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Original Research Article

INFLAMMATORY BIOMARKERS IN HYPERTENSIVE DISORDERS OF PREGNANCY: A HOSPITAL-BASED OBSERVATIONAL STUDY

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ABSTRACT

Background: Hypertensive disorders of pregnancy (HDP), including gestational hypertension (GH) and pre-eclampsia (PE), remain major contributors to maternal and fetal morbidity and mortality worldwide. Growing evidence indicates that exaggerated systemic inflammation and endothelial dysfunction play a pivotal role in the pathogenesis and progression of these disorders. Identification of reliable inflammatory biomarkers may aid in early detection and assessment of disease severity. **Objectives:** To evaluate the levels of inflammatory biomarkers in pregnant women with hypertensive disorders of pregnancy and to correlate these markers with disease severity. **Materials and Methods:** This hospital-based observational cross-sectional study was conducted in the Department of Medical Biochemistry in collaboration with the Department of Obstetrics and Gynaecology, Gandhi Medical College, Bhopal. A total of 100 pregnant women were enrolled, comprising 50 normotensive pregnant women, 30 women with gestational hypertension, and 20 women with pre-eclampsia. Fasting blood samples were analysed for serum high-sensitivity C-reactive protein (hs-CRP), ferritin, interleukin-6 (IL-6), lactate dehydrogenase (LDH), and erythrocyte sedimentation rate (ESR). Statistical analysis was performed using Epi-Info software. Data were expressed as mean ± SD. One-way ANOVA and Chi-square tests were applied and $p \leq 0.05$ was considered statistically significant.

Results: No significant differences were observed in age or body mass index among the groups. Systolic and diastolic blood pressures increased significantly from normotensive pregnancy to GH and PE ($p = 0.001$). Hypertensive disorders were more prevalent in the third trimester ($p = 0.01$). Levels of hs-CRP, ferritin, IL-6, ESR, and LDH were significantly elevated in GH and PE, with the highest values in pre-eclampsia. LDH, hs-CRP, and ferritin showed a strong positive association with disease severity ($p < 0.001$). **Conclusion:** HDP are associated with heightened systemic inflammation. Elevated hs-CRP, LDH, ferritin, and IL-6 correlate with disease severity and may serve as useful biomarkers for early identification and risk stratification of pre-eclampsia.

INTRODUCTION

Pregnancy is characterized by profound physiological, biochemical, and immunological adaptations that are essential for fetal growth and maternal well-being. However, complications arising during this period can pose serious risks to both the mother and the fetus. Among these, hypertensive disorders of pregnancy (HDP) represent one of the most common and clinically significant medical complications, affecting approximately 5–10% of all pregnancies worldwide.[1] HDP primarily include gestational hypertension (GH) and preeclampsia (PE) and remain a leading cause of maternal and perinatal morbidity and mortality.[2]

According to the International Society for the Study of Hypertension in Pregnancy (ISSHP), preeclampsia is defined as the new onset of hypertension (systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg) occurring at or after 20 weeks of gestation, accompanied by proteinuria and/or evidence of maternal organ dysfunction such as hepatic, renal, neurological, or haematological involvement, or uteroplacental dysfunction.[3,4] Preeclampsia complicates approximately 3–6% of pregnancies and is associated with adverse maternal

and fetal outcomes.[5] Gestational hypertension, defined as de novo hypertension after 20 weeks of gestation in the absence of proteinuria or systemic involvement, is also clinically important, as nearly 25% of women with GH-particularly those diagnosed before 34 weeks-progress to preeclampsia, resulting in poorer outcomes.[6] Severe preeclampsia is characterized by markedly elevated blood pressure (diastolic ≥110 mmHg), significant proteinuria, or features of end-organ damage, including neurological symptoms, hepatic dysfunction, renal impairment, thrombocytopenia, pulmonary edema, or seizures.[7]

The pathophysiology of preeclampsia is complex and multifactorial; however, systemic inflammation and endothelial dysfunction are recognized as central mechanisms in disease development and progression. While a controlled inflammatory response is essential for normal placentation and pregnancy maintenance, exaggerated or dysregulated maternal inflammation has been implicated in adverse pregnancy outcomes, including HDP.[8-10] Increasing evidence suggests that preeclampsia represents an amplified maternal inflammatory response to pregnancy, resulting in widespread endothelial activation and injury.[11]

