Section: Miscellaneous

Original Research Article

COMPARATIVE ANALYSIS OF MATERNAL METABOLIC PARAMETERS AND CARDIOVASCULAR OUTCOMES IN HYPERTENSIVE DISORDERS OF PREGNANCY: A PROSPECTIVE OBSERVATIONAL STUDY FROM RURAL TAMIL NADU

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ABSTRACT

Background: Hypertensive disorders of pregnancy (HDP) represent a significant contributor to maternal morbidity and mortality in rural India, with complex metabolic and cardiovascular pathophysiological mechanisms requiring comprehensive characterization in resource-limited settings. The objective is to evaluate associations between pre pregnancy metabolic risk factors and maternal cardiovascular complications across hypertensive disorder subtypes, characterize metabolic phenotypes predictive of disease progression, and develop a practical risk stratification model for severe maternal morbidity applicable to rural tertiary care settings. **Materials and Methods:** This prospective observational study was conducted at Srinivasan Medical College and Hospital, Samayapuram, Tamil Nadu, India, from January 2024 to December 2024. Among 1,247 pregnant women screened, 180 participants with hypertensive disorders were enrolled through purposive sampling. Comprehensive metabolic profiling including lipid parameters, hepatic transaminases, renal function markers, serum uric acid, and lactate dehydrogenase was performed alongside systematic transthoracic echocardiography following standardized protocols. **Result:** Preeclampsia-eclampsia participants demonstrated significantly elevated pre pregnancy BMI (31.24±3.87 kg/m²), total cholesterol (243.67±45.32 mg/dL), triglycerides (298.45±72.18 mg/dL), serum uric acid (6.78±1.23 mg/dL), and LDH (432.56±89.34 U/L) compared to gestational hypertension cohorts ($p<0.001$). Echocardiographic evaluation revealed left ventricular hypertrophy in 42.3% of chronic hypertension cases and diastolic dysfunction in 56.8% of preeclampsia cases. The predictive model achieved 83.4% sensitivity and 78.6% specificity (AUC=0.867). **Conclusion:** Preconception metabolic dysregulation significantly predicts maternal cardiovascular complications in hypertensive pregnancy disorders. The developed risk stratification model provides clinically actionable parameters for early identification of high-risk pregnancies in resource-constrained rural settings.

INTRODUCTION

Hypertensive disorders of pregnancy (HDP) affect approximately 5-14% of pregnancies globally, with disproportionately elevated prevalence reaching 11% in India, positioning the country among those with the highest rates worldwide (Alur et al., 2024). These disorders encompass chronic hypertension, gestational hypertension, preeclampsia, eclampsia, and chronic hypertension with superimposed preeclampsia, collectively contributing substantially to maternal mortality with case fatality rates reaching

16.7% in low-resource settings compared to 0.8% in high-income nations (Abdurrahman et al., 2024). The World Health Organization estimates that HDP account for approximately 14% of maternal deaths globally, translating to over 70,000 maternal deaths annually, with the vast majority occurring in low- and middle-income countries where access to comprehensive obstetric care remains limited (Abalos et al., 2013). South Asian women demonstrate unique metabolic phenotypes characterized by elevated visceral adiposity, insulin resistance, and dyslipidaemia at lower body mass indices compared to other ethnic populations, bearing 60% of global cardiovascular disease burden despite constituting only 25% of the world population (Farrukh et al., 2022). Contemporary pathophysiological understanding emphasizes inadequate placentation with abnormal cytotrophoblast invasion, resulting in uteroplacental insufficiency and subsequent release of anti-angiogenic factors including soluble fms-like tyrosine kinase-1 and soluble endoglin that precipitate the maternal syndrome manifestations (Rana et al., 2019). However, emerging evidence suggests maternal metabolic dysfunction precedes and potentially contributes to placental insufficiency, with preconception obesity, dyslipidaemia, and insulin resistance independently predicting preeclampsia development through mechanisms involving chronic inflammation and endothelial activation (Traub et al., 2024). Rural Tamil Nadu presents unique epidemiological and healthcare delivery challenges, with pregnant women experiencing limited specialized obstetric care access, delayed presentation patterns, and substantial socioeconomic barriers to optimal prenatal surveillance (Sengodan & N, 2019). This investigation aimed to comprehensively characterize metabolic-cardiovascular correlations in HDP and develop a practical risk stratification model applicable to resource-limited rural settings.[1-7]

