ISSN: 2457-0400

WORLD JOURNAL OF ADVANCE HEALTHCARE RESEARCH

www.wjahr.com

Impact Factor: 7.675

Volume: 10, Issue: 10

Page N. 191-198

Year: 2026

Original Article

Coden USA: WJAMA3

ASSOCIATION OF PLASMA FIBRINOGEN LEVEL AND C-REACTIVE PROTEIN LEVEL WITH SEVERITY OF PREECLAMPSIA

***[1]Marwah Mohammed Hamdoon ALraash, [2]Ahmad Jasim Mohammed**

[*1]M.B.Ch.B., FIBOG; [2]Department of Obstetrics and Gynecology, College of Medicine, University of Mosul, Iraq.

Article Received: 17 August 2026 **Article Revised: 07 September 2026** **Article Published: 01 October 2026**

***Corresponding Author: Marwah Mohammed Hamdoon ALraash**

M.B.Ch.B., FIBOG, College of Medicine, University of Mosul, Iraq.

DOI: https://doi.org/10.5281/zenodo.23014340

How to cite this Article: *[1]Marwah Mohammed Hamdoon ALraash, [2]Ahmad Jasim Mohammed (2026). Association Of Plasma Fibrinogen Level And C-Reactive Protein Level With Severity Of Preeclampsia. World Journal of Advance Healthcare Research, 10(10), 191–198.

ABSTRACT

Background: Preeclampsia is a pregnancy disorder that is a leading cause of maternal and perinatal morbidity and mortality, characterized by elevated blood pressure and proteinuria after 20 weeks of gestation. In preeclampsia there is a shift in the hemostatic balance towards a prothrombotic state together with changes in endothelial function, and C-reactive protein (CRP) is a sensitive inflammatory marker that rises in response to inflammation or tissue injury. **Aim:** To determine the association of plasma fibrinogen level and C-reactive protein level with the severity of preeclampsia. **Patients and Methods:** A prospective case-control study was conducted at Al-Khansaa and Al-Batool Teaching Hospitals, Mosul, Iraq, and included 50 pregnant women with preeclampsia in the third trimester (14 mild, 36 severe) and 50 normotensive pregnant women in the third trimester as controls. Plasma fibrinogen and C-reactive protein levels were measured and compared between the groups and subgroups. **Results:** Plasma fibrinogen level was significantly higher in women with preeclampsia than in normotensive pregnant women (259.74 ± 56.13 vs. 237.04 ± 46.51 mg/dL, P = 0.03) and increased further with disease severity, being higher in severe than in mild preeclampsia (282.44 ± 45.36 vs, 201.35 ± 35.16 mg/dL, P = 0.0001). C-reactive protein was also significantly higher in preeclamptic women than in controls (13.88 ± 6.42 vs. 10.43 ± 6.06 mg/L, P = 0.044) but showed no significant difference between mild and severe preeclampsia (P = 0.271). Fibrinogen correlated positively with systolic blood pressure and mean arterial pressure (P = 0.023 and P = 0.024, respectively) and with the degree of albuminuria (P = 0.0001), while CRP showed no significant correlation with blood pressure parameters or albuminuria. **Conclusion:** Plasma fibrinogen and C-reactive protein levels are increased in women with preeclampsia compared with normotensive pregnant women, and higher fibrinogen levels are associated with more severe disease, whereas CRP does not distinguish mild from severe preeclampsia. Plasma fibrinogen may therefore serve as a useful adjunct marker for assessing preeclampsia severity.

KEYWORDS: Preeclampsia, C-reactive protein, Fibrinogen level, Pregnancy, Mild preeclampsia, Severe preeclampsia.

