

Assessment of Cardiac Biomarkers in Pregnant Women with and without Hypertensive Disorders: A Cross-sectional Study

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ABSTRACT

Introduction: Hypertensive Disorders of Pregnancy (HDP) remain a leading cause of maternal and foetal morbidity worldwide. Given the rising burden of cardiovascular morbidity in India, there is a critical need to evaluate specific cardiac biomarkers to identify subclinical cardiac strain in regional clinical settings.

Aim: To compare the serum level of cardiac biomarkers in pregnant women with and without hypertensive disorders attending Antenatal Clinic at a Tertiary Care Hospital of Central India.

Materials and Methods: The present cross-sectional study was conducted between May 2023 to May 2024 at Hamidia Hospital a Tertiary Care Hospital associated with Gandhi Medical College, Bhopal, Madhya Pradesh, Central India. Total 312 study participants were enrolled and divided into normotensive (n=239), Gestational Hypertension (GH) (n=33) and Preeclampsia (PE) (n=40). The cardiac biomarkers such as Creatine Phosphokinase-MB (CPK-MB), Lactate Dehydrogenase (LDH), Serum Glutamic Oxaloacetic Transaminase (SGOT), High-sensitivity C-Reactive Protein (hsCRP), glucose and creatinine were measured by fully automated biochemistry analyser and Troponin-I and NT-proBNP by Enzyme Linked Immunosorbent

Assay (ELISA) method. One-way Analysis of variance (ANOVA) and Chi-square test was used for comparison of data among groups. the $p < 0.05$ was considered as significant level.

Results: No significant difference was found in age and Body Mass Index (BMI) among the groups. A significant, step-wise increase was found in the levels of multiple cardiac biomarkers as the severity escalated from GH to PE ($p < 0.001$ for all). Specifically, the median levels of NT-proBNP, a marker of ventricular wall stress, showed a dramatic surge from 29 pg/mL (Normotensive) to 40 pg/mL in GH group and a striking 155 pg/mL in the PE group. Similarly, Troponin I (myocardial injury) and CPK-MB, SGOT, and LDH (tissue damage) were all significantly elevated in the HDP groups.

Conclusion: HDP are associated with a progressive rise in cardiac and tissue injury biomarkers, with the highest levels observed in PE. The marked increase in NT-proBNP highlights significant cardiac stress and supports the potential role of these biomarkers in risk assessment and clinical monitoring. These findings advocate for the integration of specific biomarkers into routine screening to enable early risk stratification and improve maternal-foetal outcomes in HDP.

Keywords: Antenatal screening, Blood pressure, Cardiovascular disease, Dipstick, Proteinuria

INTRODUCTION

Pregnancy (Gestation) is the period between conceptions to delivery of a newborn generally completed in approximately 280 days [1]. During pregnancy many physiological changes occur in the mother's body. These changes may exacerbate a pre-existing underlying cardiovascular complication, or may predispose the women for developing new cardiovascular conditions, which are not usually observed in young healthy women [2].

Cardiovascular conditions which are an important cause for maternal morbidity and mortality, both during pregnancy and in postpartum periods, include conditions such as; Coronary Artery Disease (CAD), Pregnancy associated Myocardial Infarction (PMI), valvular diseases and peripartum cardiomyopathy [3,4]. The Pre-existing conditions which can be predisposing factors for precipitating Cardiovascular Disease (CVD) in pregnancy include hypertension, Diabetes Mellitus (DM), and Congenital Heart Diseases (CHD) [5].

The HDP complicate 3 to 10% of pregnancies globally, with increasing prevalence [6-8]. The maternal hypertensive disorder is a group of high Blood Pressure (BP) conditions. It includes PE, superimposed on chronic pre-GH and GH. Beyond maternal risks, HDP is significantly associated with long-term complications in offspring, including increased risks of CHD, hypertension, systemic vascular dysfunction, and higher BMI [9,10]. According to the

International Society for the Study of Hypertension in Pregnancy (ISSHP), PE is defined by new-onset hypertension ($\geq 140/90$ mmHg) and proteinuria or organ dysfunction after 20 weeks of gestation [11]. If untreated, PE can progress to eclampsia, characterised by seizures and coma [12,13].