Several inflammatory and biochemical biomarkers have been investigated as potential indicators of disease severity in HDP. C-reactive protein (CRP), an acute-phase reactant synthesized by the liver, serves as a sensitive marker of systemic inflammation and has been linked to endothelial dysfunction in preeclampsia. Lactate dehydrogenase (LDH), an intracellular enzyme released during cellular injury, reflects tissue damage and has been shown to correlate with disease severity in preeclampsia.[12,13] Serum ferritin, an indicator of iron stores and an acute-phase reactant, is often elevated in hypertensive pregnancies and may reflect oxidative stress and inflammatory activity, which are associated with unfavourable maternal and fetal outcomes.[14,15] Erythrocyte sedimentation rate (ESR), although non-specific, also reflects inflammatory status and has been reported to increase in hypertensive pregnancies.

Undiagnosed or inadequately managed preeclampsia can rapidly progress to eclampsia, a life-threatening condition associated with severe complications such as multi-organ failure, coagulopathies, intrauterine fetal demise, coma, and maternal mortality.[16] Early identification of women at risk and timely intervention remain critical challenges in obstetric care, particularly in resource-limited settings.

Given this background, there is a compelling need to explore readily available and cost-effective inflammatory biomarkers that may aid in the early detection and assessment of disease severity in hypertensive disorders of pregnancy. Therefore, the present study was undertaken to evaluate the levels of selected inflammatory biomarkers in pregnant women with HDP and to correlate these biomarkers with disease severity, thereby contributing to improved risk stratification and clinical management of preeclampsia.

MATERIALS AND METHODS

This hospital-based observational cross-sectional study was conducted in the Department of Medical Biochemistry in collaboration with the Department of Obstetrics and Gynaecology, Gandhi Medical College, Bhopal. The duration of study was 12 months (May 2023 to May 2024). Total of 100 pregnant women attending anti-natal care clinic during this study period were recruitment and divided into three groups: Pregnant women with diagnosed hypertensive disorders [a. Gestational hypertension (GH =30); BP>140/90 mm Hg without proteinuria) and b. Preeclampsia (PE =20); BP>140/90 mm Hg after 20 weeks gestation with proteinuria >300mg/24 hrs or >1+ dipstick)] c. Pregnant women with normal blood pressure (Normotensive =50) throughout pregnancy. The enrolment of the subjects for the study was based on the following inclusion criteria; Age between 20 and 40 years, Singleton pregnancy, Primigravida or Multigravida, Normoglycemic status and Pregnant women diagnosed with hypertensive disorders of pregnancy (for GH and PE groups). Presence of any obstetric or medical complications, fever at the time of blood sample collection, pre-existing chronic diseases such as cardiovascular disease, liver disease, renal disease, or diabetes mellitus were excluded. A written Informed Consent was taken from all study participants. The Institutional Ethics Committee (IEC letter no. 30696/MC/IEC/2022, dated- 04/08/2022), Gandhi Medical College, Bhopal was approved this study.

General and Systemic Examination: A detailed history was obtained for each participant, including gestational age, menstrual history, obstetric history, past medical history, family history, and social history. General physical examination included assessment of body temperature, pulse rate, blood pressure, and anemia, which were recorded in the case sheets. Systemic examinations of the cardiovascular system (CVS), central nervous system (CNS), and respiratory system were also performed and documented.

Anthropometric Measurements: Body mass index (BMI) was calculated using standard formula.

Sample Collection: Five millilitres of fasting venous blood was collected from each participant into a plain test tube and an EDTA vial. For the separation of serum, blood was centrifuged on 3500 RPM for 15 minutes and serum samples were separated and stored at −20°C until further biochemical analysis.

