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Association of Maternal Serum C-Reactive Protein and Procalcitonin with Severity of Preeclampsia

[1]Dr. Shivani Sharma, Senior Resident, Department of Biochemistry, Pandit Naval Kishor Sharma Government Medical College, Dausa, Rajasthan – 303501, India

[2]Dr. Sandhya Mishra, Professor and HOD, Department of Biochemistry, Geetanjali Medical College, Jaipur, Rajasthan – 302026, India

[3]Dr. Harshit Parashar, Senior Resident, Department of Biochemistry, ESIC Medical College and Hospital, Desula, Alwar, Rajasthan – 301030, India

[4]Dr. Diwakar Sharma, Junior Resident, Department of Orthopaedics, Sawai Man Singh Medical College, Jaipur, Rajasthan, India

[5]Dr. Oby Nagar, Senior Professor, Department of Obstetrics and Gynaecology, Sawai Man Singh Medical College, Jaipur, Rajasthan, India

Corresponding Author: Dr. Shivani Sharma, Senior Resident, Department of Biochemistry, Pandit Naval Kishor Sharma Government Medical College, Dausa, Rajasthan – 303501, India

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Abstract

Background: Preeclampsia is a pregnancy-related hypertensive disorder associated with systemic inflammation. C-reactive protein (CRP) and Procalcitonin (PCT) may serve as potential biomarkers of disease severity.

Aim: To evaluate serum CRP and Procalcitonin levels in healthy pregnant women and patients with mild and severe preeclampsia.

Materials and Methods: This hospital-based cross-sectional study was conducted at SMS Medical College, Jaipur, Rajasthan, from December 2023 to November 2024. A total of 102 pregnant women were included: 51 healthy controls, 33 with mild preeclampsia and 18 with severe preeclampsia. Serum CRP and Procalcitonin were estimated, and data were analysed using SPSS version 26.

Results: Mean CRP levels were 0.93 ± 0.18 mg/dL in controls, 3.24 ± 0.89 mg/dL in mild preeclampsia, and 10.29 ± 1.78 mg/dL in severe preeclampsia. Mean Procalcitonin levels were 0.0431 ± 0.0032 ng/mL in controls, 0.1481 ± 0.0246 ng/mL in mild preeclampsia,

and 0.3097 ± 0.0211 ng/mL in severe preeclampsia. Both inflammatory markers showed progressively higher levels across the three groups, with the highest concentrations observed in severe preeclampsia. Severe preeclampsia was also associated with higher blood pressure, proteinuria, liver enzyme levels, urea and creatinine, along with lower serum albumin levels.

Conclusion: Serum CRP and Procalcitonin levels increased with the severity of preeclampsia, suggesting their potential role as adjunctive inflammatory biomarkers.

Keywords: Preeclampsia, CRP, Procalcitonin, Inflammatory markers, Pregnancy.

Introduction

Preeclampsia is a pregnancy-specific hypertensive disorder that generally develops after 20 weeks of gestation and is characterized by new-onset hypertension associated with proteinuria and/or maternal organ dysfunction. It is a significant cause of maternal and perinatal morbidity and mortality worldwide. The disease may present with varying degrees of severity, ranging from mild hypertension to severe multisystem involvement. Maternal complications include eclampsia, HELLP syndrome, acute renal injury, hepatic dysfunction, placental abruption and cerebrovascular complications, while fetal complications include intrauterine growth restriction, prematurity and low birth weight. Early diagnosis, appropriate monitoring and timely intervention are essential to reduce adverse maternal and fetal outcomes [1].

The pathogenesis of preeclampsia is complex and involves abnormal placentation, impaired trophoblastic invasion and inadequate remodelling of the maternal spiral arteries. These abnormalities lead to increased placental vascular resistance, reduced uteroplacental perfusion and placental ischemia. The ischemic placenta releases several antiangiogenic and inflammatory mediators into the maternal circulation, resulting in widespread endothelial dysfunction, vasoconstriction and increased vascular permeability. Oxidative stress, immune dysregulation and disturbances in angiogenic balance further contribute to the development and progression of the disorder [2].

Systemic inflammation is considered an important component of the pathophysiology of preeclampsia. Although pregnancy is associated with physiological changes in maternal immune activity, women with preeclampsia demonstrate an exaggerated inflammatory response. Increased circulating concentrations of pro-inflammatory cytokines, acute-phase proteins and other inflammatory mediators contribute to endothelial activation and vascular injury. This inflammatory imbalance may be associated with the clinical manifestations and severity of preeclampsia [3].