1. INTRODUCTION

Hypertensive disorders of pregnancy, including preeclampsia, are major contributors to maternal and neonatal morbidity and mortality worldwide, complicating an estimated 2–8% of pregnancies globally.[1], [2] Preeclampsia is defined as new-onset hypertension after 20 weeks of gestation, most often accompanied by proteinuria, although hypertension with other features of the disease — thrombocytopenia, impaired liver function, pulmonary edema, or cerebral/visual symptoms — may occur in its absence.[3],[4] The disease may lead to serious maternal complications, including placental abruption, HELLP syndrome, and renal failure, as well as fetal complications such as intrauterine growth restriction, prematurity, and stillbirth.[2], [5] Preeclampsia is further classified into mild and severe forms according to the degree of hypertension, proteinuria, and involvement of other organ systems.[6]

Defective trophoblastic invasion of the maternal spiral arteries leads to abnormal placentation, reduced placental

perfusion, oxidative stress, and release of anti-angiogenic and inflammatory mediators that produce widespread maternal endothelial dysfunction.[7], [8] This is accompanied by a shift in the hemostatic balance toward a prothrombotic state.[9] C-reactive protein (CRP) is an acute-phase reactant synthesized by the liver in response to interleukin-6 during inflammation, and its level is known to rise further in preeclampsia as a marker of the associated systemic inflammatory response and endothelial injury.[10], [11] Fibrinogen is a 340-kDa glycoprotein synthesized in hepatocytes that is converted to fibrin during coagulation and is central to hemostasis; its plasma level normally rises progressively during pregnancy as an adaptive mechanism to limit blood loss at delivery and is reported to be further elevated in preeclampsia due to endothelial damage, fibrin deposition in the maternal microcirculation, and activation of the coagulation cascade.[12], [13], [14] Given their accessibility and low cost, CRP and fibrinogen have been proposed as potential adjunct biomarkers for identifying preeclampsia and stratifying its severity.

1.1 AIM OF THE STUDY

To determine the association of plasma fibrinogen level and C-reactive protein level with the severity of preeclampsia.

1.2 Specific Objectives

- To assess the socio-demographic characteristics of the study groups.
- To assess the symptoms and signs among the preeclamptic patients.
- To determine the level of CRP in pregnant women with preeclampsia and in normotensive pregnant women and compare between them.
- To determine the level of plasma fibrinogen in pregnant women with preeclampsia and normotensive pregnant women and compare between them.
- To determine the association between CRP level and fibrinogen level with the severity of preeclampsia.

2. PATIENTS AND METHODS

2.1 Study Design, Setting, and Population

This prospective case-control study was conducted at Al-Khansaa and Al-Batool Teaching Hospitals, Mosul, Iraq, from 7 January 2023 to 1 December 2023. The study included 100 pregnant women in the third trimester who were not in labor, allocated into two groups: 50 women with preeclampsia—14 with mild disease (systolic blood pressure 140–160 mmHg, diastolic 90–110 mmHg, and proteinuria of +1 by dipstick) and 36 with severe disease (systolic blood pressure above 160 mmHg, diastolic above 110 mmHg, with proteinuria ≥+2 and/or headache, visual disturbance, epigastric pain, or pulmonary edema)—and 50 normotensive pregnant women as controls. Eligible women had a gestational age of 28 weeks or more with a singleton viable pregnancy and were not in labor; women in labor or with chronic hypertension, chronic renal, cardiovascular, or autoimmune disease; diabetes mellitus; systemic infection; preterm rupture of membranes; conditions that could independently alter fibrinogen level (placenta previa, stillbirth, heavy vaginal bleeding); or use of anticoagulant drugs were excluded.

2.2 Data Collection and Clinical Assessment

After verbal and written informed consent, all participants underwent a standardized history, examination, and laboratory work-up. History included personal, menstrual/obstetric, present, past medical, drug, and family history. Examination included general and abdominal assessment and blood pressure measurement by sphygmomanometer with the patient resting in the left lateral position, cuff on the left arm at the level of the heart, using the auscultatory technique. Urine albumin was assessed by the dipstick method, and venous blood was collected using a clean aseptic technique.

