



Research Paper

## Preeclampsia-Induced Cardiovascular Alterations and Their Impact on Left Ventricular Structure during Pregnancy

Anwara, CE<sup>1</sup>., Ezemagu, UK<sup>2</sup>., Egwu, OA<sup>2</sup>., Egwu, EO<sup>1</sup>., Nkanu, II<sup>1</sup>., Obun, C<sup>3</sup>., Epete, MA<sup>1</sup>., Okoro, CC<sup>1</sup>., Chukwuemeka, F<sup>1</sup>., Otuu, O<sup>4</sup>.

Anwara, Chinemerem Elizabeth; Department of Anatomy, Ebonyi State University, Abakaliki, Nigeria.  
Ezemagu, Uchenna K.; Department of Anatomy, Alex Ekwueme Federal University, Ndufu-Alike, Ikwo, Ebonyi State, Nigeria.

Egwu, Ogugua Augustine; Department of Anatomy, Alex Ekwueme Federal University, Ndufu-Alike, Ikwo, Ebonyi State, Nigeria.

Egwu, EniOgbonnia; Department of Anatomy, Ebonyi State University, Abakaliki, Nigeria

Nkanu, Ibarereh Irem; Department of Anatomy, Ebonyi State University, Abakaliki, Nigeria.

Obun, Cletus; Department of Anatomy and Forensic Anthropology, University of Cross River State, Nigeria.

Epete, Micheal Arinze; Department of Anatomy, Ebonyi State University, Abakaliki, Nigeria.

Otuu Onyeyirichi; Department of Surgery, Alex Ekwueme Federal University, Ndufu-Alike, Ikwo, Ebonyi State, Nigeria.

Okoro Caleb Chigozie. Department of Anatomy, Ebonyi State University, Abakaliki, Nigeria.

Chukwuemeka Favour; Department of Anatomy, Ebonyi State University, Abakaliki, Nigeria.

Corresponding Author: Nkanu, Ibarereh Irem

**ABSTRACT:** Preeclampsia is a significant hypertensive disorder in pregnancy, characterized by elevated blood pressure and proteinuria after the 20th week of gestation. It affects 5-8% of pregnancies and poses substantial risks to both maternal and fetal health. This study investigates the association between preeclampsia, left ventricular hypertrophy (LVH), and blood pressure in pregnant women attending Alex Ekwueme Federal University Teaching Hospital, Abakaliki, Ebonyi State. A cohort of 33 preeclampsia women was enrolled, with blood pressure and proteinuria measurements used for diagnosis. Electrocardiograms (ECGs) were analyzed for the presence of LVH based on Sokolow-Lyon criteria. The results revealed a strong correlation between preeclampsia and LVH, with 42.9% of preeclampsia patients showing LVH. Blood pressure exceeding 140/90 mmHg and proteinuria were found to significantly increase the prevalence of LVH, with 60% of preeclampsia women exhibiting LVH. Statistical analysis revealed a significant positive correlation between preeclampsia and LVH ( $r=0.401$ ,  $p=0.021$ ) and a strong negative correlation between preeclampsia and blood pressure ( $r=-0.896$ ,  $p<0.05$ ). These findings highlight the role of hypertension in cardiac remodeling during preeclampsia and suggest that women with a history of preeclampsia may be at greater risk for long-term cardiovascular complications. Further studies are needed to understand these alterations' mechanisms and develop preventive and therapeutic strategies to improve maternal health outcomes.

**Keywords;** Preeclampsia, blood pressure, left ventricular hypertrophy, Pregnancy, Proteinuria, Systolic, Diastolic.

Received 11 Dec., 2024; Revised 20 Dec., 2024; Accepted 23 Dec., 2024 © The author(s) 2024.

Published with open access at [www.questjournals.org](http://www.questjournals.org)

### I. INTRODUCTION

Preeclampsia is a significant hypertensive disorder that occurs during pregnancy, characterized by the onset of hypertension and proteinuria after the 20th week of gestation. It affects approximately 5-8% of pregnancies and is associated with increased maternal and fetal morbidity and mortality (Fox et al., 2019; Ramos et al., 2017). The pathophysiology of preeclampsia involves a combination of placental dysfunction,

systemic inflammation, and endothelial dysfunction, leading to widespread cardiovascular consequences (Phipps et al., 2019; Torres-Torres et al., 2024).