C-reactive protein (CRP) is a major acute-phase protein synthesized predominantly by hepatocytes in response to inflammatory cytokines, particularly interleukin-6. It is widely used as a biochemical indicator of systemic inflammation. Increased CRP concentrations have been reported in several pregnancy-related inflammatory conditions, including preeclampsia. The elevation of maternal serum CRP may reflect placental inflammation, oxidative stress and endothelial dysfunction. Therefore, CRP has been investigated as a potential biomarker for identifying inflammatory changes associated with preeclampsia [4,5].

Previous research has demonstrated an association between elevated maternal CRP concentrations and the development of preeclampsia. However, the magnitude of this association may vary according to maternal

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characteristics, gestational age, body mass index and the presence of other inflammatory conditions. Consequently, CRP estimation may provide additional information regarding maternal inflammatory status, although its clinical interpretation requires consideration of other relevant factors [5,6].

Procalcitonin (PCT) is a 116-amino-acid precursor of the hormone calcitonin. Under normal physiological conditions, its circulating concentration is generally low. During systemic inflammatory responses, particularly bacterial infections, its production increases in several tissues. In recent years, PCT has also been investigated in non-infectious inflammatory conditions, including preeclampsia. Increased PCT concentrations have been reported in affected pregnant women, suggesting a possible relationship between systemic inflammatory activation and the development of hypertensive complications during pregnancy [7,8].

Studies evaluating PCT in preeclampsia have reported higher concentrations in affected women compared with normotensive pregnant controls. Some investigators have also observed increasing PCT concentrations with increasing disease severity and blood pressure. These observations suggest that PCT may reflect certain inflammatory changes associated with placental dysfunction and maternal endothelial injury. However, its specificity and independent diagnostic value in preeclampsia remain to be established [7–9].

The combined assessment of CRP and PCT may provide complementary information about maternal inflammatory responses. CRP primarily reflects hepatic acute-phase protein synthesis, whereas PCT is associated with systemic inflammatory activation. Evaluating these biomarkers together with conventional clinical and biochemical parameters may help in understanding the relationship between inflammation and disease severity[10,11].

Materials and Methods

Study Design and Setting

The present study was a hospital-based observational, comparative cross-sectional study conducted in the Department of Biochemistry in collaboration with the Department of Obstetrics and Gynaecology at SMS Medical College and its attached hospitals, Jaipur, Rajasthan. The study was conducted over a period of one year, from December 2023 to November 2024.

Ethical Approval

The study was initiated after obtaining approval from the Institutional Ethics Committee and Research Review Board of SMS Medical College and Hospital, Jaipur. Written informed consent was obtained from all participants before enrolment.

Study Population

A total of 102 pregnant women in their third trimester were enrolled and divided into two groups:

- Group I (Cases): 51 pregnant women diagnosed with preeclampsia.
- Group II (Controls): 51 age- and gestational age-matched healthy pregnant women without hypertension or proteinuria.

Participants were recruited from the outpatient and inpatient departments using a simple random sampling technique.

Inclusion Criteria

Cases:

1. Pregnant women aged 18–35 years in the third trimester.
2. Diagnosed with preeclampsia after 20 weeks of gestation, with blood pressure ≥140/90 mmHg and proteinuria ≥300 mg/24 hours.

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3. Willingness to participate and provide written informed consent.

Controls:

1. Healthy pregnant women aged 18–35 years in the third trimester.
2. No history of hypertension or proteinuria.
3. Willingness to participate and provide written informed consent.

Exclusion Criteria

Participants were excluded if they had:

1. Pre-existing hypertension or diabetes mellitus.
2. Renal, hepatic, autoimmune or other chronic systemic diseases.
3. Smoking or alcohol consumption.
4. Multiple pregnancies or congenital fetal anomalies.
5. Recent infections or medication use that could influence inflammatory markers.
6. Refusal to provide informed consent.

Clinical Assessment

Detailed obstetric, medical and family histories were recorded for all participants. General physical and systemic examinations were performed. Blood pressure was measured, and anthropometric parameters, including height, weight and body mass index (BMI), were documented.

The diagnosis of preeclampsia was established based on the presence of new-onset hypertension and proteinuria after 20 weeks of gestation. Clinical and laboratory findings were evaluated to assess disease severity.