2.3 CRP Measurement

Serum CRP was measured using the ichroma™ CRP fluorescence immunoassay (FIA), a quantitative sandwich immunodetection method in which detector antibodies bind antigen in the sample to form antigen-antibody complexes that migrate onto a nitrocellulose matrix and are captured by immobilized antibodies on the test strip; the instrument automatically calculates and displays the CRP concentration in mg/L.

2.4 Fibrinogen Assay

Plasma fibrinogen was measured by the Clauss method using the Bio-Fibri kit on a semi-automated coagulation analyzer (BIO SOLIA2/BIOSOLIA4), with samples collected in 3.2% sodium citrate and tested within 4 hours of collection. Diluted plasma was incubated and combined with the working thrombin reagent, and clotting time was measured automatically and converted to fibrinogen concentration using a calibration curve.

2.5 Statistical Analysis

Data were analyzed using IBM SPSS version 29. Continuous variables were expressed as mean ± SD and range and compared using the independent-samples Student's t-test (two groups) or ANOVA (more than two groups). Categorical variables were expressed as frequencies and percentages and compared using the Pearson Chi-square test, with Yates' correction or Fisher's exact test where applicable. Pearson correlation was used to assess associations between CRP, fibrinogen, and blood pressure parameters. A two-tailed P-value ≤ 0.05 was considered statistically significant.

2.6 Ethical Considerations

Ethical approval was obtained from the Iraqi Board for Medical Specializations and the Medical Research Ethics Committee, College of Medicine, University of Mosul. All participants gave verbal and written informed consent, and data were used exclusively for the purpose of this study.

3. RESULTS

This study included 100 pregnant women in the third trimester (age range 18–40 years): 50 normotensive controls and 50 women with preeclampsia, of whom 14 had mild and 36 had severe disease.

3.1 Demographic Characteristics

Table 1 shows that the mean age in preeclamptic women was 29.3 ± 6.9 years compared with 26.9 ± 6.1 years in normotensive controls, with no significant difference between groups (P = 0.062). Residence and parity distribution were also comparable between the two groups, with no significant statistical difference (P > 0.05).

Table 1: Demographic distribution of the studied groups.

Variable	Category	PE	Control	P-value
Maternal age (years)	18–20	4 (8.0)	3 (6.0)	0.297*
	20–24	12 (24.0)	19 (38.0)	
	25–29	6 (12.0)	10 (20.0)	
	30–34	12 (24.0)	8 (16.0)	
	≥35	16 (32.0)	10 (20.0)	
	Mean±SD (Range)	29.3±6.9 (18-40)	26.9±6.1 (18-39)	0.062#
Residence	Urban	30 (60.0)	29 (58.0)	0.839*
	Rural	20 (40.0)	21 (42.0)	
Parity	0	18 (36.0)	17 (34.0)	0.515*
	1	8 (16.0)	11 (22.0)	
	2	6 (12.0)	10 (20.0)	
	3	6 (12.0)	2 (4.0)	
	≥4	12 (24.0)	10 (20.0)	

*Significant difference between percentages using Pearson Chi-square test (χ^2-test) at the 0.05 level. #Significant difference between two independent means using Student's t-test at 0.05 level.

3.2 Blood Pressure

Table 2 shows significantly higher systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP) in preeclamptic women compared with controls (P = 0.0001).

Table 2: Blood pressure in both preeclamptic and normotensive pregnant women.

Blood pressure	PE	Control	P-value
SBP (mmHg)	158.8±17.7 (130-200)	113.2±7.9 (100-130)	0.0001#
DBP (mmHg)	107.5±8.7 (100-140)	70.6±7.4 (60-90)	0.0001#
MAP (mmHg)	124.6±10.9 (110.0-160.0)	84.8±6.4 (73.3-103.3)	0.0001#

3.3 Symptoms and Signs of Preeclampsia

Among preeclamptic patients, 64% had a headache, 44% had edema, 42% had blurred vision, 30% had epigastric pain, and only 8% had nausea and vomiting. Regarding albuminuria, 28% had (+), 38% had (++), and 34% had (+++) or more, as shown in Table 3.