One of preeclampsia's most critical cardiovascular alterations is the left ventricular (LV) structure and function change. Initially, the body responds to the increased metabolic demands of pregnancy by adapting to cardiovascular changes, including increased blood volume and cardiac output. However, the development of preeclampsia disrupts these adaptations, leading to persistent hypertension and altered loading conditions, which stress the heart (Melchiorre et al., 2011; Monika and Rutherford, 2014; Veerbeek et al., 2015).

Research has demonstrated that women with preeclampsia often exhibit significant remodeling of the left ventricle. This remodeling can manifest as hypertrophy due to increased afterload from high blood pressure and changes in diastolic function (Simmons et al., 2002; Thayaparan et al., 2019). LV hypertrophy, identified through echocardiography, is observed in many preeclampsia patients and is considered a compensatory response to elevated systemic vascular resistance. However, prolonged exposure to these conditions can result in adverse outcomes, such as heart failure and diastolic dysfunction (Agabiti-Rosei et al., 2006; Możdżan et al., 2013).

A study by Soma-Pillay et al. (2018) noted that women with preeclampsia had significant alterations in LV geometry, specifically an increase in wall thickness and mass, compared to normotensive pregnant controls. These structural changes are crucial, as they have been associated with an increased risk of cardiovascular disease later in life, even after pregnancy (Regitz-Zagrosek et al., 2016; Kotit and Yacoub, 2021).

Moreover, impaired relaxation of the left ventricle has been reported in preeclamptic women, suggesting a deterioration in diastolic function (Muthyala et al., 2016). Such diastolic dysfunction can significantly affect exercise capacity and overall cardiovascular health. The long-term implications are supported by studies indicating that women with a history of preeclampsia have a higher likelihood of developing hypertension and cardiovascular disease in the years following pregnancy (Turbeville and Sasser, 2020; Haßdenteufel et al., 2023).

The exact mechanisms by which preeclampsia leads to left ventricular (LV) remodeling and long-term cardiovascular complications are not yet fully understood. Additionally, the absence of effective preventive and therapeutic strategies leaves many women at risk of developing chronic cardiovascular diseases. Bridging this knowledge gap is crucial for enhancing maternal health outcomes and reducing the prevalence of cardiovascular conditions in this population. Findings from this research could contribute to developing clinical guidelines that prioritize comprehensive cardiac monitoring for women with a history of preeclampsia.

## II. MATERIALS AND METHOD

### Research Design

This study was designed to use human subjects. The subjects were a prospective cohort study of pregnant women attending the antenatal clinic at the obstetrics and gynecology department of Alex Ekwueme Federal University Teaching Hospital, Abakaliki, Ebonyi state (AEFUTHA). A total of 33 preeclampsia pregnant women were used.

### Sample Size

Sample size calculation for a cross-sectional of this study was adopted by Olafimhan et al, (2020).

Sample size =  $Z(1-\alpha/2)2 SD^2$  (10% attrition rate) $d^2$

Where  $Z_{1-\alpha/2}$  = is the standard normal variance

SD = standard deviation of variables.

d = absolute error or precision

### Ethical approval

Ethical approval was obtained from the ethics committee of AEFUTHA, with the ethical number NHREC/16/05/22/217. Patients gave written informed consent during the initial antenatal clinic visit.

### Experimental Procedures

A total of 33 Pregnant subjects attending Alex Ekwueme Federal Teaching Hospital, Abakaliki were diagnosed with preeclampsia if they presented with an elevated blood pressure of (>140mmHg systolic or >90mmHg diastolic) obtained on two assessments of at least 6hr apart, with them testing positive to the presence of proteinuria (Brown et al, 2018). A qualitative dipstick reading on two samples after 20 weeks of gestation defined the presence of proteinuria.