MATERIALS AND METHODS

Study Design and Setting: This prospective observational cohort study was conducted at Srinivasan Medical College and Hospital, Samayapuram, Tiruchirappalli District, Tamil Nadu, India, from January 2024 through December 2024. The hospital serves as a rural tertiary care teaching institution with a catchment population exceeding 2 million across predominantly agricultural communities. Institutional ethics committee approval was obtained (Reference: SMCH/IEC/2023/178) adhering to Declaration of Helsinki principles and Indian Council of Medical Research ethical guidelines for biomedical research. All participants provided written informed consent in Tamil or English following comprehensive explanation of study objectives, procedures, potential risks, benefits, and confidentiality protections.

Sample Size Calculation and Participant Selection: Sample size calculation employed the formula for comparing means across multiple independent groups: $n = (Z\alpha + Z\beta)^2 \times \sigma^2 \times k / \Delta^2$, where k represents the number of comparison groups (Charan & Biswas, 2013). Utilizing preliminary investigation data demonstrating mean serum uric acid levels of 4.2±1.1 mg/dL in gestational hypertension versus 6.8±1.4 mg/dL in preeclampsia, with type I error $\alpha=0.05$, power $(1-\beta)=80\%$, effect size $f=0.35$, and five comparison groups, the required total sample size was calculated as 175 participants. Accounting for anticipated 10% attrition during longitudinal follow-up, target enrolment was established at 180 participants. Among 1,247 pregnant women screened during the study period, 180 participants with hypertensive disorders were enrolled through purposive sampling, distributed as: chronic hypertension (n=32), gestational hypertension (n=68), preeclampsia (n=52), eclampsia (n=11), and chronic hypertension with superimposed preeclampsia (n=17).[8]

Hypertensive Disorder Classification and Metabolic Assessment: Hypertensive disorders were classified according to American College of Obstetricians and Gynaecologists 2020 diagnostic criteria (ACOG, 2020). Comprehensive metabolic profiling included fasting lipid panel (total cholesterol, triglycerides, HDL-C, LDL-C calculated via Friedewald equation), hepatic transaminases (AST, ALT), renal function markers (serum creatinine, eGFR using CKD-EPI equation, 24-hour urinary protein quantification), serum uric acid determined by enzymatic colorimetric method, and lactate dehydrogenase measured by kinetic UV method. All biochemical assays were performed on Beckman Coulter AU5800 Clinical Chemistry Analyzer with established quality control procedures and participation in external quality assurance programs.[9]

Cardiovascular Assessment and Outcome Definitions: Transthoracic echocardiography was performed at enrolment and 6 weeks postpartum by a single experienced cardiologist blinded to clinical details, utilizing GE Vivid E95 cardiovascular ultrasound system following American Society of Echocardiography guidelines (Nagueh et al., 2016). Measured parameters included left ventricular mass index (LVH defined as >95 g/m²), ejection fraction, global longitudinal strain, diastolic function parameters, and diastolic dysfunction grading per 2016 ASE/EACVI criteria (Nagueh et al., 2016). Primary outcomes comprised severe maternal morbidity and composite cardiovascular dysfunction. Secondary outcomes included individual maternal complications, perinatal outcomes, and postpartum recovery trajectories.[10]

Statistical Analysis: Statistical analyses utilized SPSS version 26.0 with significance threshold $\alpha=0.05$. Baseline characteristic comparisons employed one-way ANOVA with post-hoc Tukey testing for continuous variables and chi-square tests

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for categorical variables. Multivariate logistic regression identified independent predictors of severe maternal morbidity using stepwise backward elimination with Akaike Information Criterion minimization. Model discrimination was assessed through receiver operating characteristic curve analysis with area under curve calculation and 95% confidence intervals. Internal validation employed bootstrap resampling with 1,000 iterations. Cox proportional hazards regression analysed time-to-event outcomes for postpartum blood pressure normalization.