Sample Collection and Processing

Venous blood samples were collected after 12–14 hours of fasting in three separate vials. The first plain vial was used for routine biochemical and physiological investigations, while the second plain vial was used for serum CRP and Procalcitonin estimation. EDTA vials were used for CBC analysis. Samples were centrifuged at 3000 rpm for 5 minutes, and separated serum was aliquoted and stored at −20°C until analysis. Haemolysed and lipemic samples were excluded.

Biochemical Investigations

The following investigations were performed:

A. Routine Investigations

- Complete blood count (CBC).
- Fasting blood glucose.
- Liver function tests (AST, ALT, bilirubin and alkaline phosphatase).
- Renal function tests (serum urea and creatinine).
- Serum albumin and total protein.
- Urinary protein estimation.

B. Special Investigations

- Serum C-reactive protein (CRP).
- Serum Procalcitonin (PCT).

Laboratory Analysis

Complete Blood Count

Complete blood count was performed using an automated haematology analyser (Sysmex XN-Series or equivalent). Haemoglobin, total leukocyte count, differential leukocyte count, platelet count, haematocrit and red cell indices were assessed.

Routine Biochemical Parameters

Fasting blood glucose was estimated by the glucose oxidase-peroxidase (GOD-POD) method. Liver function parameters were analysed using spectrophotometric enzymatic methods, while serum urea and creatinine were estimated using standard automated methods.

Serum albumin was measured by the bromocresol green method, and total protein was estimated using the biuret method on the Vitros 5600 Integrated System.

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Assessment of Proteinuria

Urinary protein was initially screened using a semi-quantitative dipstick method. Quantitative assessment was performed using 24-hour urinary protein estimation or spot urine protein-to-creatinine ratio.

Estimation of Inflammatory Markers

Serum C-Reactive Protein (CRP)

Serum CRP concentration was estimated using a fixed-point immunoradiometric immunoassay on the Vitros 5600 Integrated System. The assay is based on an antigen-antibody reaction involving CRP-specific antibodies, with the resulting signal used to quantify CRP concentration.

Results were expressed in mg/L. A concentration below 5 mg/L was considered within the stated reference range.

Serum Procalcitonin (PCT)

Serum Procalcitonin was measured using a chemiluminescent immunometric assay on the Vitros 5600 Integrated System. The assay employed PCT-specific monoclonal antibodies labelled with acridinium ester and antibody-coated magnetic particles. The emitted chemiluminescent signal was measured to determine PCT concentration.

Results were expressed in ng/mL. PCT levels below 0.1 ng/mL were considered within the stated reference range.

Statistical Analysis

Data were entered into Microsoft Excel and analysed using Statistical Package for Social Sciences (SPSS), version 26. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as frequencies and percentages. Student's independent t-test was used to compare continuous variables between cases and controls. Pearson's correlation coefficient was applied to assess the relationship between inflammatory markers and relevant clinical and neonatal parameters. A p-value of less than 0.05 was considered statistically significant.

A total of 102 participants were included: 51 healthy normotensive controls, 33 women with mild preeclampsia and 18 women with severe preeclampsia.

Results and Observations

Table 1: Comparison of Routine Physiological and Biochemical Parameters

Parameter	Healthy Controls (n=51)	Mild Preeclampsia (n=33)	Severe Preeclampsia (n=18)
Age (years)	26.98 ± 2.57	27.94 ± 2.37	29.44 ± 1.79
BMI (kg/m²)	22.69 ± 0.78	26.43 ± 0.59	27.34 ± 0.67
SBP (mmHg)	109.39 ± 1.77	146.73 ± 2.07	164.94 ± 3.06
DBP (mmHg)	64.86 ± 2.06	98.97 ± 1.86	113.11 ± 2.03
Proteinuria (mg/24 hours)	203.73 ± 25.61	1457.58 ± 185.46	3950.00 ± 400.37
Serum Blood Sugar (mg/dL)	87.85 ± 9.28	103.69 ± 14.23	121.77 ± 12.07
AST (U/L)	25.29 ± 3.96	35.50 ± 11.78	51.55 ± 15.52
ALT (U/L)	30.24 ± 6.51	40.94 ± 7.07	59.29 ± 9.37
Total Bilirubin (mg/dL)	0.82 ± 0.19	1.03 ± 0.31	1.18 ± 0.42
Direct Bilirubin (mg/dL)	0.30 ± 0.10	0.42 ± 0.10	0.54 ± 0.23