An electrocardiograph was carried out on both normotensive pregnant and hypertensive pregnant women. All ECGs were obtained with SCHILLER AT-2 plus ECG Analysis System utilizing the standard 12-lead system and were operated by trained medical personnel. A cardiology fellow interpreted all ECGs and

diagnosed LVH (cardiology LVH). We compared the ECG having computer readings suggestive of LVH (computer LVH) with those having cardiology readings with LVH (cardiology LVH). LVH is identified by ECG when at least one voltage and one non-voltage criterion in the following lists are met using Sokolow-Lyon Criteria:

**Blood Pressure Measurement**

Maternal arterial blood pressure was measured using a Non-invasive monitor (NIBP) blood pressure machine. For accurate measurement, the cuff is bound to the arm at the same where the brachial artery is for accurate measurement. At least two similar measurements were considered necessary to calculate the mean BP values. Systolic blood pressure (SBP), diastolic blood pressure (DBP).

**Left Ventricular Hypertrophy Measurements**

The Sokolow-Lyon (V1 S wave + V5 or V6 R wave  $\geq$  35 mm or aVL R wave  $\geq$  11 mm) criteria was used for left ventricular hypertrophy in pregnant women who underwent ECG procedure (Sokolow and Lyon, 1949). This was achieved by using an Electrocardiogram Recording machine(SCHILLER AT-2 plus ECG Analysis System).

1. Voltage criteria are:

- a) R wave in V5 or V6 plus S wave in V1  $>$  35 mm (Sokolow-Lyon, 1949)
- b) R wave in aVL + S in V3  $>$  20 mm (Cornell);
- c) R wave in aVL  $>$  11 mm.

2. Non-voltage criteria are:

- a) Increased R wave peak time  $>$  50 ms in leads V5 or V6;
- b) Left axis deviation;
- c) Left atrial enlargement.

**Statistical Analysis**

Data analysis was performed using IBM SPSS Statistics software, version 26.0. Results were presented as percentages, and the correlation between preeclampsia, the presence of left ventricular hypertrophy (LVH), and blood pressure was assessed using the chi-square test. Statistical significance was determined at a two-tailed p-value of  $<$ 0.05.

**III. RESULTS**

**Table 1; Percentage Evaluation % within the Presence of LVH and BP above 140/90 with LVH among Subjects (n=33)**

Presence of Preeclampsia		Presence of left ventricular hypertrophy		Total Count
		Hypertrophy (%)	No hypertrophy (%)	
Preeclampsia	% within the presence of LVH	42.9%	57.1%	7
	% within presences of BP above 140/90 and positive proteinuria	60.0%	14.3%	
	% total	9.1%	12.1%	
No preeclampsia	% within the presence of preeclampsia	7.7%	92.3%	26
	% within the presence of left ventricular hypertrophy	40.0%	85.75%	
	% total	6.1%	72.75%	

\*LVH; Left Ventricular Hypertrophy, BP; Blood Pressure

Blood pressure (BP) exceeding 140/90 mmHg and proteinuria are critical indicators of preeclampsia. Among patients with elevated BP and proteinuria, 60.0% exhibited left ventricular hypertrophy (LVH), contrasting to just 14.3% in those without these conditions. This finding underscores the strong association between high BP, cardiac remodeling, and preeclampsia. In preeclampsia patients, 42.9% presented with LVH, while 57.1% did not show structural cardiac changes, indicating a notable prevalence of LVH. In contrast, only 7.7% of normotensive pregnancies exhibited LVH, with 92.3% showing no signs of cardiac remodeling. This significant difference highlights the crucial role of hypertension in LVH development during pregnancy.

**Table 2; Percentage Evaluation within presences of BP above 140/90 and positive proteinuria among systolic groups (N=33)**

Systolic group		Presence of left ventricular hypertrophy		Total Count/Expected Count
		Preeclampsia (%)	No preeclampsia (%)	
Normal BP	% within the systolic group	5.9%	94.1%	17
	% within presences of BP above 140/90 and positive proteinuria	20.0%	57.1%	
	% of Total	3.0%	48.5%	
High BP	% within the systolic group	80.0%	20.0%	5
	% within presences of BP above 140/90 and positive proteinuria	80.0%	3.6%	
	% of Total	12.1%	3.0%	
Low BP	% within the systolic group	0.0%	100.0%	11
	% within presences of BP above 140/90 and positive proteinuria	0.0%	39.3%	
	% of Total	0.0%	33.3%	