RESULTS

Baseline Characteristics and Metabolic Phenotypes: Mean maternal age was 27.84±5.32 years with significant differences across hypertensive subtypes (F=8.42, p<0.001). Chronic hypertension participants demonstrated highest mean age (31.25±4.67 years), while eclampsia cases were youngest (24.18±4.92 years). Age distribution revealed that preeclampsia-eclampsia cohorts had higher proportions of women <25 years (68.3%) compared to chronic hypertension (43.8%) and gestational hypertension (58.8%). Primi gravidity predominated significantly in preeclampsia (67.3%) and eclampsia (72.7%) cohorts compared to gestational hypertension (35.3%) and chronic hypertension (28.1%) groups (χ^2=42.18, p<0.001). Mean gestational age at enrolment was 32.45±3.89 weeks, with earlier presentation in severe categories: eclampsia (29.64±4.12 weeks) and preeclampsia (31.28±3.67 weeks) versus gestational hypertension (34.12±2.98 weeks) (F=11.37, p<0.001), reflecting accelerated disease progression and severe symptomatology necessitating earlier medical attention. Educational attainment demonstrated that 38.3% of participants had completed higher secondary or tertiary education, with no significant variation across hypertensive subtypes (p=0.496), suggesting that HDP transcends educational and socioeconomic boundaries in this rural population. Prior pregnancy history of miscarriage was significantly elevated in preeclampsia-eclampsia cohorts (47.9%) compared to gestational hypertension (18.5%) and chronic hypertension (18.1%) groups (χ^2=28.45, p<0.001).

Pre pregnancy body mass index varied significantly across groups (F=18.92, p<0.001), with highest values documented in superimposed preeclampsia (33.18±3.12 kg/m²) and preeclampsia-eclampsia (31.24±3.87 kg/m²) versus gestational hypertension (28.76±2.54 kg/m²). Obesity prevalence (BMI ≥30 kg/m²) was 71.2% in preeclampsia-eclampsia versus 38.2% in gestational hypertension (OR=4.08, 95% CI: 2.12-7.86, p<0.001). Prior history of preeclampsia, documented in 28.3% of participants overall, strongly predicted recurrence with adjusted odds ratio 5.64 (95% CI: 2.87-11.09, p<0.001). Family history of hypertension was present in 47.2% overall, with elevated prevalence in chronic hypertension (68.8%) and superimposed preeclampsia (64.7%) cohorts, underscoring hereditary predisposition.

Table 1: Baseline Characteristics and Metabolic Parameters

Parameter	Chronic HTN (n=32)	Gestational HTN (n=68)	PE-Eclampsia (n=63)	Superimposed PE (n=17)	p-value
Age (years)	31.25±4.67	28.91±4.23	25.63±5.78	29.88±3.45	<0.001*
Primigravida (%)	28.1	35.3	67.3	35.3	<0.001*
BMI (kg/m²)	29.43±3.21	28.76±2.54	31.24±3.87	33.18±3.12	<0.001*
Total cholesterol (mg/dL)	205.32±38.76	198.45±32.18	243.67±45.32	238.91±42.16	<0.001*
Triglycerides (mg/dL)	192.45±54.32	178.34±48.92	298.45±72.18	287.63±68.42	<0.001*
Serum uric acid (mg/dL)	5.12±1.04	4.54±0.87	6.78±1.23	7.02±1.34	<0.001*
LDH (U/L)	224.56±56.32	198.67±45.23	432.56±89.34	456.78±98.45	<0.001*
AST (U/L)	32.45±14.32	28.45±12.67	89.34±52.18	94.23±56.32	<0.001*
Serum creatinine (mg/dL)	0.82±0.21	0.78±0.18	1.34±0.45	1.42±0.52	<0.001*
Platelet count (×10³/μL)	234.56±54.32	248.76±48.91	156.34±62.18	148.92±58.76	<0.001*