When the cardiac tissues get damaged, under similar conditions of stress and modification, it releases certain substances in the blood-stream, which are referred to as cardiac biomarkers [14]. In recent years, cardiac biomarkers have taken a central place in the assessment of CVD from prediction to management and prognosis. On the other hand, enough evidence exists to assume that HDP share a certain connection with CVDs from common risk factors and underlying mechanisms to the presence of a higher risk for women for the development of a great number of CVDs [14]. The levels of cardiac biomarkers such as Cardiac Troponin-I, Creatinine Phosphokinase (CPK), CK-MB fraction, Lactate Dehydrogenase (LDH) and myoglobin constantly fluctuate with advancing gestational age. Knowledge and tracking of these variances from the normal values can be used to diagnose and clinically manage the CVD during pregnancy [15]. The increase in the risk of Myocardial Infarction (MI) has been associated with pregnancy, particularly in women with PE [16]. As high blood levels of cardiac specific Troponins (cTn) indicate MI [17], although this may be elevated in women with PE without MI [18], which increases diagnostic uncertainty. Leiserowitz

GS et al., have reported elevations in total CK and CK-MB during the peripartum period [19]. However; there is no data available from Central India on early-stage prediction of cardiac disease risk during pregnancy in pregnant women, in spite of the increasing number of the same.

Therefore, keeping this fact in view, the present study was designed to compare the serum level of cardiac biomarkers in pregnant women with and without hypertensive disorders attending antenatal clinic at a Tertiary Care Hospital of Central India.

MATERIALS AND METHODS

The present cross-sectional study was carried out in the Department of Biochemistry in collaboration with the Department of Obstetrics and Gynaecology, Gandhi Medical College, Bhopal, Madhya Pradesh, India, from May 2023 to May 2024. A written Informed Consent was taken from all study participants. The Institutional Ethics Committee (IEC letter no. 11596/MC/IEC/2020, dated-14/04/2023) approved this study.

Inclusion criteria: Second and third trimester pregnant women aged between 20-40 years, and with singleton pregnancy (*Primigravida/Multigravida*) were included in the study.

Exclusion criteria: The pregnant women who did not give consent for participation, had termination of pregnancy without indications for delivery, had history of medical complications other than HDP, had a history of prenatal CVD and HTN, had gestational diabetes, had renal disease and liver disease were excluded.

Sample size calculation: The sample size for this study was calculated based on the reported prevalence of HDP at Hamidia Hospital, Gandhi Medical College, Bhopal, Madhya Pradesh, India, which was 14.6%. Using the formula $n = Z^2 P (1-P) / d^2$ with a 95% confidence level ($Z=1.96$), a prevalence (P) of 0.146, and a precision (d) of 4%, the minimum required sample size was 300. To compensate for a potential 4% attrition rate or incomplete clinical records, the final sample size was considered at 312.

Study Procedure

Total 312 pregnant women attending anti-natal care clinic during this study period were recruitment and divided in two categories [20]: a) Pregnant women with diagnosed hypertensive disorders ((GH); BP>140/90 mmHg without proteinuria) and (PE; BP>140/90 mmHg after 20 weeks gestation with proteinuria >300mg/24 hours or >1+ dipstick); b) Pregnant women with normal BP throughout pregnancy (Normotensive).

The height, weight, Body Mass Index (BMI), Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP) noted in the case data sheet.

Laboratory investigation: Five millilitre fasting blood samples were collected in a plain vial. For the separation of serum, blood was centrifuged on 3500 RPM for 15 minutes and serum was used for the assessment of cardiac biomarkers and other routine investigations.