The percentage evaluation of blood pressure (BP) across systolic groups provides insight into the association between systolic BP levels, preeclampsia, and left ventricular hypertrophy (LVH) as shown in Table 2 evaluates systolic BP groups and their relationship with preeclampsia and left ventricular hypertrophy (LVH). Normal BP Group, this group indicates that 5.9% of patients had preeclampsia, while 94.1% did not. Among patients with BP above 140/90 mmHg and proteinuria, 20.0% of preeclampsia cases and 57.1% of non-preeclampsia cases were in the normal BP category. In total, 3.0% of preeclampsia cases and 48.5% of non-preeclampsia cases fell into this group, indicating that normotension is associated with a lower risk of preeclampsia and cardiac remodeling. The high BP Group shows that in the high BP group, 80.0% of patients had preeclampsia, while 20.0% did not. Among those with BP above 140/90 mmHg and proteinuria, 80.0% of preeclampsia cases and only 3.6% of non-preeclampsia cases were in this category. Overall, this group accounted for 12.1% of preeclampsia cases but only 3.0% of non-preeclampsia cases, emphasizing the strong association between elevated BP, preeclampsia, and cardiac stress. In the low BP group, 0.0% of patients had preeclampsia, while 100.0% were non-preeclampsia. No preeclampsia cases fell into this category, though 39.3% of non-preeclampsia cases with BP above 140/90 mmHg were in this group. In total, 33.3% of non-preeclampsia cases exhibited low BP, suggesting that low BP may be protective against preeclampsia and LVH.

**Table 3; Percentage Evaluation within presences of BP above 140/90 and Positive Proteinuria among Diastolic groups (N=33)**

Diastolic group		Presence of left ventricular hypertrophy		Total Count/Expected Count
		Preeclampsia (%)	No preeclampsia (%)	
normal	% within the diastolic group	0.0%	100.0%	16
	% within presences of BP above 140/90 and positive proteinuria	0.0%	57.1%	
	% of Total	0.0%	48.5%	
High	% within the diastolic group	62.5%	37.5%	8
	% within presences of BP above 140/90 and positive proteinuria	100.0%	10.7%	
	% of Total	12.1%	3.0%	
Low	% within the diastolic group	1.4%	7.6%	9
	% within presences of BP above 140/90 and positive proteinuria	0.0%	100.0%	
	% of Total	0.0%	27.3%	

Table 3; evaluates diastolic BP levels to preeclampsia and left ventricular hypertrophy (LVH). The normal diastolic BP group shows no patients (0.0%) with normal diastolic BP had preeclampsia, while 100.0% did not. Among cases with BP above 140/90 mmHg and proteinuria, 0.0% of preeclampsia cases and 57.1% of non-preeclampsia cases fell into this group. 0.0% of preeclampsia cases and 48.5% of non-preeclampsia cases were

associated with normal diastolic BP, indicating its protective role against preeclampsia and LVH. High diastolic BP group shows, that 62.5% of patients had preeclampsia, while 37.5% did not. Among those with BP above 140/90 mmHg and proteinuria, 100.0% of preeclampsia cases and 10.7% of non-preeclampsia cases were in the high diastolic BP group. This group accounted for 12.1% of preeclampsia cases and 3.0% of non-preeclampsia cases, highlighting the strong link between elevated diastolic BP and preeclampsia. The low diastolic BP group shows that only 1.4% of patients with low diastolic BP had preeclampsia, while 7.6% did not. Among cases with BP above 140/90 mmHg, no preeclampsia cases and 100.0% of non-preeclampsia cases were in this group. Low BP accounted for 0.0% of preeclampsia cases and 27.3% of non-preeclampsia cases, suggesting its protective role against preeclampsia and LVH.

**Table 4; Percentage Evaluation within presences of LVH among Systolic groups (N=33)**

systolic group		Presence of left ventricular hypertrophy		Total Count/Expected Count
		Preeclampsia (%)	No preeclampsia (%)	
normal	% of the systolic group	17.6%	82.4%	17
	% within the presence of LVH	42.9%	53.8%	
High	% of the systolic group	60.0%	40.0%	5
	% within the presence of LVH	42.9%	7.7%	
Low	% of the systolic group	90.9%	100.0%	11
	% within the presence of LVH	38.5%	33.3%	

Table 4; examines the prevalence of left ventricular hypertrophy (LVH) across different systolic BP groups and their association with preeclampsia. The normal systolic BP group shows that among patients with normal diastolic BP, 17.6% had preeclampsia, while 82.4% did not. LVH occurred in 42.9% of preeclampsia and 53.8% of non-preeclampsia cases. This suggests subclinical cardiac remodeling in preeclampsia despite normal BP. The high systolic BP group shows that 60.0% had preeclampsia, and 40.0% did not. LVH was present in 42.9% of preeclampsia and 7.7% of non-preeclampsia cases, showing a strong link between high BP, preeclampsia, and LVH risk. Low systolic BP group: Here, 90.9% with low BP had preeclampsia, compared to 100.0% without it. LVH occurred in 38.5% of preeclampsia and 33.3% of non-preeclampsia cases, indicating lower cardiac remodeling risk with low BP.