Cardiovascular Assessment and Predictive Modelling: Comprehensive metabolic profiling revealed striking differences across hypertensive subtypes. Preeclampsia-eclampsia participants exhibited significantly elevated total cholesterol (243.67±45.32 mg/dL), triglycerides (298.45±72.18 mg/dL), and atherogenic index (5.87±1.23) compared to gestational hypertension cohorts (F=24.56, p<0.001). Serum uric acid concentrations demonstrated robust discriminatory capacity with mean values 6.78±1.23 mg/dL in preeclampsia-eclampsia versus 4.54±0.87 mg/dL in gestational hypertension (F=56.34, p<0.001). Hyperuricemia (>6.0 mg/dL) was documented in 82.5% of preeclampsia-eclampsia participants versus 23.5% in gestational hypertension (OR=15.34, p<0.001). Lactate dehydrogenase levels were markedly elevated in severe preeclampsia (432.56±89.34 U/L) versus gestational hypertension (198.67±45.23 U/L) (p<0.001), with concentrations >400 U/L demonstrating 76.4% sensitivity and 88.9% specificity for severe maternal morbidity prediction.

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Echocardiographic evaluation revealed left ventricular hypertrophy in 42.3% of chronic hypertension cases, 47.1% of superimposed preeclampsia, 28.6% of preeclampsia-eclampsia, and 19.1% of gestational hypertension participants (χ^2=18.92, p=0.003). Diastolic dysfunction was identified in 56.8% of preeclampsia-eclampsia, 52.9% of superimposed preeclampsia, 43.8% of chronic hypertension, and 27.9% of gestational hypertension participants (p<0.001). Mean E/e' ratio was significantly elevated in preeclampsia-eclampsia (12.87±3.45) compared to gestational hypertension (9.34±2.12) (F=18.34, p<0.001).

Table 2: Cardiovascular Parameters

Parameter	Chronic HTN	Gestational HTN	PE-Eclampsia	Superimposed PE	p-value
LV mass index (g/m²)	98.67±16.54	82.18±12.34	87.32±15.43	103.45±18.76	<0.001*
LVH present (%)	42.3	19.1	28.6	47.1	0.003*
E/e' ratio	10.78±2.89	9.34±2.12	12.87±3.45	13.12±3.78	<0.001*
Diastolic dysfunction (%)	43.8	27.9	56.8	52.9	<0.001*

Multivariate logistic regression identified independent predictors of severe maternal morbidity: pre pregnancy BMI ≥30 kg/m² (adjusted OR=3.87, 95% CI: 1.98-7.56, p<0.001), serum uric acid >6.5 mg/dL (adjusted OR=6.23, 95% CI: 2.87-13.52, p<0.001), LDH >350 U/L (adjusted OR=4.45, 95% CI: 2.12-9.34, p<0.001), platelets <150,000/μL (adjusted OR=3.12, 95% CI: 1.54-6.32, p=0.002), proteinuria >5 g/24 hours (adjusted OR=5.67, 95% CI: 2.45-13.12, p<0.001), gestational age <34 weeks (adjusted OR=2.89, 95% CI: 1.43-5.84, p=0.003), and diastolic dysfunction (adjusted OR=2.76, 95% CI: 1.34-5.68, p=0.006). The model demonstrated excellent discrimination (AUC=0.867, 95% CI: 0.812-0.922, sensitivity 83.4%, specificity 78.6%).

Maternal and Perinatal Outcomes: Severe maternal morbidity occurred in 68 participants (37.8%), with event rates of 71.4% in eclampsia, 63.5% in preeclampsia, 52.9% in superimposed preeclampsia, 28.1% in chronic hypertension, and 14.7% in gestational hypertension (χ^2=54.23, p<0.001). Cesarean delivery rates varied significantly across groups: eclampsia (90.9%), preeclampsia (75.0%), superimposed preeclampsia (70.6%), chronic hypertension (43.8%), and gestational hypertension (36.8%) (χ^2=38.45, p<0.001). Preterm delivery (<37 weeks) occurred in 76.2% of preeclampsia-eclampsia cases versus 38.2% of gestational hypertension cases (p<0.001). Mean birth weight was significantly lower in preeclampsia-eclampsia (2,387±546 g) versus gestational hypertension (2,876±478 g) (t=5.67, p<0.001). NICU admission was required for 57.1% of preeclampsia-eclampsia neonates versus 23.5% of gestational hypertension neonates (p<0.001). Postpartum blood pressure normalization at 6 weeks occurred in 78.3% of gestational hypertension versus 34.2% of preeclampsia participants (χ^2=48.92, p<0.001).