Table 3: Symptoms and signs of preeclampsia.

Symptom/Sign		PE, No.	PE, %
Headache	Yes	32	64.0
	No	18	36.0
Edema	Yes	22	44.0
	No	28	56.0
Blurred vision	Yes	21	42.0
	No	29	58.0
Epigastric pain	Yes	15	30.0
	No	35	70.0
Nausea & vomiting	Yes	4	8.0
	No	46	92.0
Albumin in urine	[+]	14	28.0

	[++]	19	38.0
	[+++] and more	17	34.0

3.4 C-Reactive Protein Levels

CRP was positive in 66% of preeclamptic patients and 46% of normotensive controls. Table 4 shows a significant increase in CRP level in preeclamptic patients (13.88 ± 6.42 mg/L) compared with controls (10.43 ± 6.06 mg/L, P = 0.044). There was no significant difference in CRP level between mild (16.29 ± 7.52 mg/L) and severe preeclampsia (13.23 ± 6.09 mg/L, P = 0.271), as illustrated in Figures 1 and 2.

Table 4: Comparison of C-reactive protein between PE women and normotensive pregnant women and between mild and severe PE.

Variable	Category	PE	Control	P-value
C-Reactive protein	Positive	33 (66.0)	23 (46.0)	0.044*
	Negative	17 (34.0)	27 (54.0)	
	Mean±SD (Range)	13.88±6.42 (6.0-24.0)	10.43±6.06 (6.0-24.0)	0.048#
	Mild PE (n=7)	16.29±7.52 (6.0-24.0)	–	0.271
	Severe PE (n=26)	13.23±6.09 (6.0-24.0)	–	

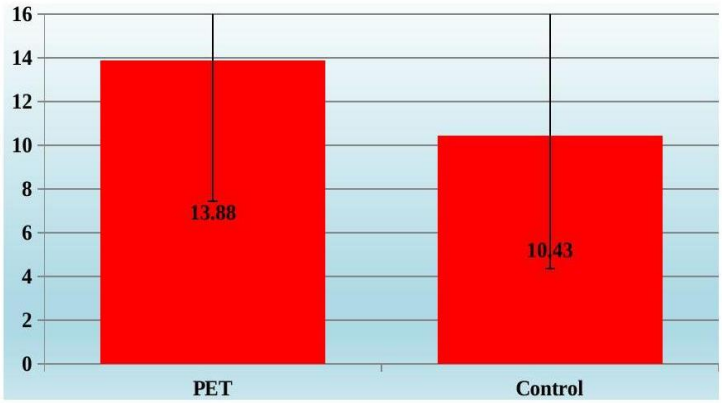

Figure 1: C-reactive protein level in preeclamptic and control groups.

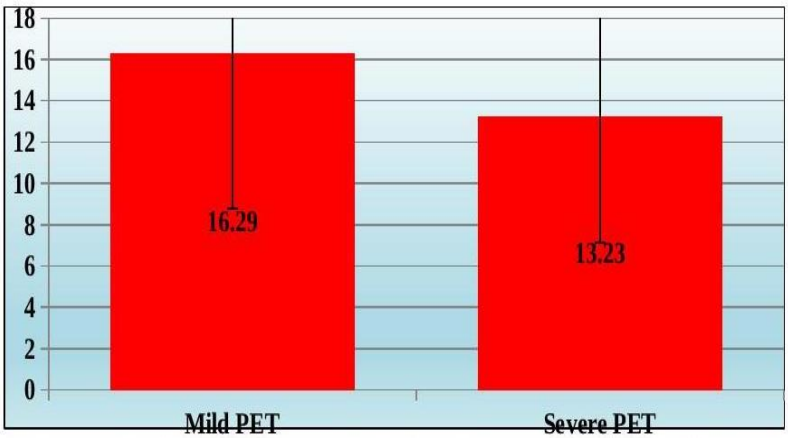

Figure 2: C-reactive protein level between mild and severe preeclampsia.