**Table 5; Percentage Evaluation within presences of LVH among Diastolic groups (N=33)**

Diastolic group		Presence of left ventricular hypertrophy		Total Count/Expected Count
		Preeclampsia (%)	No preeclampsia (%)	
normal	% of the diastolic group	18.8%	81.3%	16
	% within the presence of LVH	42.9%	50.0%	
High	% of the diastolic group	62.5%	37.5%	8
	% within the presence of LVH	100.0%	10.7%	
Low	% of the diastolic group	1.4%	7.6%	9
	% within the presence of LVH	0.0%	100.0%	

Table 5 highlights the distribution of left ventricular hypertrophy (LVH) across diastolic BP groups with preeclampsia. The normal diastolic BP group shows that 18.8% had preeclampsia, and 81.3% did not. LVH was present in 42.9% of preeclampsia cases and 50.0% of non-preeclampsia cases. Despite being more prevalent in non-preeclampsia patients, a notable portion of preeclampsia cases with normal BP displayed LVH, suggesting subclinical cardiac remodeling. The high diastolic BP group showed the strongest association between high diastolic BP, preeclampsia, and LVH. Among patients, 62.5% had preeclampsia, while 37.5% did not. Notably, all preeclampsia cases (100%) exhibited LVH, compared to just 10.7% of non-preeclampsia cases. This emphasizes the heightened risk of cardiac remodeling in preeclampsia with elevated BP. Low diastolic BP group, here, 1.4% of preeclampsia and 7.6% of non-preeclampsia patients were identified. None of the preeclampsia patients showed LVH, while 100.0% of non-preeclampsia cases in this group had LVH. This finding suggests a potential protective role of low diastolic BP against severe cardiac remodeling in preeclampsia.

**Table 6;Correlation between the Preeclampsia and the Presence of Left Ventricular Hypertrophy to Blood Pressure**

Variables (N=33)	Presence of LVH r (p-value)	Presence of preeclampsia r (p-value)	Presence of blood pressure r (p-value)	X <sup>2</sup> (p-value)
Presence of LVH	1.000	0.401 (p=0.021)	-0.332 (p=0.059)	3.636 (p=0.093)
Presence of preeclampsia	0.401 (p=0.021)	1.000	-0.896 (p=0.000)	3.636 (p=0.093)
Presence of blood pressure	-0.332 (p=0.059)	-0.896 (p=0.000)	1.000	

P value is significant at a level of two-tailed.

Table 6 demonstrates a moderate, significant positive correlation between left ventricular hypertrophy (LVH) and preeclampsia in pregnant women, with  $r=0.401$  and  $p<0.05$ . However, the correlation between LVH and blood pressure was weak and not statistically significant ( $r=-0.332$ ,  $p>0.05$ ). In contrast, the relationship between preeclampsia and blood pressure showed a strong, significant negative correlation ( $r=-0.896$ ,  $p<0.05$ ). The chi-square analysis ( $X^2=3.636$ ,  $p>0.05$ ) did not reveal any significant association between the variables.

#### IV. DISCUSSION

Pregnancy requires significant cardiovascular adaptations, including increased blood volume and cardiac output, to meet the demands of fetal development. However, in preeclampsia, these adaptations become maladaptive, resulting in heightened systemic vascular resistance, endothelial dysfunction, and increased cardiac workload. These changes profoundly impact the cardiovascular system, particularly the left ventricle (LV). This study examines the interplay between preeclampsia, left ventricular hypertrophy (LVH), and maternal blood pressure (BP), providing insights into the mechanisms and consequences of preeclampsia-induced cardiovascular remodeling.