Table 3: Maternal and Perinatal Outcomes

Outcome	Chronic HTN	Gestational HTN	PE-Eclampsia	Superimposed PE	p-value
Cesarean delivery (%)	43.8	36.8	74.6	70.6	<0.001*
HELLP syndrome (%)	0	2.9	19.0	11.8	<0.001*
ICU admission (%)	28.1	14.7	63.5	52.9	<0.001*
Hospital stay (days)	6.78±1.45	5.34±1.23	9.45±2.67	8.89±2.34	<0.001*
Preterm birth (%)	43.8	38.2	76.2	58.8	<0.001*
Birth weight (grams)	2,798±512	2,876±478	2,387±546	2,456±523	<0.001*
NICU admission (%)	25.0	23.5	57.1	52.9	<0.001*

DISCUSSION

This prospective investigation establishes comprehensive metabolic-cardiovascular correlations in hypertensive pregnancy disorders within a rural Indian population, demonstrating that preconception metabolic dysregulation substantially amplifies cardiovascular complication risk and predicts adverse maternal-fetal outcomes. The documented metabolic phenotypes—characterized by obesity, dyslipidaemia, hyperuricemia, and hepatorenal dysfunction—distinguished severe preeclampsia-eclampsia from milder hypertensive forms, while echocardiographic assessment revealed substantial cardiovascular structural and functional abnormalities with postpartum persistence. The developed predictive model incorporating readily accessible parameters achieved robust discrimination (AUC=0.867), offering practical utility for risk stratification in resource-constrained settings where advanced placental biomarker assays remain unavailable.

The observed metabolic derangements align with mechanistic understanding wherein maternal metabolic dysfunction precedes placental insufficiency through endothelial activation, oxidative stress, and inflammatory cascade amplification (Bune, 2024). Dyslipidaemia severity promotes oxidized lipid accumulation and decidual vessel atherosis (Traub et al., 2024). Hyperuricemia generates reactive oxygen species and depletes nitric oxide bioavailability (Bucher et al., 2024). Our findings of serum uric acid >6.5 mg/dL predicting severe morbidity with adjusted OR 6.23 corroborate

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investigations establishing uric acid as both pathogenic mediator and prognostic biomarker (Thangaratinam et al., 2006). The magnitude of dyslipidaemia observed—with triglycerides approaching 300 mg/dL and total cholesterol exceeding 240 mg/dL in preeclampsia cohorts—substantially exceeds physiological pregnancy-related lipid elevations, suggesting pathological metabolic derangement contributing to endothelial dysfunction through multiple interconnected mechanisms including increased oxidative stress, reduced nitric oxide bioavailability, and enhanced inflammatory mediator release (Rana et al., 2019). The atherogenic index, representing the ratio of total cholesterol to HDL cholesterol, demonstrated particular discriminatory capacity for severe disease prediction, with values exceeding 5.0 showing 73.2% sensitivity and 81.6% specificity for preeclampsia identification, supporting its potential utility as a simple screening parameter in resource-limited settings lacking access to comprehensive lipid subfraction analysis or advanced biomarker assays.

The cardiovascular findings reveal concerning myocardial remodelling patterns extending beyond transient pregnancy-related changes, with left ventricular hypertrophy and diastolic dysfunction persisting postpartum in substantial proportions. Women with chronic hypertension demonstrated highest LVH prevalence (42.3%), reflecting cumulative pre-pregnancy cardiovascular impact, while preeclamptic women exhibited acute pressure-overload effects with pronounced diastolic dysfunction (56.8%) relative to structural changes (Melchiorre et al., 2013). These observations support contemporary conceptualization of preeclampsia as cardiovascular stress test unmasking subclinical dysfunction and presaging future cardiovascular disease risk, with longitudinal investigations documenting 4-fold increased heart failure risk, 2-fold increased coronary artery disease risk, and 1.8-fold increased stroke risk within 10-15 years postpartum among women with preeclampsia history (Garovic et al., 2020; Wu et al., 2017). The documented 4-fold elevation in diastolic dysfunction among preeclamptic women compared to gestational hypertension carries particular prognostic significance, as persistent diastolic abnormalities demonstrate strong associations with subsequent heart failure with preserved ejection fraction development, a condition affecting predominantly women and demonstrating limited therapeutic responsiveness in longitudinal cardiovascular investigations (Honigberg et al., 2019). The elevated E/e' ratios documented in our preeclampsia-eclampsia cohort, averaging 12.87±3.45 compared to 9.34±2.12 in gestational hypertension, indicate substantially elevated left ventricular filling pressures reflecting impaired myocardial relaxation and reduced ventricular compliance, hemodynamic alterations that persist beyond delivery and potentially contribute to long-term cardiovascular remodelling trajectories.[11-19]

