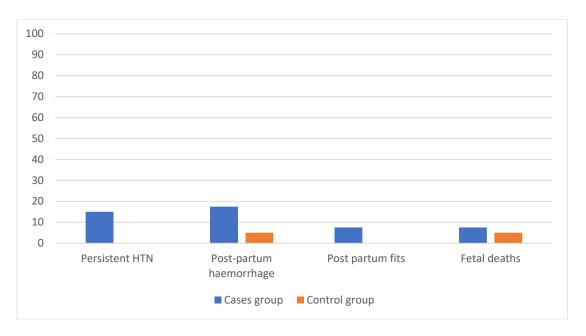


Figure (9): Comparison between cases and controls regarding complications after 3 months.

DISCUSSION

Hypertensive disorders in pregnancy (HDP) represent one of the most frequent complications throughout pregnancy. This entity involves numerous hypertensive disorders and all of them are related to raised death and morbidity throughout gestation and especially in its late stage ^[13].

Regarding comparison of all studied groups as regard maternal and fetal complications. There were non-significant variances among the groups for eclampsia, maternal hemorrhage, preterm labor, IUFD, IUGR, persistent HTN, post-partum hemorrhage, post-partum fits and fetal deaths. Our study disagrees with Masoura et al. [16] stated that there was statistically significant greater IUGR in the PE group in comparison with controls. Additionally, Davies et al. [7] stated that a significant positive correlation was found among PE and preterm birth.

In our study cases had significantly greater mean diastolic and systolic BPs compared to controls. No significant difference has been observed in the SD of systolic and diastolic BP. After 3 months, cases had significantly higher mean systolic BP compared to controls and significantly lower SD of systolic BP compared to controls. Mean diastolic BP was significantly higher in cases vs. controls. No significant difference in SD of diastolic BP. Large study by Jieyu et al. [17] stated comparable outcomes with significantly greater blood pressure variability among females has GH and PE in comparison with in normotensive controls. Additionally, Mesquita et al. [18] reported hypertensive pregnancy disorders exhibited higher systolic and diastolic BP than females whose diagnosis of HTN didn't occur throughout gestation.

In agreement with our result about LV, RV parameters and RV strain, Paudel et al. ^[19] illustrated that preeclamptic cases had a significantly greater left atrium, a thicker interventricular septum, greater systolic pulmonary artery pressure, and an increased mitral E/e' ratio in comparison with controls throughout gestation, whereas left ventricular ejection fraction remained comparable. Preeclamptic cases demonstrated significantly decreased left ventricular and RV GLS throughout gestation in comparison with controls. Çağlar et al. ^[20] stated a significant enlargement of right ventricular and RA, and deterioration of right ventricular diastolic and systolic function in females had PE in comparison with controls.

In cases, mean systolic BP correlated significantly with age, creatinine, RV mid diameter, RV longitudinal diameter, TAPSE, RAVI, global RV and free wall RV strain. No significant correlations were found with other parameters. Mean diastolic BP significantly correlated with



BMI, creatinine, RV basal diameter, RV longitudinal diameter, TAPSE, S, global RV and free wall RV strain. No significant correlations were found with other parameters. In controls, mean systolic BP shows no significant correlations with other parameters. Mean diastolic BP significantly negatively correlates with BMI and parity. No other significant correlations are found. A study by Melchiorre et al. [21] reported that global diastolic dysfunction has been discovered more frequently in PE against control pregnancies. Raised cardiac work and left ventricular mass indices recommend that left ventricular remodeling was an adaptive response to sustain myocardial contractility with PE at term. Furthermore Ganesh et al. [22] reported significant risk factors recognized in univariate analysis involved pre-pregnancy body maa index above twenty-five, history of chronic hypertension, history of kidney illness, history of diabetes, family history of hypertension, history of pre-eclampsia in previous gestation and numerous gestation.

After 3 months in cases, mean systolic BP significantly positively correlated with RV mid diameter, right ventricular basal diameter, RV longitudinal diameter, global right ventricular and free wall RV strain. It negatively correlated with TAPSE, and S. Mean diastolic BP significantly positively correlated with right ventricular basal diameter, RV longitudinal diameter, right ventricular mid diameter, global RV and free wall RV strain. It negatively correlated with TAPSE and S. In controls after 3 months, mean systolic BP showed no significant correlations with other parameters. Mean diastolic BP had a significant negative correlation with S. No other significant correlations were found. A study by Countouris et al. [23] revealed that Compared with females with normotensive pregnancies, those with Hypertensive disorders in pregnancy history were more likely to have present HTN. Following adjusting for race, age, MVM lesions, current HTN, BMI, and hemoglobin A1c, females have hypertensive disorders in pregnancy history had greater 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. ^[13] reported that twenty-four hours, daytime, and nighttime diastolic and systolic blood pressures, in addition to visit-to-visit diastolic and systolic blood pressures, were significantly raised in females with pre-eclampsia and gestational hypertension compared to the control group. The parameters of short- and long-term blood pressure variability gradually raised from the control group, through those with pre-eclampsia, to those with GH. The right ventricular diameter, E/e' ratio, and PAP were significantly greater in females with GH and PE compared to the control group.

Limitations of the study:

- The research was in an only one center.
- the sample size was comparatively small.
- The monitoring of cases has been restricted for relatively short duration.
- 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 impacts RV remodeling in pregnant women with PE and GH. Elevated BP during pregnancy is associated with pronounced RV structural changes and impaired function, as evidenced by increased RV dimensions, decreased TAPSE, and altered RV strain measurements. Despite similar maternal and fetal complication rates between groups, the persistence of elevated BP and RV abnormalities postpartum underscores the need for ongoing CV monitoring and management. These results emphasize the critical importance of early intervention and comprehensive postpartum care to address the long-term CV risks associated with HDP.