The cardiac biomarkers measured in this study were serum CPK-MB assessed by photometric method (Normal Range: 0-25 IU/L), serum LDH by IFCC method (Normal Range: 0-247 IU/L), serum SGOT by IFCC method (Normal Range: 0-50 IU/L), serum high sensitivity CRP by immunoturbidimetric method (Normal Range: 0-5 mg/L), serum glucose by enzymatic hexokinase method (Normal Range: 70-110 mg/dL) and creatinine by Jaffe's method (Normal Range: 0.2-1.2 mg/dL) were estimated by using fully automated biochemistry analyser (Backman Coulter 5800 and Transasia XL-1000), urine protein by dipstick method and serum Troponin-I by ELISA method (Sensitivity; 0.23 ng/mL, Detection Range; 0.39-25 ng/mL) and NT-proBNP by ELISA method (Sensitivity; 11.5 pg/mL, Detection Range; 21.9-1400 pg/mL) at the Clinical Biochemistry Laboratory and Multidisciplinary Research Unit (MRU) of Hamidia Hospital and Gandhi Medical College, Bhopal, Madhya Pradesh, India. The cut-off values for each parameter were applied according

to the manufacturer's specifications provided with the assay kits. Urine protein testing was performed solely for the diagnosis and confirmation of PE prior to enrollment of participants in the study.

STATISTICAL ANALYSIS

Epi-Info statistical software was used for the statistical analysis of data. Continue variables were checked for normality and results were expressed accordingly as Mean±SD and Median (IQR). One-way Analysis of variance (ANOVA) was used for comparison of data among groups. Chi-square test was also done. A statistical significance was considered as the $p < 0.05$.

RESULTS

This [Table/Fig-1] presents a comparative analysis of key demographic and clinical parameters across a cohort of 312 pregnant women, subdivided into three distinct clinical groups: normotensive, GH, and PE. No significant difference was found in age and BMI among the groups. A significant and sequential rise was found in both SBP and DBP from the normotensive state to GH and then to the more severe PE. No significant difference was found in fasting serum glucose suggests that impaired glucose metabolism is not a primary factor distinguishing these hypertensive conditions in this cohort. The finding regarding serum creatinine is particularly notable. While PE is often associated with renal dysfunction and elevated creatinine, the results of this study show no significant difference.

Parameter(s)	Total pregnant women (n=312)	Pregnant women with Normotensive (n=239)	Pregnant women with GH (n=33)	Pregnant women with PE (n=40)	*p-value
Age (years)	27.21±3.09	24.40±2.98	24.18±3.35	25.24± 3.00	0.987
BMI (Kg/m ²)	24.79±2.15	24.18±5.02	24.92±4.13	25.28±2.96	0.876
SBP (mmHg)	125.51±16.2	108.66±6.39	146.3±12.8	155.11±20.13	0.001
DBP (mmHg)	78.11±6.73	74.20±5.13	92.6±8.9	90.34±10.55	0.001
Fasting serum glucose (mg/dL)	84.3±8.21	86±6.67	89.12±7.76	90±4.53	0.854
Serum Creatinine (mg/dL)	0.74±0.12	0.70±0.10	0.61±0.11	0.72±0.10	0.528

[Table/Fig-1]: Demographic, clinical and biochemical characteristics of study participants. Data are presented as number (n), percentage (%) and Mean±SD *p<0.05 was significance level

Among the study cohort of 312 pregnant women, 154 (49%) were multigravida and 158 (51%) were primigravida. While the normotensive group (n=239) showed a relatively balanced distribution, the hypertensive groups exhibited a clear shift toward primigravidity. Specifically, primigravida women constituted 20 (60.6%) of the GH group and 28 (70%) of the PE group, indicating a higher prevalence of hypertensive disorders in first-time pregnancies [Table/Fig-2].

