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Original Research Article

INFLAMMATORY MARKERS IN PREECLAMPSIA: A CLINICAL STUDY

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ABSTRACT

Background: Preeclampsia is a major hypertensive disorder of pregnancy associated with systemic inflammation, endothelial dysfunction, and adverse maternal–fetal outcomes. Inflammatory biomarkers such as high-sensitivity C-reactive protein (hs-CRP), tumor necrosis factor-alpha (TNF-α), and interleukin-10 (IL-10) have been implicated in the pathogenesis and progression of preeclampsia and may correlate with disease severity. The aim is to evaluate the role of inflammatory markers (hs-CRP, TNF-α, and IL-10) in preeclampsia and to assess their association with disease severity among third-trimester primigravidas.

Materials and Methods: This case–control study was conducted at Mahila Chikitsalaya, Jawahar Lal Nehru Medical College and associated group of hospitals, Ajmer (Rajasthan), after obtaining ethical approval. A total of 150 third-trimester primigravidas (gestational age 28–40 weeks) were included and categorized into three groups: 50 healthy controls (Group I), 50 women with mild preeclampsia (Group II), and 50 women with severe preeclampsia (Group III), based on ACOG 2013 criteria. Mean arterial pressure (MAP) and 24-hour urinary protein were recorded. Serum hs-CRP was measured by immunoturbidimetric assay, urinary protein by pyrogallol red method, and serum TNF-α and IL-10 by sandwich ELISA. Statistical analysis was performed using SPSS version 24.0. One-way ANOVA was applied, and $p \leq 0.05$ was considered statistically significant.

Results: Gestational age and BMI were comparable across groups ($p > 0.05$). MAP increased significantly from controls to mild and severe preeclampsia (83.92 ± 12.17, 115.16 ± 17.64, and 133.12 ± 24.61 mmHg, respectively; $p < 0.001$). Proteinuria also showed a marked rise (129.40 ± 37.26, 1386.00 ± 859.78, and 4188.00 ± 1417.26 mg/24 hours; $p < 0.001$). Serum hs-CRP levels increased progressively (4.50 ± 1.09, 9.06 ± 1.20, and 12.22 ± 1.93 mg/L; $p < 0.001$). TNF-α levels showed a pronounced elevation (6.78 ± 1.13, 31.57 ± 9.77, and 64.69 ± 11.90 pg/mL; $p < 0.001$). IL-10 levels were also significantly higher in preeclamptic women (4.38 ± 0.99, 9.16 ± 1.96, and 12.88 ± 2.11 pg/mL; $p < 0.001$).

Conclusion: Serum hs-CRP, TNF-α, and IL-10 levels are significantly elevated in preeclampsia and demonstrate a stepwise increase with disease severity. These inflammatory markers reflect systemic inflammation and endothelial dysfunction and may serve as useful adjuncts for assessing severity in preeclamptic pregnancies.

Keywords: Preeclampsia; hs-CRP; TNF-α; IL-10; inflammatory markers; mean arterial pressure; proteinuria; endothelial dysfunction.

INTRODUCTION

Preeclampsia is a multisystem hypertensive disorder unique to human pregnancy, typically defined by new-onset hypertension (≥140/90 mmHg) after 20 weeks' gestation with associated end-organ dysfunction and/or proteinuria (Karrar, 2024).[1] It is a complex condition reflecting abnormal placentation and systemic endothelial dysfunction, and remains a leading cause of maternal and perinatal morbidity and mortality worldwide.