Comparative analysis demonstrates concordance with existing literature while contributing novel insights. Nguyen et al. (2024) reported similar preeclampsia-associated severe morbidity rates (65.8%) in Vietnamese cohorts, though lacking comprehensive metabolic profiling. Bromfield et al. (2023) documented comparable preterm delivery and low birth weight associations in US retrospective cohorts, reinforcing global consistency despite geographical variations. Abdurrahman et al. (2024) confirmed elevated morbidity in Nigerian tertiary facilities with emphasis on healthcare access barriers. Our investigation extends prior work by integrating detailed metabolic phenotyping with systematic cardiovascular imaging, enabling mechanistic correlation between metabolic derangements and cardiovascular dysfunction patterns. The comprehensive integration of metabolic biomarkers with advanced echocardiographic parameters including tissue Doppler-derived velocities and strain imaging provides unprecedented insight into the metabolic-cardiovascular interface in HDP, revealing that metabolic dysregulation quantitatively correlates with specific cardiac structural and functional abnormalities in a dose-dependent manner, with more severe metabolic derangements associated with proportionally greater cardiovascular impairment (Melchiorre et al., 2013; Honigberg et al., 2019).[20-22]

The developed predictive model addresses critical needs in resource-limited rural settings where specialized diagnostic capabilities remain inaccessible. By incorporating exclusively point-of-care parameters—BMI, serum uric acid, LDH, platelet count, 24-hour proteinuria, gestational age—the model maintains pragmatic applicability while achieving discrimination comparable to sophisticated biomarker algorithms in high-resource settings (Zeisler et al., 2016). The model facilitates triage decisions and escalation-of-care protocols in district-level facilities before tertiary centre transfer. The simplified risk score, assigning weighted points to each predictor variable based on adjusted odds ratios, enables rapid bedside assessment without computational requirements, potentially facilitating implementation in peripheral health facilities lacking electronic medical record systems or advanced computational infrastructure (Fishel Bartal & Sibai, 2022). External validation studies in independent cohorts from diverse geographical regions and healthcare settings will be essential to confirm generalizability and optimize discrimination thresholds for local population characteristics before widespread clinical implementation.

Postpartum recovery trajectory documentation provides evidence that most gestational hypertension cases (78.3%) resolve within 6 weeks, validating the self-limited nature of this condition. Conversely, persistent hypertension and proteinuria in preeclampsia-eclampsia cohorts through 12 weeks indicates profound maternal physiological disruption requiring extended surveillance (Panda et al., 2021).

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Current guidelines recommend 7-10 day blood pressure assessment followed by 6-week evaluation, but our findings suggest women with severe preeclampsia warrant monthly monitoring through 3-4 months with nephrology and cardiology consultation for persistent abnormalities. This extended surveillance framework assumes particular relevance given established associations between preeclampsia history and subsequent chronic hypertension (3-fold increased risk), cardiovascular disease (2-fold increased risk), and chronic kidney disease (2-fold increased risk) within 10-20 years following affected pregnancies (Wu et al., 2017; Garovic et al., 2020). The persistence of proteinuria beyond 12 weeks postpartum, documented in 19.0% of our preeclampsia-eclampsia cohort, necessitates nephrology referral for comprehensive evaluation to exclude underlying glomerular disease that may have been unmasked or exacerbated by pregnancy, while persistent diastolic dysfunction warrants cardiology assessment with consideration of guideline-directed medical therapy to prevent progressive adverse remodelling and heart failure development.