Laboratory Investigations: Routine laboratory investigations including hemoglobin estimation, erythrocyte sedimentation rate (ESR) were performed in the clinical laboratory and recorded. Serum interleukin-6 (IL-6) was measured using the sandwich enzyme-linked immunosorbent assay (ELISA) method. Serum high-sensitivity C-reactive protein (hs-CRP) was quantified using the immunoturbidimetric assay. Serum lactate dehydrogenase (LDH) and ferritin were estimated

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using a fully automated clinical chemistry analyser at the Clinical Biochemistry Laboratory and Multidisciplinary Research Unit (MRU) of Hamidia Hospital and Gandhi Medical College, Bhopal.

Statistical Analysis: Statistical analysis was performed using Epi Info software. Data were expressed as mean ± standard deviation (SD). One-way analysis of variance (ANOVA) was used to compare variables among the three groups. Data distribution was assessed for normality prior to applying ANOVA. The Chi-square test was applied for categorical variables. A p-value ≤ 0.05 was considered statistically significant.

RESULTS

Table 1: Demographic and Clinical Characteristics of Study Participants

Parameter (s)	Pregnant women with Normotensive (n=50)	Pregnant women with GH (n=30)	Pregnant women with PE (n=20)	*p-value
Age (Year)	24.40 ± 2.98	24.18 ± 3.35	25.24± 3.00	0.967
BMI (Kg/m^2)	24.18 ± 5.02	24.92 ± 4.13	25.28 ± 2.96	0.866
SBP (mmHg)	108.66±6.39	130.34±18.70	155.11±20.13	<0.001
DBP (mmHg)	74.20±5.13	82.85±9.55	90.34±10.55	<0.001
Haemoglobin (g/dL)	11.8±0.7	10.8±0.5	10.5±0.6	0.760

Data are expressed as mean ± SD. One-way analysis of variance (ANOVA) was used for comparison among groups. A p-value ≤ 0.05 was considered statistically significant.

No statistically significant difference was observed in maternal age, BMI and haemoglobin among normotensive, GH, and PE groups. However, both systolic and diastolic blood pressures showed a significant and progressive increase from normotensive pregnancy to gestational hypertension and further to preeclampsia (p<0.001), indicating worsening disease severity.

Table 2: Trimester-wise distribution of hypertensive disorders of pregnancy

Group	Second trimester n (%)	Third trimester n (%)
Normotensive (n=50)	24 (48%)	26 (52%)
Gestational hypertension (n = 30)	9 (30%)	21 (70%)
Pre-eclampsia (n =20)	6 (30%)	14 (70%)
*p <0.01		

Data are expressed as number (n) & percentage (%). Association between trimester and hypertensive disorders of pregnancy was assessed using the Chi-square test. A *p-value ≤ 0.05 was considered statistically significant.

The trimester-wise distribution demonstrated a significantly higher frequency of hypertensive disorders in the third trimester compared to the second trimester (p<0.01). This finding indicates a strong association between advancing gestational age and the development of hypertensive disorders of pregnancy. The increased prevalence in later gestation is unlikely to be due to random variation and suggests a biologically plausible progression of disease severity with gestational advancement.

Table 3: Inflammatory biomarker levels in study participants

Biomarker (s)	Pregnant women with Normotensive (n = 50)	Pregnant women with Gestational Hypertension (n = 30)	Pregnant women with pre-eclampsia (n = 20)	*p-value
Serum hsCRP (mg/L)	5.8±1.9	10.4±2.1	16.2±2.4	< 0.001
Serum Ferritin (ng/mL)	24.5±6.2	48.3±12.6	86.9±22.7	< 0.001
Serum IL-6 (pg/mL)	1.8±0.6	3.2±1.1	7.4±1.8	< 0.01
ESR (mm/hr)	25.0±8.0	42.0±12.0	58.0±15.0	< 0.05
Serum LDH (U/L)	278±90	450±125	632±189	< 0.001

Data are expressed as mean ± SD. One-way analysis of variance (ANOVA) followed by post-hoc multiple comparison test was applied. A *p-value ≤ 0.05 was considered statistically significant.

