ORIGINAL RESEARCH PAPER
Volume - 14 | Issue - 03 | March - 2025 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

ECHOCARDIOGRAPHIC ALTERATIONS IN PREGNANCY INDUCED HYPERTENSIVE PATIENT ATTENDING RURAL TERTIARY CARE HOSPITAL-DBVPRMC, LONI.

Anaesthesiology

Dr. Tushar D. Bhavar — Professor, Department Of Anesthesiology & Critical Care, Dr. Balasaheb Vikhe Patil Rural Medical College, Pravara Institute Of Medical Sciences (deemed To Be University), Loni, Maharashtra, India.

Dr. Priyanka B. Gulve* — Associate Professor, Department Of Pharmacology, Dr. Balasaheb Vikhe Patil Rural Medical College, Pravara Institute Of Medical Sciences (deemed To Be University), Loni, Maharashtra, India. *Corresponding Author

Dr. Vaijayanti K. Badhe — Professor, Department Of Anesthesiology & Critical Care, Dr. Balasaheb Vikhe Patil Rural Medical College, Pravara Institute Of Medical Sciences (deemed To Be University), Loni, Maharashtra, India.

Dr. Mukesh K. Khunt — Resident, Department Of Anesthesiology & Critical Care, Dr. Balasaheb Vikhe Patil Rural Medical College, Pravara Institute Of Medical Sciences (deemed To Be University), Loni, Maharashtra, India.

ABSTRACT

Pregnancy-induced hypertension (PIH) is a significant contributor to maternal morbidity and mortality, affecting cardiovascular function. This study aimed to evaluate the echocardiographic and hemodynamic alterations in PIH patients at a rural tertiary care hospital. PIH patients exhibited significantly higher systolic (151.62 ± 11.58 vs. 110.4 ± 8.0 mmHg) and diastolic blood pressure (95.71 ± 7.22 vs. 70.0 ± 5.0 mmHg), mean arterial pressure (114.34 ± 8.04 vs. 83.5 ± 6.0 mmHg), and pulse rate (87.73 ± 5.83 vs. 80.0 ± 10.0 bpm). Echocardiographic assessment revealed increased left atrial size, stroke volume (77.51 ± 5.36 vs. 70.0 ± 4.5 ml), and cardiac output (6796.93 ± 618.28 vs. 5600.0 ± 500.0 ml/min), with reduced MAPSE and left ventricular ejection fraction (LVEF). Additionally, left ventricular mass and mass index were significantly elevated, suggesting hypertrophic changes. These findings highlight the critical need for routine echocardiographic evaluation in PIH patients to detect early cardiovascular changes and implement timely interventions, thereby reducing the risk of long-term cardiovascular complications.

KEYWORDS

Echocardiography, Pregnancy, Pregnancy Induced Hypertension

INTRODUCTION

Pregnancy-induced hypertension (PIH) is defined as systolic blood pressure (SBP) >140 mmHg and diastolic blood pressure (DBP) >90 mmHg. It is classified as mild (SBP 140-149 and DBP 90-99 mmHg), moderate (SBP 150-159 and DBP 100-109 mmHg) and severe (SBP > 160 and DBP> 110 mmHg). [1]

As reported by the WHO, PIH significantly contributes to mortality and morbidity in mothers, fetuses, and newborns. It ranks as the leading cause of maternal fatalities in Europe. In a retrospective analysis conducted between 2000 and 2009 at a tertiary center in India, PIH was identified as the third leading cause of maternal death. [2,3,4]