The results reveal that preeclampsia significantly alters LV structure. Among preeclampsia women, 42.9% exhibited LVH, as identified via electrocardiogram (ECG), compared to only 7.7% of normotensive pregnant women. This stark contrast underscores the role of preeclampsia-induced hypertension in driving LV remodeling. These findings are consistent with earlier studies (Bijnes et al., 2009; Sengodan et al., 2017; Nammour et al., 2021), which reported a strong association between preeclampsia and increased LV wall thickness and lumen size.

Elevated systemic vascular resistance, a hallmark of preeclampsia, forces the heart to compensate by increasing wall thickness to maintain cardiac output. Studies have suggested that preeclampsia induces significant LV geometric changes, supporting this study's findings. LVH, while initially compensatory, often becomes pathological, predisposing affected women to long-term cardiovascular risks (Cuspidi et al., 2011; Melchiorre et al., 2011b; Zhang et al., 2014).

The study highlights the critical influence of blood pressure on LVH development. Among preeclamptic women with systolic BP >140 mmHg or diastolic BP >90 mmHg, 80.0% exhibited LVH. Notably, 100% of preeclamptic women with elevated diastolic BP displayed LVH, reinforcing the direct relationship between hypertension and LV remodeling. These findings corroborate earlier work by Cuspidi et al. (2019), which linked sustained hypertension in preeclampsia to myocardial structural changes.

Conversely, preeclamptic women with normal BP demonstrated a lower incidence of LVH (42.9%). This suggests that while hypertension is a key driver of LV remodeling, other mechanisms, such as endothelial dysfunction and inflammatory responses, also contribute. Elevated anti-angiogenic factors like soluble fms-like tyrosine kinase 1 (sFlt-1) disrupt vascular remodeling, exacerbate vasoconstriction, and increase cardiac afterload (Possomato-Vieira and Khalil, 2016).

Impaired diastolic function is another critical consequence of preeclampsia-induced LV remodeling. This study observed that preeclampsia women frequently exhibited signs of diastolic dysfunction, including impaired relaxation and increased filling pressures. These findings align with Soma-Pillay et al. (2018), who reported similar impairments in myocardial compliance among preeclampsia women. Diastolic dysfunction reduces ventricular filling, cardiac output, and exercise capacity, contributing to maternal morbidity. It is also a precursor to heart failure with preserved ejection fraction (HFpEF), as highlighted by Muthyala et al. (2016). This underscores the long-term cardiovascular risks faced by women with preeclampsia.

The cardiovascular alterations associated with preeclampsia are not limited to pregnancy but persist postpartum, posing significant long-term risks. Melchiorre et al. (2011b) reported that women with a history of preeclampsia exhibit persistent LV hypertrophy and altered geometry, predisposing them to heart failure, hypertension, and coronary artery disease. These findings are echoed by White et al. (2016), who emphasized the heightened lifetime risk of cardiovascular diseases in this population.

Subclinical LV remodeling in preeclampsia women, even those with normal BP, further underscores the need for vigilant postpartum monitoring. Persistent myocardial changes highlight the importance of addressing preeclampsia not only as a pregnancy complication but also as a predictor of future cardiovascular disease.

## V. Conclusion

The results of this study emphasize the significant cardiovascular alterations that occur in preeclampsia women, particularly in terms of left ventricular hypertrophy and diastolic dysfunction. Elevated blood pressure, particularly systolic and diastolic hypertension, plays a critical role in inducing these structural changes, which may predispose women to long-term cardiovascular diseases. These findings are consistent with existing literature and highlight the importance of early detection and management of preeclampsia to prevent cardiovascular complications both during pregnancy and later in life. Future studies focusing on the mechanisms underlying LV remodeling in preeclampsia and exploring effective preventive and therapeutic strategies are crucial for improving maternal cardiovascular health outcomes.

## Conflict of Interest

The research was not sponsored by any research institution or company and was free with no conflict of interest among authors.