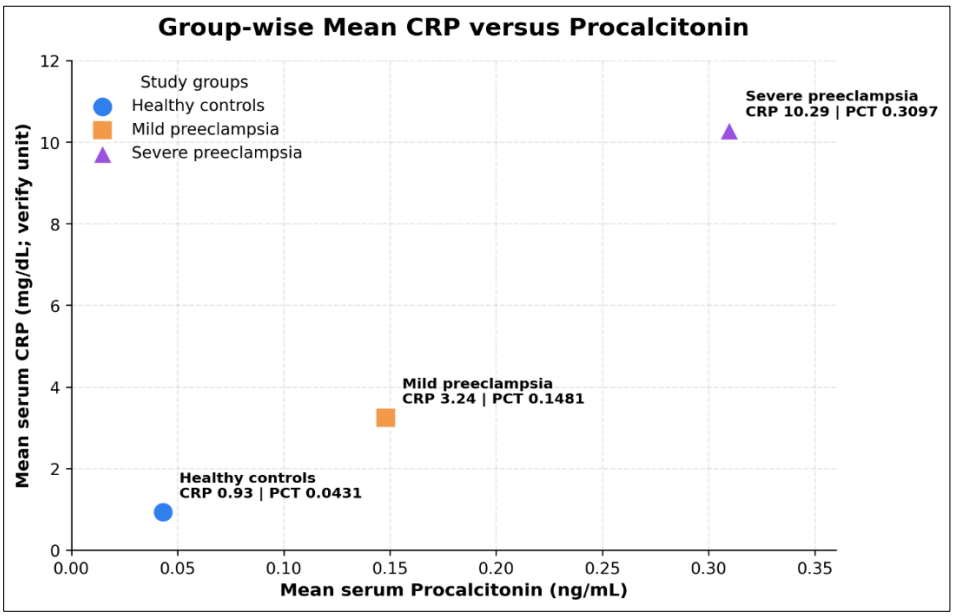
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Serum Urea (mg/dL)	23.99 ± 4.99	35.99 ± 7.17	45.36 ± 6.40
Serum Creatinine (mg/dL)	0.80 ± 0.19	0.95 ± 0.26	1.24 ± 0.33
Serum Albumin (g/dL)	4.20 ± 0.42	3.99 ± 0.48	3.21 ± 0.52
Total Protein (g/dL)	7.04 ± 0.47	6.88 ± 0.50	6.62 ± 0.71

Table 2: Comparison of CRP and Procalcitonin Across Study Groups

Parameter	**Healthy Controls (n=51)**	**Mild Preeclampsia (n=33)**	**Severe Preeclampsia (n=18)**
CRP (mg/dL)	0.93 ± 0.18	3.24 ± 0.89	10.29 ± 1.78
Procalcitonin (ng/mL)	0.0431 ± 0.0032	0.1481 ± 0.0246	0.3097 ± 0.0211

Figure 1:



Discussion

The present study was conducted to investigate the association of maternal serum C-reactive protein (CRP) and Procalcitonin (PCT) concentrations with the severity of preeclampsia. A total of 102 pregnant women were included, comprising 51 healthy normotensive controls, 33 women with mild preeclampsia and 18 women with severe preeclampsia. The study evaluated demographic characteristics, physiological parameters, routine biochemical investigations and two important inflammatory biomarkers.

The major finding was a progressive increase in maternal serum CRP and PCT concentrations from healthy controls to mild and severe preeclampsia. In addition, women with severe preeclampsia demonstrated higher blood pressure, proteinuria, liver enzymes, serum urea and creatinine, together with reduced serum albumin concentrations. These findings indicate that increasing disease severity was accompanied by changes in inflammatory markers and several biochemical parameters associated with maternal organ involvement.

Demographic and Anthropometric Characteristics

In the present study, the mean maternal age was 26.98 ± 2.57 years among healthy controls, 27.94 ± 2.37 years among women with mild preeclampsia and 29.44 ± 1.79 years among women with severe preeclampsia. A gradual increase in mean maternal age was observed across the study groups.

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The mean BMI was 22.69 ± 0.78 kg/m² in controls, 26.43 ± 0.59 kg/m² in mild preeclampsia and 27.34 ± 0.67 kg/m² in severe preeclampsia. Thus, women with preeclampsia had comparatively higher BMI values than healthy pregnant controls.