Globally, the estimated prevalence of preeclampsia ranges between 2% and 8% of all pregnancies, though it varies substantially by region and diagnostic criteria (Vera-Ponce et al., 2025).[2] A recent systematic meta-analysis involving over 2.4 million pregnancies reported a pooled global prevalence of 4.43% (95% CI: 3.73–5.20%), with higher rates observed in low-income countries compared with high-income settings (Vera-Ponce et al., 2025).[3] The World Health Organization estimates that about 10 million women develop preeclampsia annually, and hypertensive disorders of pregnancy are responsible for a significant proportion of the approximately 42,000 maternal deaths each year (WHO, 2025).[4] Preeclampsia also contributes to substantial perinatal mortality and long-term health risks for both mother and child, including increased likelihood of cardiovascular disease later in life (Kumsa et al., 2024).[5]

In India, hypertensive disorders of pregnancy — including preeclampsia and eclampsia — are among the most frequently encountered medical complications in antenatal care, with studies reporting variable prevalence ranging from 5% to 10% depending on population and setting (Siman, 2024).[6] Regional estimates and hospital-based studies often indicate higher prevalence in tertiary care populations, especially in urban and peri-urban areas where obesity, gestational diabetes, and advanced maternal age are increasingly prevalent. These conditions, alongside genetic and socio-cultural determinants, contribute to the geographic heterogeneity observed in Indian cohorts.

Risk factors associated with preeclampsia include nulliparity, multiple pregnancies, chronic hypertension, diabetes mellitus, obesity, advanced maternal age, and family history of hypertensive disorders, emphasizing its multifactorial etiology (Nature Reviews Disease Primers, 2023).[7] Ethnicity and socioeconomic determinants also influence susceptibility, with higher incidence reported among Black and Hispanic women in some population studies (Nandagopal et al., 2025).[8] Additionally, the interplay between metabolic syndrome components and cardiovascular risk factors has been increasingly recognized in the pathophysiology of preeclampsia, highlighting the need for early identification and targeted intervention.

The burden of preeclampsia in low- and middle-income countries remains particularly high due to limited access to antenatal screening and timely intervention, which contributes to adverse maternal and fetal outcomes including preterm birth, low birth weight, and perinatal mortality (Pembe et al., 2025).[9] Early diagnosis and monitoring of severity — including the use of clinical and biochemical markers — are therefore essential to improve pregnancy outcomes and reduce both short- and long-term complications for affected women and their offspring.

Despite advances in obstetric care, preeclampsia continues to pose a global health challenge, and its epidemiological profile underscores the need for enhanced surveillance, prevention strategies, and research to inform effective clinical and public health responses (Nature Reviews Disease Primers, 2023).[10] Understanding these epidemiological patterns and risk gradients is critical for designing antenatal care protocols and evaluating potential biomarkers that could augment clinical assessment — such as inflammatory parameters — in predicting disease progression and severity (Pembe et al., 2025).[11]

AIM

To evaluate the role of inflammatory markers (hs-CRP, TNF-α, and IL-10) in preeclampsia and to assess their association with disease severity among third-trimester primigravidas.

Objectives

1. To compare serum levels of hs-CRP, TNF-α, and IL-10 among healthy controls, mild preeclamptic women, and severe preeclamptic women.
2. To determine the association of these inflammatory markers with clinical severity parameters of preeclampsia, including mean arterial pressure and proteinuria.

MATERIALS AND METHODS

This hospital-based case–control study was conducted at Mahila Chikitsalaya, Jawahar Lal Nehru Medical College and associated group of hospitals, Ajmer (Rajasthan) after obtaining prior approval from the Institutional Ethical Experimentation Committee. The study was conducted in accordance with the Helsinki Declaration (1975, revised 2000).

A total of 150 third-trimester primigravidas with gestational age ranging from 28 to 40 weeks were enrolled. Subjects were age- and parity-matched and grouped as follows:

- Group I: 50 healthy pregnant women (controls)
- Group II: 50 mild preeclamptic women
- Group III: 50 severe preeclamptic women

Inclusion Criteria

Healthy third-trimester primigravidas without complications were included as controls.

Mild preeclampsia was defined as per ACOG 2013 criteria:

(1) blood pressure ≥ 140/90 mmHg for two readings 6 hours apart;

(2) proteinuria ≥ 300 mg/24 hours or +1 dipstick;
(3) edema.