Preeclampsia is a complex condition exclusive to pregnancy that generally appears after the 20-week mark. The spiral arteries undergo inadequate remodeling, resulting in decreased placental blood flow and increased ischemia. In response to placental hypoxia, there is an elevation of soluble fms-like tyrosine kinase-1 and soluble endoglin anti-angiogenic factors in the maternal blood. [5] The underlying mechanism of pregnancy-induced hypertension (PIH) is predominantly affected by placental dysfunction. Abnormal development and operation of the placenta can trigger a chain reaction that may lead to widespread issues, including elevated blood pressure in the mother. In the initial phase of pregnancy, the placental cells known as trophoblasts penetrate the mother's spiral arteries, transforming them into vessels with a higher capacity to deliver additional blood to the placenta. In cases of PIH, this invasion often remains superficial and incomplete, resulting in inadequate remodeling of the spiral arteries. As a consequence, placental blood flow is reduced due to the persistence of high-resistance, narrow spiral arteries. A proper balance between pro-angiogenic factors and anti-angiogenic factors is essential for normal placental development. [6] Endothelial dysfunction plays a crucial role in the pathogenesis of PIH, including preeclampsia and gestational hypertension. In PIH, there is an imbalance between pro-angiogenic factors (VEGF, PlGF) and anti-angiogenic factors (sEng, sFlt-1), inhibiting angiogenesis. Reduced placental perfusion triggers ischemia-reperfusion injury, increasing reactive oxygen species (ROS) production, which damages endothelial cells, decreases nitric oxide (NO) availability, and promotes vasoconstriction. Additionally, placental ischemia induces the release of inflammatory cytokines like TNF-α and IL-6, further activating endothelial cells, increasing vascular permeability, and promoting leukocyte adhesion, exacerbating systemic inflammation and vascular dysfunction. [7]

OBSERVATION & RESULTS

Tabel -1 Demographics Of PIH Pregnant Females And Normotensive Pregnant Female

Demographics	PIH pregnant female (Mean ± SD)	Normotensive pregnant female reference value
Age	23.70 ± 4.83	26.5
Height	158.45 ± 3.55	159.2
Weight	61.55 ± 4.27	58.7
Gestational age	36.13 ± 2.35	37.5
BSA	1.64 ± 0.07	1.61
BMI	24.56 ± 1.44	23.2

Table -2 Hemodynamic Parameters Of PIH Pregnant Females And Normotensive Pregnant Female

Hemodynamic Parameters	PIH pregnant female (Mean ± SD)	Normotensive pregnant female reference value
Pulse	87.73 ± 5.83	80.0
Systolic BP	151.62 ± 11.58	110.4
Diastolic BP	95.71 ± 7.22	70.0
Map	114.34 ± 8.04	83.5

The normotensive values reported by **Loerup, L., Pullon, R.M., Birks, J. et al.** [8] are based on findings showing that the average systolic blood pressure (SBP) in healthy pregnancies is approximately 110.4 mmHg, while the average diastolic blood pressure (DBP) is about 70 mmHg and heart rate 80 beats per min.

Table -3 Echocardiographic Parameters Of PIH Pregnant Females And Normotensive Pregnant Female

Echocardiographic parameters	PIH pregnant female (Mean ± SD)	Normotensive pregnant female reference value (Mean ± SD)
LA	30.65 ± 3.25	28.5 ± 2.5
MAPSE	16.70 ± 1.68	18.5 ± 1.5
TAPSE	25.96 ± 1.59	24.0 ± 2.0

EPSS	7.83 ± 0.92	6.5 ± 0.8
LVOT	19.27 ± 1.84	20.0 ± 1.5
FS	40.84 ± 3.78	36.5 ± 3.0
LVEF	60.54 ± 3.80	65.0 ± 2.5
LA VOLUME	27.75 ± 2.05	26.0 ± 1.8
SV	77.51 ± 5.36	70.0 ± 4.5
CO	6796.93 ± 618.28	5600.0 ± 500.0
MVA	3.53 ± 0.53	3.8 ± 0.4
lv MASS	212.30 ± 17.46	190.0 ± 15.0
LV mass index	130.72 ± 12.46	110.0 ± 10.0
SI= SV/BSA	47.71 ± 3.93	43.5 ± 3.2
MCOT	0.351 ± 0.019	0.340 ± 0.015
IVRT	0.094 ± 0.007	0.080 ± 0.005
LVET	0.250 ± 0.015	0.260 ± 0.012
PASP	17.08 ± 1.21	16.0 ± 1.0