Increased maternal BMI is an important factor associated with hypertensive disorders of pregnancy. Excess adipose tissue may contribute to chronic low-grade inflammation, oxidative stress and endothelial dysfunction. These mechanisms may influence the inflammatory environment associated with preeclampsia. Rebelo et al. reported that the relationship between maternal CRP concentrations and subsequent preeclampsia may be influenced by maternal weight status, highlighting the importance of considering BMI while interpreting inflammatory biomarkers [6].

The higher BMI observed in the preeclampsia groups may therefore have contributed to the differences in inflammatory marker concentrations. However, the present study did not establish an independent association between BMI and disease severity.

Comparison of Blood Pressure Parameters

Blood pressure measurements demonstrated a progressive increase across the study groups. The mean systolic blood pressure was 109.39 ± 1.77 mmHg in healthy controls, 146.73 ± 2.07 mmHg in mild preeclampsia and 164.94 ± 3.06 mmHg in severe preeclampsia.

Similarly, the mean diastolic blood pressure increased from 64.86 ± 2.06 mmHg in controls to 98.97 ± 1.86 mmHg in mild preeclampsia and 113.11 ± 2.03 mmHg in severe preeclampsia.

The progressive elevation of systolic and diastolic blood pressure reflects the increasing hypertensive burden among women with severe preeclampsia. Abnormal placentation and the release of placental-derived factors contribute to systemic endothelial dysfunction, increased vascular resistance and abnormal vascular reactivity.

Phipps et al. described endothelial dysfunction as a central mechanism in the development of preeclampsia, contributing to hypertension and multisystem maternal complications [2]. Redman and Sargent also highlighted the importance of the maternal systemic inflammatory response in the development of endothelial abnormalities associated with preeclampsia [3].

The higher blood pressure values observed in the severe preeclampsia group provide an important clinical context for interpreting the increased concentrations of CRP and PCT in these women.

Comparison of Proteinuria

Proteinuria demonstrated a marked increase with increasing severity of preeclampsia. The mean 24-hour urinary protein concentration was 203.73 ± 25.61 mg in healthy controls, 1457.58 ± 185.46 mg in mild preeclampsia and 3950.00 ± 400.37 mg in severe preeclampsia.

The highest proteinuria was observed among women with severe preeclampsia, suggesting greater renal involvement in this group. Proteinuria in preeclampsia is primarily associated with glomerular endothelial injury, altered glomerular permeability and impaired renal endothelial function.

Phipps et al. described glomerular endotheliosis and renal endothelial dysfunction as important pathological features of preeclampsia [2]. These abnormalities contribute to impaired glomerular filtration and increased urinary protein excretion.

The present findings are consistent with the involvement of renal dysfunction in severe preeclampsia. However, the amount of proteinuria alone should not be considered

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a reliable indicator of disease severity or maternal prognosis. Current clinical guidelines emphasize the importance of assessing other maternal organ dysfunction and severe clinical features [1].

Comparison of Liver Function Parameters

The present study demonstrated a progressive increase in liver enzyme concentrations among women with increasing severity of preeclampsia.

Mean AST levels were 25.29 ± 3.96 U/L in controls, 35.50 ± 11.78 U/L in mild preeclampsia and 51.55 ± 15.52 U/L in severe preeclampsia. Similarly, mean ALT concentrations increased from 30.24 ± 6.51 U/L in controls to 40.94 ± 7.07 U/L in mild preeclampsia and 59.29 ± 9.37 U/L in severe preeclampsia.

Total bilirubin and direct bilirubin also demonstrated an increasing trend across the study groups. Total bilirubin increased from 0.82 ± 0.19 mg/dL in controls to 1.18 ± 0.42 mg/dL in severe preeclampsia, whereas direct bilirubin increased from 0.30 ± 0.10 mg/dL to 0.54 ± 0.23 mg/dL.

These findings suggest progressive hepatic biochemical abnormalities with increasing disease severity. Hepatic involvement in preeclampsia may result from endothelial injury, vasoconstriction and impaired hepatic microcirculation. In severe cases, hepatic ischemia and hepatocellular injury may occur, occasionally progressing to HELLP syndrome.

The observations are consistent with the established understanding of preeclampsia as a multisystem disorder involving hepatic, renal and cardiovascular dysfunction[1,2].

Comparison of Renal Function Parameters

Renal function parameters demonstrated a progressive increase in serum urea and creatinine concentrations across the study groups.