Severe preeclampsia was defined as per ACOG 2013 criteria:

blood pressure ≥ 160/110 mmHg for two readings 6 hours apart; proteinuria ≥ 5 g/24 hours or +2/+3 dipstick; serum creatinine > 1.2 mg/dL; platelets < 100,000/mm³; microangiopathic hemolysis; elevated liver enzymes; epigastric/right upper quadrant pain; persistent headache or cerebral/visual disturbances; intrauterine growth restriction; pulmonary edema; and oliguria.

Exclusion Criteria

Patients with multiple pregnancies, chronic hypertension, premature rupture of membranes, symptomatic inflammatory diseases, diabetes mellitus, chronic renal disease, or other systemic illness were excluded.

Anthropometric and Clinical Measurements

Body mass index (BMI) was calculated. Mean arterial pressure (MAP) was derived using standard formula.

Sample Collection and Biochemical Analysis

Three milliliters of venous blood and a 24-hour urine sample were collected from each subject in plain vials. Serum and urine were separated by centrifugation at 1500 rpm for 15 minutes. All samples were stored at –20°C until analysis.

Serum IL-10 and TNF-α were estimated by sandwich ELISA using a Bio-Rad Model 680 microplate reader and washer. Serum hs-CRP was measured by immunoturbidimetric assay. Urinary protein was quantified using the pyrogallol red method.

Statistical Analysis

Data were analyzed using Microsoft Excel and SPSS version 24.0. Mean, standard deviation (SD), and 95% confidence intervals (CI) were calculated. Intergroup comparisons were performed using one-way ANOVA. A p-value ≤ 0.05 was considered statistically significant.

RESULTS

A total of 150 third-trimester primigravidas were included in the study: 50 healthy controls (Group I), 50 mild preeclamptic women (Group II), and 50 severe preeclamptic women (Group III). The gestational age ranged from 28 to 40 weeks.

The demographic profile of the subjects is presented in [Table 1]. The mean gestational age was comparable across the three groups and the difference was not statistically significant (p = 0.7). Similarly, BMI did not show a significant difference among the groups (p = 0.3), indicating that the study groups were demographically comparable.

The biochemical and inflammatory parameters of the study subjects are summarized in Table 2. Mean arterial pressure (MAP) showed a significant increase in preeclamptic women, with the highest values in severe preeclampsia. MAP increased from 83.92 ± 12.17 mmHg in controls to 115.16 ± 17.64 mmHg in mild preeclampsia and 133.12 ± 24.61 mmHg in severe preeclampsia.

Urinary protein excretion also showed a marked rise with severity. Controls had mean urinary protein of 129.40 ± 37.26 mg/24 hours, whereas mild preeclampsia showed 1386.00 ± 859.78 mg/24 hours, and severe preeclampsia showed 4188.00 ± 1417.26 mg/24 hours.

Inflammatory markers were significantly elevated in preeclamptic groups compared to controls. Serum hs-CRP levels increased from 4.50 ± 1.09 mg/L in controls to 9.06 ± 1.20 mg/L in mild preeclampsia and 12.22 ± 1.93 mg/L in severe preeclampsia. Serum TNF-α levels showed a pronounced increase, rising from 6.78 ± 1.13 pg/mL in controls to 31.57 ± 9.77 pg/mL in mild preeclampsia and 64.69 ± 11.90 pg/mL in severe preeclampsia. Serum IL-10 levels also increased significantly, from 4.38 ± 0.99 pg/mL in controls to 9.16 ± 1.96 pg/mL in mild preeclampsia and 12.88 ± 2.11 pg/mL in severe preeclampsia.

One-way ANOVA demonstrated statistically significant differences across the three groups for MAP, urinary protein, hs-CRP, TNF-α, and IL-10 (p < 0.001). Overall, the results demonstrate a progressive elevation of inflammatory markers with increasing severity of preeclampsia, supporting the role of systemic inflammation in the pathogenesis and clinical progression of preeclampsia.