DISCUSSION

Acute pre-eclampsia is associated with significantly higher prevalence of asymptomatic abnormal global left ventricular (LV) abnormal function/geometry and myocardial injury. [9] In the research conducted by **Gogaram et al.** [10], the largest proportion of patients were aged between 23 and 27 years (35.57%), while the smallest proportion was found in those over 32 years (19.5%). According to the findings of **Hirpara et al.** [11] most women in both categories fell within the age range of 21 to 30 years. Consistent with our demographic results, patients with PIH were notably younger than their normotensive counterparts (23.70 ± 4.83 vs. 26.5 ± 3.8 years).They also had a higher weight (61.55 ± 4.27 vs. 58.7 ± 4.8 kg) and BMI (24.56 ± 1.44 vs. 23.2 ± 1.8). **Bhavadharini B, Anjana RM, Deepa M, et al.** [12] mean age of pregnant women was 27.4 ± 4 years, mean BMI was 25.1 ± 4.8 kg/m2, and mean weight gain during pregnancy was 8.8 ± 4.8 kg. Gestational age was lower in PIH patients (36.13 ± 2.35 vs. 37.5 ± 1.8 weeks), indicating a higher likelihood of preterm delivery.

In terms of hemodynamics, patients with Pregnancy-Induced Hypertension (PIH) displayed significantly elevated systolic blood pressure (151.62 ± 11.58 compared to 110.4 ± 8.0 mmHg), diastolic blood pressure (95.71 ± 7.22 versus 70.0 ± 5.0 mmHg), and mean arterial pressure (114.34 ± 8.04 versus 83.5 ± 6.0 mmHg), which indicates increased vascular resistance and hypertension. Moreover, their pulse rate was higher (87.73 ± 5.83 compared to 80.0 ± 10.0 bpm), suggesting an intensified cardiovascular reaction. The normotensive values reported by **Loerup, L., Pullon, R.M., Birks, J. et al.** [8].

Solanki et al. [13] conducted a comparable study to assess different systolic parameters in women with pre-eclampsia and those who are normotensive, discovering a statistically significant difference in mean stroke volume between the two groups examined. The stroke volume recorded for women with pre-eclampsia and normotensive individuals was 73.3 ± 14.19 ml versus 70.8 ± 3.22 ml, respectively, which aligns with our study results of 77.51 ± 5.36 ml versus 70.0 ± 4.5 ml, respectively.

The outcomes of our study (6796.93 ± 618.28 vs. 5600.0 ± 500.0) were similar to those reported by **Solanki et al** [13], who evaluated cardiac output in 40 patients. In their findings, cardiac output for pre-eclamptic patients was 6600.85 ± 4.56 ml/min, in contrast to 5600.1 ± 1.77 ml/min, which was noted to be significantly elevated. Our results also align with those of **Dennis et al.** [14], who examined systolic parameters among pre-eclamptic women and healthy pregnant individuals. In their study, 40 pre-eclamptic participants and 40 healthy pregnant women underwent Doppler echocardiography to assess cardiac output, revealing higher values in the PIH group compared to the controls (4789 ± 14 vs. 4109 ± 59 ml/min). They concluded that untreated pre-eclampsia is associated with increased cardiac output, primarily due to a rise in stroke volume.

CONCLUSION

Pre-eclampsia is a multifaceted condition that can arise unexpectedly and impact various systems, especially the cardiovascular system. [15] This research indicates that significant alterations in cardiovascular dynamics are present in individuals with preeclampsia, which can be assessed through echocardiography. Therefore, this study highlights the crucial need to recognize this group of preeclamptic patients who are at a greater risk for developing cardiovascular issues later in life by getting timely echocardiograms. [16]