3.5 Plasma Fibrinogen Levels

Plasma fibrinogen level in the control group ranged from 135.6 to 284.6 mg/dL (mean 237.04 ± 46.51), while in PE patients it ranged from 163.7 to 398.9 mg/dL (mean 259.74 ± 56.13), a significant difference (P = 0.03). Fibrinogen was significantly higher in severe (282.44 ± 45.36 mg/dL) than mild preeclampsia (201.35 ± 35.16 mg/dL, P = 0.0001), as shown in Table 5 and Figures 3 and 4.

Table 5: Plasma fibrinogen levels in PE women, normotensive pregnant women, and mild and severe PE.

Variable	Category	PE	Control	P-value
Fibrinogen level (mg/dL)	100–149	–	2 (4.0)	0.024*
	150–199	8 (16.0)	11 (22.0)	
	200–249	13 (26.0)	10 (20.0)	
	250–299	20 (40.0)	27 (54.0)	
	300–349	5 (10.0)	–	
	350–399	4 (8.0)	–	
	Mean±SD (Range)	259.74±56.13 (163.7-398.9)	237.04±46.51 (135.6-284.6)	0.030#
	Mild PE (n=14)	201.35±35.16 (163.7-258.8)	–	0.0001#
	Severe PE (n=36)	282.44±45.36 (216.4-398.9)	–	

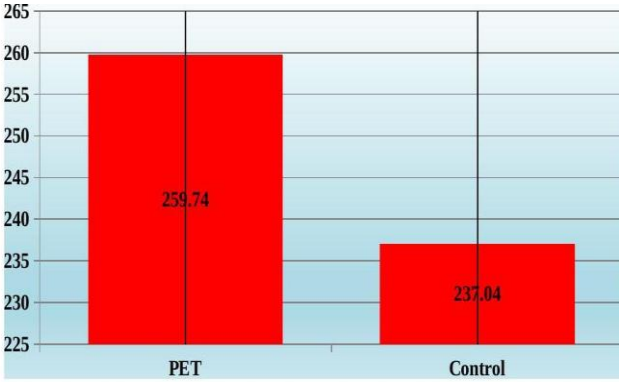

Figure 3: Fibrinogen level in PE and control groups.

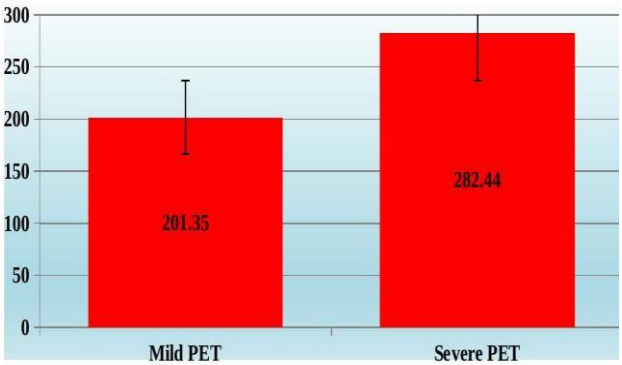

Figure 4: Fibrinogen level in mild and severe PE.

3.6 Relation of Symptoms and Albuminuria with CRP

Of the 33 preeclamptic patients with positive CRP, mean CRP levels were similar regardless of the presence of edema, headache, blurred vision, nausea/vomiting, or epigastric pain (all P > 0.05) and did not differ significantly across grades of albuminuria (P = 0.516), as shown in Table 6.

Table 6: Relation of symptoms of preeclampsia and amount of albumin in urine with CRP level.