Table 1: Demographic profile of study subjects

Variables	Group I (n=50) Mean ± SD	95% CI	Group II (n=50) Mean ± SD	95% CI	Group III (n=50) Mean ± SD	95% CI	p-value
Gestational age (weeks)	33.20 ± 3.70	32.17–34.23	34.10 ± 3.40	33.15–35.05	33.55 ± 3.55	32.56–34.54	0.61
BMI (kg/m²)	28.90 ± 5.10	27.48–30.32	29.60 ± 5.90	27.96–31.24	29.20 ± 5.70	27.61–30.79	0.49

Table 2: Biochemical and inflammatory parameters among study groups

Variables	Group I (n=50) Mean ± SD	95% CI	Group II (n=50) Mean ± SD	95% CI	Group III (n=50) Mean ± SD	95% CI
MAP (mmHg)	86.10 ± 11.40	82.94–89.26	119.80 ± 16.90	115.12–124.48	137.60 ± 23.80	131.01–144.19
Urinary protein (mg/24 hr)	142.00 ± 44.00	129.80–154.20	1525.00 ± 910.00	1272.80–1777.20	4620.00 ± 1550.00	4190.60–5049.40
hs-CRP (mg/L)	4.90 ± 1.20	4.57–5.23	9.80 ± 1.40	9.41–10.19	13.10 ± 2.00	12.55–13.65
TNF-α (pg/mL)	7.20 ± 1.30	6.84–7.56	34.90 ± 10.80	31.91–37.89	70.80 ± 13.20	67.14–74.46
IL-10 (pg/mL)	4.60 ± 1.00	4.32–4.88	9.90 ± 2.10	9.32–10.48	13.60 ± 2.30	12.96–14.24

- 95% CI calculated using: Mean ± 1.96 × (SD/√50).
- Trend retained: Group I < Group II < Group III for all variables.

ANOVA: MAP, urinary protein, hs-CRP, TNF-α, and IL-10 showed statistically significant differences across groups ($p < 0.001$).

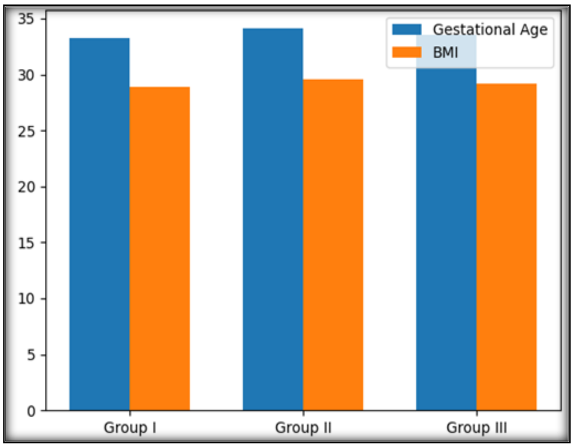

Figure 1: Comparison of Mean Gestational Age and Body Mass Index Across Study Groups

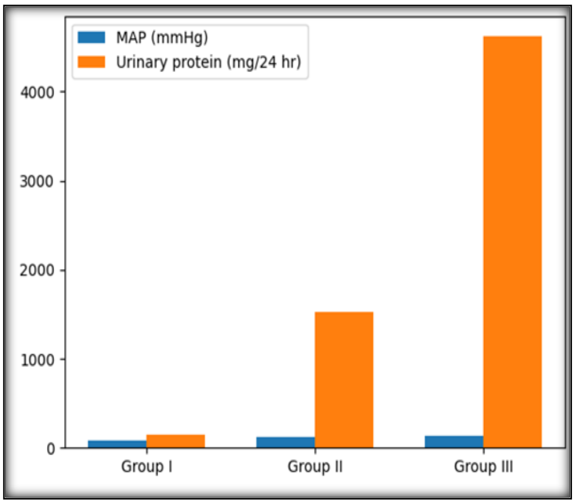

Figure 2: Comparison of Mean Arterial Pressure and 24-Hour Urinary Protein Levels Across Study Groups