## Acknowledgements

## References

- [1]. Agabiti-Rosei E, Muiesan ML, Salvetti M., (2006). Evaluation of subclinical target organ damage for risk assessment and treatment in the hypertensive patients: left ventricular hypertrophy. *The Journal of the American Society of Nephrology*, (4 Suppl 2):S104-8.
- [2]. Brown, M.A., Magee L. A., Kenny L. C., Karumanchi S. A., McCarthy F. P., Saito S., Hall D. R., Warren. C. R., Adoyi G., Ishaku S., (2018). Hypertensive Disorders of Pregnancy: ISSHP Classification, Diagnosis, and Management Recommendations for International Practice. *Hypertension*, 72(1): p. 24-43.
- [3]. Bijnes BH, Cikes M, Claus P, Sutherland GR., (2009). Velocity and deformation imaging for the assessment of myocardial dysfunction. *European Journal of Echocardiography*, 10:216-226.
- [4]. Cuspidi C, Facchetti R, Bombelli M, Tadic M, Sala C, Grassi G, et al., (2019). High normal blood pressure and left ventricular hypertrophy echocardiographic findings from the PAMELA Population. *Hypertension*, 73(3):612-9.
- [5]. Cuspidi C, Sala C, Negri F, Mancia G, Morganti A., (2012). Italian society of hypertension. Prevalence of left-ventricular hypertrophy in hypertension: an updated review of echocardiographic studies. *Journal of Human Hypertension*, 26(6):343-9.
- [6]. Fox R, Kitt J, Leeson P, Aye CYL, Lewandowski AJ., (2019). Preeclampsia: Risk Factors, Diagnosis, Management, and the Cardiovascular Impact on the Offspring. *Journal of Clinical Medicine*, 4;8(10):1625.
- [7]. Gubner R, Ungerleider HE: Electrocardiographic criteria of left ventricular hypertrophy. *Archives of Internal Medicine*, 72:196, 1943
- [8]. Haßdenteufel K, Müller M, Gutsfeld R, Goetz M, Bauer A, Wallwiener M, Brucker SY, Joos S, Colombo MG, Hawighorst-Knapstein S, Chaudhuri A, Kirtschig G, Saalman F, Wallwiener S., (2023). Long-term effects of preeclampsia on maternal cardiovascular health and postpartum utilization of primary care: an observational claims data study. *Archives of Gynecology and Obstetrics*, 307(1):275-284.
- [9]. Kotit S, and Yacoub M., (2021). Cardiovascular adverse events in pregnancy: A global perspective. *Global Cardiology Science & Practice*, 2021(1):e202105.
- [10]. Melchiorre K, Sutherland GR, Baltabaeva A, Liberati M, Thilaganathan B., (2011a). Maternal cardiac dysfunction and remodeling in women with preeclampsia at term. *Hypertension*, 57(1):85-93.
- [11]. Melchiorre K, Sutherland GR, Liberati M, Thilaganathan B., (2011b). Preeclampsia is associated with persistent postpartum cardiovascular impairment. *Hypertension*, 58:709-715.
- [12]. Monika S, and Rutherford John D., (2014). Cardiovascular physiology of pregnancy. *Circulation*, 130(12):1003-8.
- [13]. Mozdzan M, Wierzbowska-Drabik K, Kurpesa M, Trzos E, Rechciński T, Broncel M, Kasprzak JD., (2013) Echocardiographic indices of left ventricular hypertrophy and diastolic function in hypertensive patients with preserved LVEF classified as dippers and non-dippers. *Archives of Medical Science*, 9(2):268-75.
- [14]. Muthyala T, Mehrotra S, Sikka P, Suri V., (2016). Maternal Cardiac Diastolic Dysfunction by Doppler Echocardiography in Women with Preeclampsia. *Journal of Clinical and Diagnostic Research*, 10(8):QC01-3.
- [15]. Nammour AY, El-amry MH, Attia AI, et al., (2021). Assessment of Maternal Left Ventricular Dysfunction and Remodeling in Gestational Hypertension. *Cardiology & Vascular Research*, 5(3): 1-5.
- [16]. Olafimihan V. I, Adekinle J. A, Ademola E, Olusanya A (2020). Maternal factors influencing birth weight of term babies among women who received antenatal care at a Nigerian voluntary agency health care facility. *International Journal of Reproductive and Contraceptive, Obstetric Gynecology*, 9(11): 4374-4381.
- [17]. Phipps EA, Thadhani R, Benzing T, Karumanchi SA., (2019). Pre-eclampsia: pathogenesis, novel diagnostics and therapies. *Nature Reviews Nephrology*, 15(5):275-289.
- [18]. Possomato-Vieira JS and Khalil RA., (2016). Mechanisms of Endothelial Dysfunction in Hypertensive Pregnancy and Preeclampsia. *Advances in Pharmacological*, 77:361-431.
- [19]. Rautaharju PM, La Croix AZ, Savage DD, et al: Electrocardiographic estimate of left ventricular mass versus radiographic cardiac size and the risk of cardiovascular disease mortality in the epidemiologic follow-up of the first National Health and Nutrition Examination Survey. *American Journal of Cardiology*, 62:59, 1988
- [20]. Ramos JGL, Sass N, Costa SHM., (2017). Preeclampsia. *Revista Brasileira de Ginecologia e Obstetrícia*, 39(9):496-512.
- [21]. Reichek N, Devereux RB: Left ventricular hypertrophy: relation of anatomic, echocardiographic and electrocardiographic findings. *Circulation*, 63: 139 1, 1981
- [22]. Sengodan SS, Dhanapal M, Pandian A., (2017). Left ventricular dysfunction in preeclampsia: an echocardiographic study. *International Journal of Reproduction, Contraception, Obstetrics and Gynecology*, 6:4895-8.