Mean serum urea was 23.99 ± 4.99 mg/dL in controls, 35.99 ± 7.17 mg/dL in mild preeclampsia and 45.36 ± 6.40 mg/dL in severe preeclampsia.

Similarly, serum creatinine increased from 0.80 ± 0.19 mg/dL in healthy controls to 0.95 ± 0.26 mg/dL in mild preeclampsia and 1.24 ± 0.33 mg/dL in severe preeclampsia.

The observed increase in renal function parameters may indicate progressive impairment of renal filtration and renal endothelial function in women with severe preeclampsia. Glomerular endotheliosis, reduced renal perfusion and altered glomerular permeability are important pathological mechanisms responsible for renal involvement in this condition.

Phipps et al. emphasized that renal abnormalities are important manifestations of preeclampsia and are closely associated with the underlying endothelial dysfunction [2].

The findings of the present study further demonstrate that women with severe preeclampsia had greater biochemical abnormalities involving renal function than healthy pregnant controls.

Comparison of Serum Albumin and Total Protein

Serum albumin concentrations demonstrated a decreasing trend with increasing severity of preeclampsia. Mean serum albumin was 4.20 ± 0.42 g/dL in controls, 3.99 ± 0.48 g/dL in mild preeclampsia and 3.21 ± 0.52 g/dL in severe preeclampsia.

Similarly, total protein decreased from 7.04 ± 0.47 g/dL in controls to 6.88 ± 0.50 g/dL in mild preeclampsia and 6.62 ± 0.71 g/dL in severe preeclampsia.

The reduction in serum albumin among women with severe preeclampsia may be associated with increased urinary protein loss, altered vascular permeability and changes in maternal protein metabolism.

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Albumin is also considered a negative acute-phase protein, and its concentration may decrease during systemic inflammatory responses. The reduced albumin levels observed in severe preeclampsia may therefore partly reflect the combined effects of renal protein loss and systemic inflammation [3].

These observations indicate that alterations in protein metabolism and vascular permeability may accompany the increasing severity of preeclampsia.

Comparison of Maternal Serum C-Reactive Protein Levels

One of the major findings of the present study was the progressive increase in maternal serum CRP concentrations across the three study groups.

The mean CRP concentration was 0.93 ± 0.18 mg/dL in healthy controls, 3.24 ± 0.89 mg/dL in mild preeclampsia and 10.29 ± 1.78 mg/dL in severe preeclampsia.

The highest CRP concentration was observed in women with severe preeclampsia, followed by women with mild preeclampsia and healthy controls. The mean CRP concentration in severe preeclampsia was approximately 11 times higher than that observed in the control group.

This progressive increase suggests an association between maternal systemic inflammatory activation and the clinical severity of preeclampsia.

CRP is synthesized predominantly by hepatocytes in response to inflammatory cytokines, particularly IL-6. In preeclampsia, placental ischemia, oxidative stress and endothelial injury may stimulate the release of inflammatory mediators, leading to increased hepatic CRP synthesis.

The findings of the present study are supported by Guan et al., who conducted a systematic review and meta-analysis evaluating inflammatory biomarkers in preeclampsia. Their analysis demonstrated significantly higher CRP concentrations among women with preeclampsia compared with normotensive pregnant controls, supporting the involvement of inflammatory imbalance in the disease [4].

Similarly, Hamadeh et al. evaluated the role of CRP in the prediction and early detection of preeclampsia. Their systematic review identified a positive association between CRP concentrations and the development of preeclampsia in several included studies, suggesting that CRP may have potential as an additional inflammatory biomarker [5].

Rebelo et al. also reported an association between elevated maternal CRP concentrations and subsequent development of preeclampsia. Their systematic review and meta-analysis highlighted that maternal BMI may influence this relationship, emphasizing the importance of considering anthropometric characteristics when interpreting CRP concentrations [6].

Puttaiah et al. (2024), in their systematic review and meta-analysis of inflammatory markers in pregnant women, reported increased maternal circulating inflammatory markers in preeclampsia compared with normotensive pregnancy. Their findings demonstrated a significant increase in hs-CRP and moderate increases in CRP and other inflammatory markers, providing further evidence of the role of inflammation in preeclampsia [12].

The findings of the present study are in agreement with these published observations and demonstrate a progressive increase in CRP concentrations from healthy pregnancy to mild and severe preeclampsia.