Total pregnant women (N=312)	Pregnant women with normotensive N=239 (76.6%)	Pregnant women with Gestational Hypertension (GH) N=33 (10.6%)	Pregnant women with Preeclampsia (PE) N=40 (12.8%)
Trimester			
Second trimester (N=138 (44%))	115 (48%)	10 (30.3%)	13 (32.5%)
Third trimester (N=174 (56%))	124 (52%)	23 (69.7%)	27 (67.5%)
p-value=0.01*			
Gravida			
Multigravida N=154 (49%)	129 (54%)	13 (39.3%)	12 (30%)
Primigravida N = 158 (51%)	110 (46%)	20 (60.60%)	28 (70%)
p-value=0.01*			

[Table/Fig-2]: Frequency of hypertensive disorders among pregnant women trimester and gravida-wise. Data is presented as N (%), *Chi-square test and p<0.05 was considered as significant level

The median serum troponin I level shows a progressive increase from the normotensive group (3 ng/mL) to the GH group (4 ng/mL) and is highest in the PE group (6 ng/mL). A dramatic and significant rise in the median serum NT-proBNP level is observed. The value increases from 29 pg/mL in the normotensive group to 40 pg/mL in the GH group and spikes to a very high level of 155 pg/mL in the PE group. Both markers show a highly significant difference across the groups ($p < 0.001$). Serum CPK-MB, SGOT, and LDH: The mean values for all three markers show a significant and step-wise increase from the normotensive group to the GH and then to the PE group. All these markers show a highly significant difference among the groups ($p < 0.001$ for CPK-MB, SGOT, and LDH; $p = 0.027$ for hsCRP) [Table/Fig-3].

Cardiac biomarker (s)	Pregnant women with normotensive (n=239)	Pregnant women with gestational hypertension (n=33)	Pregnant women with Preeclampsia (PE) (n=40)	p-value
Serum Troponin I (ng/mL)	3 (3, 4)	4 (3, 6)	6 (5, 8)	0.001 [§]
Serum NT-proBNP (pg/mL)	29 (19, 40)	40 (23, 63)	155 (73, 290)	0.001 [§]
Serum CPK-MB (U/L)	13.66 (4.14)	38.43 (11.78)	69.45 (19.29)	0.001 [#]
Serum SGOT (IU/L)	14.2 (4.4)	32.9 (6.5)	56.1 (12.9)	0.001 [#]
Serum LDH (U/L)	106.55 (8.09)	160.75 (37.35)	289 (98.47)	0.001 [#]
Serum hsCRP (mg/dL)	0.8 (0.5, 2.2)	2.6 (1.6, 5.2)	2.2 (0.6, 4.1)	0.027 [§]

[Table/Fig-3]: Biomarker levels in pregnant women with hypertensive disorders. Data are reported as n (%), Mean (SD), median (IQR). #Analysis of variance (ANOVA), §Kruskal-Wallis test. $p < 0.05$ was considered as significant level.

DISCUSSION

The present study reports an overall incidence of HDP of 23.4%, which is higher than that reported in earlier studies by Sengodan SS and Sreerathi N and Preet A and Anand AR [21,22]. Globally, PE is estimated to affect approximately 2-8% of pregnancies [23]; however, in the current study, the incidence of PE was higher at 10.6%, while GH accounted for 12.8% of cases. The elevated prevalence observed in this study may reflect regional variations, or delayed presentation of high-risk pregnancies, emphasising the need for region-specific data on HDP burden. Importantly, no statistically significant differences were observed in maternal age or BMI among the groups. This suggests that age and BMI were not major differentiating factors within this study.

In this study, the significant rise in both SBP and DBP from the normotensive state to GH and then to PE directly validates the clinical diagnostic criteria that define these conditions [24]. In support to this study, Sukmanee J et al., also reported increased BP in women with HDP [25]. Interestingly, the lack of significant differences in fasting serum glucose suggests that impaired glucose metabolism is not a primary factor distinguishing these hypertensive conditions in this particular study. The finding regarding serum creatinine is particularly notable. While PE is often associated with renal dysfunction and elevated creatinine [26], the results of this study show no significant difference. This study shown the distribution of pregnant women by parity and trimester offers strong evidence supporting known epidemiological trends in HDP. The stark shift in parity distribution across the groups-with primigravida women being a clear minority in the normotensive group (46%) but becoming the overwhelming majority in the GH (60.6%) and PE (70%) groups-is highly significant. This statistically confirms the well-documented phenomenon of nulliparity as a major, independent risk factor for PE [27].