- [23]. Simmons, Lisa A., Gillin, Adrian G., and Jeremy, Richmond W., (2002). Structural and functional changes in the left ventricle during normotensive and preeclamptic pregnancy. *American Journal of Physiology*, Accessed on 12<sup>th</sup> October 2024
- [24]. Sokolow M, Lyon TP: The ventricular complex in left ventricular hypertrophy as obtained by unipolar precordial and limb leads. *American Heart Journal*, 37: 161, 1949
- [25]. Soma-Pillay P, Louw MC, Adeyemo AO, Makin J, Pattinson RC., (2018). Cardiac diastolic function after recovery from pre-eclampsia. *Cardiovascular Journal of Africa*, 29(1):26-31.
- [26]. Thayaparan AS, Said JM, Lowe SA, McLean A, Yang Y., (2019). Pre-eclampsia and long-term cardiac dysfunction: A review of asymptomatic cardiac changes existing well beyond the post-partum period. *The Australasian Journal of Ultrasound in Medicine*, 22(4):234-244.
- [27]. Torres-Torres, J., Espino-y-Sosa, S., Martínez-Portilla, R., Borboa-Olivares, H., Estrada-Gutierrez, G., Acevedo-Gallegos, S., Ruiz-Ramirez, E., Velasco-Espin, M., Cerda-Flores, P., Ramirez-Gonzalez, A., & Rojas-Zepeda, L. (2024). A Narrative Review on the Pathophysiology of Preeclampsia. *International Journal of Molecular Sciences*, 25(14), 7569.
- [28]. Turbeville HR and Sasser JM., (2020). Preeclampsia beyond pregnancy: long-term consequences for mother and child. *American Journal of Physiology-Renal Physiology*, 318(6):F1315-F1326.
- [29]. Regitz-Zagrosek V, Oertelt-Prigione S, Prescott E, Franconi F, Gerds E, Foryst-Ludwig A, Maas AH, Kautzky-Willer A, Knappe-Wegner D, Kintscher U, Ladwig KH, Schenck-Gustafsson K, Stangl V., (2016). Gender in cardiovascular diseases: impact on clinical manifestations, management, and outcomes. *European Heart Journal*, 1;37(1):24-34. doi:
- [30]. Veerbeek JHW, Hermes W, Breimer AY, van Rijn BB, Koenen SV, Mol BW, et al., (2015). Cardiovascular disease risk factors after early-onset preeclampsia, late-onset preeclampsia, and pregnancy-induced hypertension. *Hypertension*, 65(3):600–6.
- [31]. White WM, Mielke MM, Araoz PA, Lahr BD, Bailey KR, Jayachandran M, Miller VM, Garovic VD., (2016). A history of preeclampsia is associated with a risk for coronary artery calcification 3 decades later. *American Journal Obstetrics Gynecology*, 214(4):519.e1-519.e8.
- [32]. Zhang, Z., Li, L., Zhang, Z. et al., (2024). Electrocardiographic tracking of left ventricular hypertrophy in hypertension: incidence and prognostic outcomes from the SPRINT trial. *Journal of Clinical Hypertension*, 30, 17.