REFERENCES

1. Visintin, C., Mugglestone, M. A., Almerie, M. Q., Nherera, L. M., James, D., & Walkinshaw, S. (2010). Management of hypertensive disorders during pregnancy: summary of NICE guidance. BMJ, 341.
2. WHO Recommendations for prevention and treatment of pre-eclampsia and eclampsia. (n.d.).
3. Wildman, K., & Bouvier-Colle, M. H. (2004). Maternal mortality as an indicator of obstetric care in Europe. BJOG, 111, 164–169.
4. Khumanthem, P. D., Chanam, M. S., & Samjetshabam, R. D. (2012). Maternal mortality and its causes in a tertiary center. J Obstet Gynaecol India, 62, 168–171.
5. Gluhovschi, G., Gluhovschi, A., Petrica, L., Anastasiu, D., Gluhovschi, C., & Velciov, S. (2012). Pregnancy-induced hypertension--a particular pathogenic model. Similarities with other forms of arterial hypertension. Revue Roumaine de Medecine Interne [Romanian Journal of Internal Medicine], 50(1), 71–81. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/22788095
6. Thapa, T., Sharma, S., Sigdel, D., Silwal, K., & Joshi, A. (2021). Pregnancy Induced Hypertension among pregnant women delivering in a tertiary care hospital: A descriptive cross-sectional study. JNMA; Journal of the Nepal Medical Association, 59(244), 1209–1214. doi:10.31729/jnma.6392
7. Momodu, O. A., Horner, R. D., Liu, J., Crouch, E. L., & Chen, B. K. (2023). Participation in the CenteringPregnancy program and pregnancy-induced hypertension. American Journal of Preventive Medicine, 65(3), 476–484. doi:10.1016/j.amepre.2023.04.010
8. Loerup, L., Pullon, R. M., Birks, J., Fleming, S., Mackillop, L. H., Gerry, S., & Watkinson, P. J. (2019). Trends of blood pressure and heart rate in normal pregnancies: a systematic review and meta-analysis. BMC medicine, 17(1), 167. https://doi.org/10.1186/s12916-019-1399-1
9. Melchiorre, K., Sutherland, G. R., Baltabaeva, A., Liberati, M., & Thilaganathan, B. (2011). Maternal cardiac dysfunction and remodeling in women with preeclampsia at term. Hypertension, 57(1), 85–93. doi:10.1161/HYPERTENSIONAHA.110.162321
10. Gogaram, P. H., & Misha, S. (2018). Prevalence of pregnancy induced hypertension in Churu District. Indian Journal of Basic and Applied Medical Research, 7(3), 271–276.
11. Hirpara, S., Ghevariya, R., Ghadia, P., Hada, T., & Pandit, N. (2017). Study of Risk Factors for Pregnancy Induced Hypertension (A Hospital Based Case Control Study). NJIRM, 8, 49–52.
12. Bhavadharini, B., Anjana, R. M., Deepa, M., Jayashree, G., Nrutya, S., Shobana, M., Malanda, B., Kayal, A., Belton, A., Joseph, K., Rekha, K., Uma, R., & Mohan, V. (2017). Gestational Weight Gain and Pregnancy Outcomes in Relation to Body Mass Index in Asian Indian Women. Indian journal of endocrinology and metabolism, 21(4), 588–593. https://doi.org/10.4103/ijem.IJEM_557_16.
13. Solanki, R., & Maitra, N. (2011). Echocardiographic assessment of cardiovascular hemodynamics in preeclampsia. Journal of Obstetrics and Gynaecology of India, 61(5), 519–522. doi:10.1007/s13224-011-0084-x
14. Dennis, A. T., Castro, J., Carr, C., Simmons, S., Permezel, M., & Royse, C. (2012). Haemodynamics in women with untreated pre-eclampsia. Anaesthesia, 67(10), 1105–1118. doi:10.1111/j.1365-2044.2012.07193.x
15. Shivananjiah, C., Nayak, A., & Swarup, A. (2016). Echo changes in hypertensive disorder of pregnancy. Journal of Cardiovascular Echography, 26(3), 94–96. doi:10.4103/2211-4122.187961
16. Jain, N., Verma, A., & Rajoria, L. (2016). Evaluation of echocardiographic systolic parameters in pre-eclamptics and normotensives women. Journal of Obstetrics and Gynaecology of India, 66(Suppl 1), 187–191. doi:10.1007/s13224-015-0823-5