However, CRP is a nonspecific inflammatory marker and may also increase in infections and other inflammatory conditions. Therefore, its interpretation

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should be made alongside clinical findings and other laboratory investigations.

Comparison of Maternal Serum Procalcitonin Levels

The present study also demonstrated a progressive increase in maternal serum Procalcitonin concentrations across the three study groups.

The mean PCT concentrations were 0.0431 ± 0.0032 ng/mL in healthy controls, 0.1481 ± 0.0246 ng/mL in mild preeclampsia and 0.3097 ± 0.0211 ng/mL in severe preeclampsia.

The highest PCT concentration was observed in women with severe preeclampsia, followed by women with mild preeclampsia and healthy controls. The mean PCT concentration in severe preeclampsia was approximately seven times higher than that in healthy controls.

This progressive increase suggests that PCT may be associated with systemic inflammatory activation accompanying the progression of preeclampsia.

Although Procalcitonin is primarily recognized as a biomarker of bacterial infection, its concentrations may also increase in certain non-infectious inflammatory conditions. Placental dysfunction, endothelial injury and systemic inflammatory activation may contribute to altered PCT concentrations in women with preeclampsia [10].

Artunc-Ulkumen et al. investigated the relationship between neutrophil gelatinase-associated lipocalin and Procalcitonin concentrations with the presence and severity of preeclampsia. They reported significantly higher PCT concentrations in women with preeclampsia compared with healthy pregnant controls. Their findings also suggested an association between PCT concentrations and disease severity [7].

Can et al. evaluated inflammatory markers in preeclamptic patients and reported increased inflammatory marker concentrations among affected women. Their findings supported the involvement of systemic inflammatory responses in the pathophysiology of preeclampsia [8].

Montagnana et al. specifically investigated the relationship between Procalcitonin concentrations and the severity of preeclampsia. They reported higher PCT concentrations in women with severe preeclampsia compared with those with milder disease, suggesting a possible relationship between PCT and disease severity[9].

Mangogna et al., in their review of Procalcitonin during pregnancy and preeclampsia, discussed the potential role of PCT in pregnancy-related inflammatory responses. They also highlighted the differences in findings across published studies and the need for further investigation before its routine clinical application [10].

Lumbreras-Marquez et al. evaluated maternal and umbilical cord concentrations of PCT, high-sensitivity CRP and IL-6 in preeclamptic and normotensive pregnant women. Their findings demonstrated increased maternal inflammatory marker concentrations in women with preeclampsia, further supporting the potential relevance of inflammatory biomarkers in this condition [11].

The observations of the present study are consistent with these reports and suggest that maternal serum PCT concentrations increase with increasing clinical severity of preeclampsia.

Nevertheless, PCT should not be considered a specific diagnostic marker for preeclampsia because increased concentrations may also occur in bacterial infections and other systemic inflammatory conditions.

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Combined Association of CRP and Procalcitonin with Preeclampsia Severity

An important finding of the present study was the simultaneous increase in CRP and PCT concentrations with increasing severity of preeclampsia.

Both inflammatory markers demonstrated a similar pattern, with the highest mean concentrations in severe preeclampsia, intermediate concentrations in mild preeclampsia and the lowest concentrations in healthy controls.

CRP and PCT represent different aspects of the inflammatory response. CRP primarily reflects hepatic acute-phase protein synthesis, whereas PCT is associated with systemic inflammatory activation. Their simultaneous elevation may therefore provide complementary information regarding the inflammatory changes associated with preeclampsia.

The findings of Guan et al. and Puttaiah et al. support the association between increased circulating inflammatory markers and preeclampsia [4,12]. Similarly, Mangogna et al. discussed the possible involvement of PCT in pregnancy-related inflammatory mechanisms [10].

The combined assessment of CRP and PCT alongside blood pressure, proteinuria, liver enzymes and renal function parameters may provide additional information regarding the inflammatory profile of women with preeclampsia.

However, the present study demonstrates differences in group-wise mean concentrations and does not independently establish a diagnostic or prognostic role for either biomarker. Correlation analysis, receiver operating characteristic curve analysis and prospective follow-up would be required to determine their clinical utility.

Conclusion

Serum CRP and Procalcitonin levels increased progressively with the severity of preeclampsia, indicating their potential role as adjunctive inflammatory biomarkers. Further studies are needed to establish their diagnostic and prognostic value.

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