Disclosure Statement: There were no conflicts of interest for the authors.



Financial support and sponsorship: None.

Author Contribution:

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

REFERENCES

- 1. Opichka MA, Rappelt MW, Gutterman DD, Grobe JL, McIntosh JJ. Vascular dysfunction in preeclampsia. Cells. 2021;10:30-55.
- 2. Khedagi AM, Bello NA. Hypertensive disorders of pregnancy. Cardiol Clin. 2021;39:77-90.
- 3. Phoswa WN, Khaliq OP. The role of oxidative stress in hypertensive disorders of pregnancy (preeclampsia, gestational hypertension) and metabolic disorder of pregnancy (gestational diabetes mellitus). Oxid Med Cell Longev. 2021;20:558-70.
- 4. Wang W, Xie X, Yuan T, Wang Y, Zhao F, Zhou Z, et al. Epidemiological trends of maternal hypertensive disorders of pregnancy at the global, regional, and national levels: a population-based study. BMC Pregnancy Childbirth. 2021;21:364-50.
- 5. Hu B, He X, Li F, Sun Y, Sun J, Feng L. Childhood obesity and hypertension in pregnancy: a two-sample Mendelian randomization analysis. J Hypertens. 2023;41:1152-8.
- 6. Leeson P. Chronic hypertension in pregnancy project and the control of hypertension in pregnancy study: Impact of blood pressure control in pregnancy on maternal and fetal outcomes. Cardiovasc Res. 2022;118:98-100.
- 7. Davies EL, Bell JS, Bhattacharya S. Preeclampsia and preterm delivery: a population-based case—control study. Pregnancy Hypertens. 2016;35:510-9.
- 8. Gemechu KS, Assefa N, Mengistie B. Prevalence of hypertensive disorders of pregnancy and pregnancy outcomes in Sub-Saharan Africa: A systematic review and meta-analysis. Womens Health (Lond). 2020;16:300-40.
- 9. Sinkey RG, Battarbee AN, Bello NA, Ives CW, Oparil S, Tita AT. Prevention, diagnosis, and management of hypertensive disorders of pregnancy: a comparison of international guidelines. Curr Hypertens Rep. 2020;22:1-10.
- 10. Narang K, Szymanski LM. Multiple gestations and hypertensive disorders of pregnancy: what do we know? Curr Hypertens Rep. 2021;23:1-14.
- 11. Qin Y, Bily D, Aguirre M, Zhang K, Xie L. Understanding PPARγ and its agonists on trophoblast differentiation and invasion: Potential therapeutic targets for gestational diabetes mellitus and preeclampsia. Nutr. 2023;15:100-30.
- 12. Mitchell C, Rahko PS, Blauwet LA, Canaday B, Finstuen JA, Foster MC, et al. Guidelines for performing a comprehensive transthoracic echocardiographic examination in adults: recommendations from the American Society of Echocardiography. JASE. 2019;32:1-64.
- 13. Tadic M, Cuspidi C, Suzic Lazic J, Vukomanovic V, Mihajlovic S, Savic P, et al. Blood pressure variability correlates with right ventricular strain in women with gestational hypertension and preeclampsia. J Hum Hypertens. 2022;36:826-32.
- 14. Association WM. World Medical Association Declaration of Helsinki: ethical principles for medical research involving human subjects. Jama. 2013;310:2191-4.
- 15. Moher D, Hopewell S, Schulz KF, Montori V, Gøtzsche PC, Devereaux PJ, et al. CONSORT 2010 explanation and elaboration: updated guidelines for reporting parallel group randomised trials. BmJ. 2010;340:869-40.



- 16. Masoura S, Kalogiannidis I, Makedou K, Theodoridis T, Koiou K, Gerou S, et al. Biomarkers of endothelial dysfunction in preeclampsia and neonatal morbidity: a case—control study. Eur J Obstet Gynecol Reprod Biol 2014;175:119-23.
- 17. Jieyu L, Yingying C, Tian G, Jiaxiang W, Jiawen L, Yingjie G, et al. Visit-to-visit blood pressure variability is associated with gestational hypertension and pre-eclampsia. Pregnancy Hypertens. 2019;18:126-31.
- 18. Mesquita RF, Reis M, Beppler AP, Bellinazzi VR, Mattos SS, Lima-Filho JL, et al. Onset of hypertension during pregnancy is associated with long-term worse blood pressure control and adverse cardiac remodeling. J Am Soc Hypertens. 2014;80:827-31.
- 19. Paudel A, Tigen K, Yoldemir T, Guclu M, Yildiz I, Cincin A, et al. The evaluation of ventricular functions by speckle tracking echocardiography in preeclamptic patients. Int J Cardiovasc Imaging. 2020;36:1689-94.
- 20. Çağlar FNT, Ozde C, Bostancı E, Çağlar İM, Çiftçi S, Unğan İ, et al. Assessment of right heart function in preeclampsia by echocardiography. Pregnancy Hypertens. 2016;6:89-94.
- 21. Melchiorre K, Sutherland GR, Baltabaeva A, Liberati M, Thilaganathan B. Maternal cardiac dysfunction and remodeling in women with preeclampsia at term. Hypertens. 2011;57:85-93.
- 22. Ganesh KS, Unnikrishnan B, Nagaraj K, Jayaram S. Determinants of pre-eclampsia: a case control study in a district hospital in South India. Indian J Community Med 2010;35:502-5.
- 23. Countouris ME, Villanueva FS, Berlacher KL, Cavalcante JL, Parks WT, Catov JM. Association of hypertensive disorders of pregnancy with left ventricular remodeling later in life. J Am Coll Cardiol. 2021;77:1057-68.