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A statistically significant stepwise increase in inflammatory and biochemical markers was observed from normotensive pregnancy to GH and PE. Serum hs-CRP, ferritin, and LDH demonstrated the strongest association with disease severity ($p<0.001$). Serum IL-6 levels were significantly elevated in GH and PE compared to normotensive pregnancy ($p<0.01$). ESR showed a modest but significant increase, reflecting the generalized inflammatory state of pregnancy. LDH, a marker of cellular injury and endothelial dysfunction, showed a marked rise in preeclampsia, highlighting its role as a potential indicator of disease progression. Overall, the progression from normotensive pregnancy to gestational hypertension and preeclampsia was characterized by increasing systemic inflammation and endothelial damage.

DISCUSSION

In the present study, maternal age, haemoglobin and BMI did not differ significantly among the study groups. These findings contrast with those reported by Sharma K et al, who observed significantly higher age and BMI in women who developed hypertension during pregnancy compared to normotensive women.[17] The trimester-wise distribution in this study demonstrated a higher prevalence of hypertensive disorders in the third trimester. In contrast, Revathi J et al. reported that hypertension was most frequently diagnosed during the second trimester, followed by the third and first trimesters.[18] These differences may be attributed to variations in study population, diagnostic criteria, and healthcare-seeking behaviour. The primary objective of this study was to assess systemic inflammation and endothelial dysfunction in hypertensive disorders of pregnancy and to evaluate the role of inflammatory markers in predicting disease severity. Serum hs-CRP levels were significantly elevated across GH and PE groups compared to normotensive pregnant women. These findings are consistent with studies conducted by Bansal P et al., Nanda et al., and Oancea et al., all of which reported significantly higher hs-CRP levels in preeclamptic women.[16,19,20] Similarly, Imaralu JO et al. reported markedly elevated hs-CRP levels in preeclamptic women compared to non-preeclamptic controls.[21] The elevation of hs-CRP in pre-eclampsia may reflect a state of subclinical inflammation, which is known to play a crucial role in disease pathogenesis. Reduced plasma volume and the influence of BMI may also contribute to increased CRP level.[11] CRP has been shown to directly affect endothelial cells by inducing adhesion molecule expression and promoting thrombogenesis through tissue factor synthesis in monocytes.[22] These mechanisms support the inflammatory hypothesis of preeclampsia.[23] However, Savvidou M et al. reported no significant association between CRP levels and maternal inflammatory response in preeclampsia, indicating ongoing debate in this area.[24] Serum ferritin levels were significantly higher in both GH and PE groups. Similar findings were reported by Mo H et al., who demonstrated a positive association between elevated ferritin levels and the risk of hypertensive disorders of pregnancy.[25] Elevated ferritin, an acute-phase reactant, may reflect oxidative stress and inflammatory activity rather than iron overload. Studies by Mukul S et al. and Akhtar N et al. further support these observations. [26,27] Serum IL-6 levels were significantly increased in hypertensive groups compared to normotensive pregnant women. These findings align with those reported by Gencheva D et al. and several other studies that demonstrated elevated IL-6 levels in gestational hypertension and preeclampsia.[28-32] However, Borekci et al. did not observe a similar association, suggesting population-specific variations.[33] Consistent with previous studies by Eleti MR et al. and Kumari N et al., serum LDH levels were significantly elevated in women with preeclampsia, underscoring its role as a marker of cellular injury and disease severity.[34,35] ESR was also significantly increased, reflecting systemic inflammation, as supported by findings from Lianes JM et al.[36]

Limitations of the Study: Despite the important findings, the present study has certain limitations: First, the study employed a cross-sectional design, which limits the ability to establish a causal relationship between inflammatory biomarkers and the development or progression of hypertensive disorders of pregnancy. Second, the sample size was relatively small, particularly in the preeclampsia subgroup, which may limit the generalizability of the findings and reduce statistical power. Third, as this was a single-centre, hospital-based study, selection bias cannot be excluded.

CONCLUSION

The findings of this study support the role of systemic inflammation and endothelial dysfunction in the pathogenesis and progression of hypertensive disorders of pregnancy. Biomarkers such as hs-CRP, ferritin, IL-6, and LDH may serve as valuable indicators for assessing disease severity and progression from gestational hypertension to preeclampsia.

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