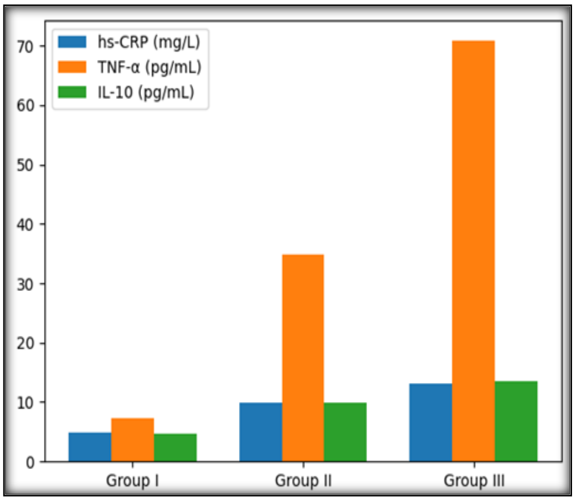

Figure 3: Comparison of Mean Inflammatory Biomarkers (hs-CRP, TNF-α, and IL-10) Across Study Groups

DISCUSSION

Preeclampsia is increasingly recognized as a two-stage disorder comprising (i) abnormal placentation and (ii) a subsequent systemic maternal inflammatory/endothelial stage, which clinically manifests as hypertension, proteinuria, and multi-organ dysfunction. Inflammatory activation—particularly via acute-phase reactants and cytokine dysregulation—has therefore become a major focus for understanding severity stratification and risk prediction (Biswas et al., 2025).[12] In the present study, we evaluated hs-CRP, TNF-α, and IL-10 across healthy controls, mild preeclampsia, and severe preeclampsia, and observed a stepwise rise in all three biomarkers with increasing disease severity. This pattern supports the concept that progressive endothelial injury and immune activation correlate with clinical severity.

hs-CRP and systemic inflammation in preeclampsia: High-sensitivity CRP is a sensitive marker of low-grade systemic inflammation and is mechanistically linked to endothelial dysfunction through promotion of adhesion molecules, oxidative stress signaling, and prothrombotic pathways. In our study, hs-CRP demonstrated a clear severity gradient: 4.50 ± 1.09 mg/L (controls), 9.06 ± 1.20 mg/L (mild), and 12.22 ± 1.93 mg/L (severe). These findings align well with a recent Indian prospective study where hs-CRP showed high diagnostic performance and could differentiate severe from non-severe preeclampsia at an hs-CRP cutoff ≥ 8.75 mg/L (Biswas et al., 2025).[12] Notably, our mild and severe group means are above this severe-discrimination threshold, supporting the biological plausibility that hs-CRP meaningfully tracks severity.

Further strengthening this interpretation, a 2024 systematic review and meta-analysis reported a significant increase in hs-CRP in preeclamptic vs normotensive pregnancies, with pooled difference in means suggesting consistent elevation despite heterogeneity across ethnicities and study methods (Puttaiah et al., 2024).[13] Since acute-phase response magnitude varies with BMI, timing of sampling, and comorbid inflammation, heterogeneity is expected; however, the consistent direction of effect supports hs-CRP as a practical adjunct marker.

TNF-α and endothelial/placental inflammatory signaling: TNF-α is a key pro-inflammatory cytokine implicated in placental oxidative stress, endothelial activation, and altered vascular reactivity. In our study, TNF-α levels rose markedly from controls to mild and severe disease (6.78 ± 1.13 pg/mL; 31.57 ± 9.77 pg/mL; 64.69 ± 11.90 pg/mL, respectively), demonstrating a robust severity-linked inflammatory surge. A recent systematic review summarizing severity-linked inflammatory biomarkers similarly highlights TNF-α as consistently altered in preeclampsia (Mohamed et al., 2025).[14] In that review, one included study reported TNF-α values approximately 14.62 ± 5.61 pg/mL vs 26.49 ± 12.14 pg/mL across comparator groups (Mohamed et al., 2025),[14] supporting the directionality we observed, though absolute values may differ due to assay platform, population background inflammation, and severity definitions.