Similarly, the analysis by trimester highlights the time-bound nature of HDP. Both GH (69.7%) and PE (67.5%) predominantly affect women in the third trimester, with a significantly different distribution from the more balanced normotensive group. In the present study, the median serum troponin I level shows a progressive increase

from the normotensive group to the GH group and is highest in the PE group. Ravichandran J et al., also reported elevated troponin I in PE and GH [28]. A dramatic and significant rise in the median serum NT-proBNP level is also observed. The value increases from 29 pg/mL in the normotensive group to 40 pg/mL in the GH group and spikes to a very high level of 155 pg/mL in the PE group. Nguyen TX et al also reported the statistically significantly increased NT-proBNP level in the patients with PE [29]. As both Troponin I and NT-proBNP are indicators of cardiac stress and injury so the study results demonstrate a clear and progressive increase in myocardial strain as the severity of the hypertensive disorder escalates from GH to PE. The extremely high NT-proBNP level in the PE group is a critical finding, suggesting significant cardiac remodelling and dysfunction, which aligns with the known pathophysiology of severe PE.

A highly significant increase in both Troponin I and NT-proBNP is a critical result of this study. Troponin I, an indicator of myocardial injury, progressively increases. The NT-proBNP surge is particularly dramatic in the PE group (155 pg/mL), spiking far above the levels seen in normotensive (29 pg/mL) and GH (40 pg/mL) women. This extreme elevation in NT-proBNP, a key marker of ventricular wall stress and cardiac dysfunction, is a critical finding [30]. It strongly supports the growing evidence that PE, particularly severe PE, is a state of significant cardiovascular maladaptation involving increased cardiac afterload and microvascular damage, leading to early signs of cardiac remodeling and potential heart failure [31]. The highly significant elevation of LDH, SGOT and CPK-MB, particularly in the PE group provides clear biochemical evidence of widespread cellular and tissue damage [32]. However, Preet A et al., reported LDH levels at 12-16 weeks of gestation are not early predictor of HDP and it is a prognostic factor in HDP. This finding not much supported to our findings [22]. Increases in SGOT and CPK-MB suggest multisystem involvement, corroborating the clinical picture of organ system dysfunction. Furthermore, the significant increase in the highly sensitive hsCRP, confirms that a state of low-grade, subclinical, or overt systemic inflammation is integral to the pathophysiology of both GH and PE [33]. Aziz R et al., also reported significantly elevated LDH and AST in pre-eclamptic women compared to normal pregnant women [34].

Limitation(s)

In the current study, the association between BMI and cardiac biomarker's levels has not been calculated. The other hypertensive conditions such as; chronic, eclampsia and chronic with superimposed PE were not included. The spectrum of maternal cardiac diseases and the maternal, foetal outcomes of women with cardiac diseases have also not been analysed. This study, however, still going on, but it throws light on the alarming need for large scale research to determine the effectiveness of cardiac biomarkers during pregnancy with hypertensive disorders.

CONCLUSION(S)

The present study highlights a 23.4% incidence of HDP in Bhopal, Madhya Pradesh, Central India, underscoring the urgent need for early screening. Findings suggest that elevated cardiac biomarkers, specifically Troponin-I and NT-proBNP, serve as critical indicators of cardiac dysfunction and increased cardiovascular risk in HDP patients. Integrating these biomarkers into routine antenatal care could facilitate timely intervention, potentially reducing adverse maternal and foetal outcomes. Ultimately, these results support the emerging field of cardio-obstetrics and advocate for larger-scale studies to standardize biomarker utility in managing complicated pregnancies.

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