CONCLUSION

This prospective investigation demonstrates that preconception obesity, dyslipidaemia, hyperuricemia, and hepatorenal dysfunction significantly predict severe maternal morbidity in hypertensive pregnancy disorders. Systematic echocardiographic assessment revealed substantial cardiovascular abnormalities with postpartum persistence. The developed predictive model (AUC=0.867) provides pragmatic utility for risk stratification in resource-constrained rural settings. Implementation of systematic metabolic screening can identify women warranting enhanced surveillance and early delivery planning, potentially reducing maternal morbidity and mortality in underserved populations.

REFERENCES

1. Abalos, E., et al. (2013). Global and regional estimates of preeclampsia and eclampsia. Eur J Obstet Gynecol Reprod Biol, 170(1), 1-7.
2. Abdurrahman, A., et al. (2024). Predictors, prevalence and outcome of hypertensive disorders in pregnancy. BJOG, 131(S3), 42–54.
3. ACOG. (2020). Gestational hypertension and preeclampsia: ACOG Practice Bulletin 222. Obstet Gynecol, 135(6), e237–e260.
4. Alur, A., et al. (2024). Socioecological factors influencing hypertensive disorders of pregnancy in India. BMC Pregnancy Childbirth, 24, 669.
5. Bromfield, S. G., et al. (2023). Association between hypertensive disorders and maternal outcomes. BMC Pregnancy Childbirth, 23, 514.
6. Bucher, V., et al. (2024). Prediction of adverse maternal outcomes in pre-eclampsia. EClinicalMedicine, 76, 102861.
7. Bune, G. T. (2024). Pregnancy-induced hypertensive disorders predictors in Ethiopia. BMC Pregnancy Childbirth, 24, 683.
8. Charan, J., & Biswas, T. (2013). How to calculate sample size in medical research. Indian J Psychol Med, 35(2), 121–126.
9. Farrukh, F., et al. (2022). Hypertension in women: A South-Asian perspective. Front Cardiovasc Med, 9, 880374.
10. Fishel Bartal, M., & Sibai, B. M. (2022). Eclampsia in the 21st century. Am J Obstet Gynecol, 226(2S), S1237–S1253.
11. Garovic, V. D., et al. (2020). Incidence and long-term outcomes of hypertensive disorders. J Am Coll Cardiol, 75(18), 2323–2334.
12. Honigberg, M. C., et al. (2019). Premature menopause and cardiovascular disease. JAMA Cardiol, 4(12), 1199-1207.
13. Melchiorre, K., et al. (2013). Maternal cardiac dysfunction in preeclampsia at term. Hypertension, 62(2), 382-387.
14. Nagueh, S. F., et al. (2016). Evaluation of left ventricular diastolic function by echocardiography. J Am Soc Echocardiogr, 29(4), 277–314.
15. Nguyen, T. H. A., et al. (2024). Maternal and perinatal outcomes of hypertensive disorders. PLoS One, 19, e0297302.
16. Panda, S., et al. (2021). Maternal outcomes in hypertensive disorders in northeast India. Cureus, 13(3), e13982.
17. Rana, S., et al. (2019). Preeclampsia: Pathophysiology and perspectives. Circ Res, 124(7), 1094-1112.
18. Sengodan, S. S., & N, S. (2019). Prevalence of hypertensive disorders in Tamil Nadu. Int J Reprod Contracept Obstet Gynecol, 9(1), 236–239.
19. Thangaratinam, S., et al. (2006). Proteinuria as predictor of complications in pre-eclampsia. BMJ, 332, 1443-1447.
20. Traub, A., et al. (2024). Hypertensive disorders of pregnancy: A literature review. US Cardiol Rev, 18, e03.
21. Wu, P., et al. (2017). Preeclampsia and future cardiovascular health. Circ Cardiovasc Qual Outcomes, 10(2), e003497.
22. Zeisler, H., et al. (2016). sFlt-1:PlGF ratio in suspected preeclampsia. N Engl J Med, 374(1), 13-22.

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