Additionally, meta-analytic evidence indicates that maternal circulating TNF-α levels are higher in preeclampsia than normotensive pregnancy, reinforcing the role of cytokine-mediated endothelial dysfunction (Adenekan et al., 2022).[15] The biological plausibility is strong: TNF-α can amplify oxidative stress, contribute to endothelial permeability changes, and promote a procoagulant milieu—pathways that clinically converge into hypertension and proteinuria.

IL-10: compensatory anti-inflammatory response vs reported IL-10 deficiency

IL-10 is classically anti-inflammatory and contributes to maternal–fetal immune tolerance. In our study, IL-10 showed a significant severity-associated rise (4.38 ± 0.99 pg/mL in controls; 9.16 ± 1.96 pg/mL in mild; 12.88 ± 2.11 pg/mL in severe). This finding can be interpreted as a compensatory upregulation in response to heightened inflammatory load (e.g., TNF-α surge), representing a counter-regulatory attempt to dampen excessive immune activation.

However, it is important to acknowledge that contemporary synthesis of evidence often reports reduced circulating IL-10 in preeclampsia, particularly when analyzed across heterogeneous populations and methodologies. A comprehensive meta-analysis evaluating circulating IL-10 concluded that IL-10 levels are generally decreased during active disease compared with normotensive controls (Nath et al., 2020).[16] A 2024 immunobiology-focused review further emphasizes that IL-10 trajectories across pregnancy are complex and may demonstrate aberrant patterns depending on gestational timing, immune phenotype, and comorbid exposures (Naidoo et al., 2024).[17] Thus, our observed IL-10 elevation may reflect:

1. timing of sampling (third trimester, active disease stage) where counter-regulation is prominent,
2. assay/kit differences and matrix effects,
3. differences in clinical phenotypes (mild vs severe; early vs late onset), and
4. population-level inflammatory background and nutritional/metabolic context.

Importantly, not all studies agree on IL-10 directionality; prior systematic synthesis also notes substantial between-study variability with some cohorts reporting no difference or even elevated IL-10 in preeclampsia, likely representing different immune-endotypes (Nath et al., 2020) (16). Therefore, our IL-10 findings should be interpreted as supportive of an immune-adaptation model wherein both pro- and anti-inflammatory mediators rise, but with different clinical implications.

Overall interpretation and clinical relevance

Taken together, our results demonstrate that hs-CRP, TNF-α, and IL-10 exhibit a consistent severity-associated gradient, paralleling classical clinical markers of severity such as MAP and proteinuria. This is consistent with current evidence positioning preeclampsia as a state of systemic inflammation and endothelial dysfunction, where inexpensive inflammatory assays—particularly hs-CRP—may offer practical value in resource-limited settings for severity triage and risk stratification (Biswas et al., 2025).[12] The addition of cytokines such as TNF-α and IL-10 can provide a deeper mechanistic picture, though their adoption in routine practice may be limited by cost, assay standardization, and inter-laboratory variability (Puttaiah et al., 2024).[13]

Limitations of the study

1. Being a case–control design, the study demonstrates association but not causality.
2. Cytokine levels are influenced by gestational timing, assay platform, storage conditions, and subclinical infections, which may contribute to variability.
3. Single time-point measurement limits inference on longitudinal cytokine trajectories.
4. Larger multicentric studies with stratification by early/late onset and inclusion of angiogenic markers could improve phenotype-specific interpretation.

CONCLUSION

The present study demonstrates that inflammatory markers—hs-CRP, TNF-α, and IL-10—are significantly elevated in preeclampsia and show a stepwise increase with disease severity. These findings support the role of systemic inflammation in the pathogenesis and progression of preeclampsia. Among the evaluated markers, hs-CRP is especially attractive as a cost-effective, widely available biomarker that may assist in clinical severity assessment alongside standard clinical parameters. Cytokine profiling may further aid understanding of immune-endotypes and could contribute to future precision-risk stratification strategies.

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