Symptom/Sign		No.	CRP Mean±SD	P-value
Edema	Yes	14	14.00±7.23	0.928
	No	19	13.79±5.96	
Headache	Yes	21	13.52±6.78	0.681
	No	12	14.50±5.98	
Blurred vision	Yes	13	13.23±6.91	0.648
	No	20	14.30±6.23	
Nausea & vomiting	Yes	2	9.00±4.24	0.274
	No	31	14.19±6.46	
Epigastric pain	Yes	10	12.00±6.93	0.275
	No	23	14.70±6.17	

Albumin in urine	[+]	7	16.29±7.52	0.516
	[++]	13	13.69±6.58	
	[+++] and more	13	12.77±5.80	

3.7 Relation of Symptoms and Albuminuria with Fibrinogen

The fibrinogen level showed no significant difference according to edema, headache, blurred vision, nausea/vomiting, or epigastric pain (all P > 0.05) but increased significantly with higher grades of albuminuria (P = 0.0001), as shown in Table 7.

Table 7: Relation of symptoms of preeclampsia and level of albumin in urine with fibrinogen level.

Symptom/Sign		No.	Fibrinogen Mean±SD	P-value
Edema	Yes	22	268.1±58.8	0.358
	No	28	253.2±54.1	
Headache	Yes	32	268.5±46.0	0.141
	No	18	244.1±69.4	
Blurred vision	Yes	21	271.9±57.7	0.196
	No	29	250.9±54.3	
Nausea & vomiting	Yes	4	272.5±19.0	0.641
	No	46	258.6±58.2	
Epigastric pain	Yes	15	269.2±48.8	0.439
	No	35	255.7±59.2	
Albumin in urine	[+]	14	201.4±35.2	0.0001^
	[++]	19	284.5±46.6	
	[+++] and more	17	280.1±45.2	

3.8 Relation Between CRP and Fibrinogen

Among preeclamptic patients, mean fibrinogen level was 265.5 ± 54.5 mg/dL in those with positive CRP and 248.5 ± 59.1 mg/dL in those with negative CRP, with no significant difference between the two groups (P = 0.855), as shown in Table 8.

Table 8: Relation between CRP and fibrinogen levels in PE patients with positive and negative CRP.

		No.	Fibrinogen Mean±SD
C-Reactive protein	Positive	33	265.5±54.5
	Negative	17	248.5±59.1
	P-value		0.855

3.9 Correlation with Blood Pressure Parameters

Table 9 shows that the correlation of CRP with SBP, DBP, and MAP was not significant (P > 0.05). Fibrinogen level showed a significant positive correlation with SBP (r = 0.322, P = 0.023) and MAP (r = 0.318, P = 0.024), but not with DBP (r = 0.270, P = 0.058).

Table 9: Correlation of CRP and plasma fibrinogen levels in PE patients with SBP, DBP, and MAP.

Variable	CRP: r	CRP: P-value	Fibrinogen: r	Fibrinogen: P-value
SBP (mmHg)	-0.245	0.169	0.322*	0.023
DBP (mmHg)	-0.096	0.597	0.270	0.058
MAP (mmHg)	-0.179	0.318	0.318*	0.024

4. DISCUSSION

4.1 Baseline Characteristics

Preeclampsia is one of the leading causes of maternal morbidity and the second most common cause of maternal mortality worldwide[15] and is associated with an exaggerated systemic inflammatory response to endothelial damage[10] as well as changes in the hemostatic system and an increased risk of venous thromboembolism.[18] In this study, the mean age of preeclamptic women was 29.3 ± 6.9 years, comparable to Shao et al.[15] (30.4 ± 5.6 years) and higher than Meena et al.[16], with no significant age difference between groups in either study, consistent with the present findings. Most cases were nulliparous (36%), likely reflecting immunological inadequacy between fetoplacental and maternal tissue[17], and parity and gestational age also did not differ significantly between groups, in agreement with Alwan et al.[18] and Gharib et al.[10] Blood pressure parameters (SBP, DBP, MAP) were significantly higher

in preeclamptic women than controls, consistent with Han et al.[19], Alwan et al.[18], and Ren et al.[20]

4.2 C-Reactive Protein in Preeclampsia

CRP was significantly higher in preeclamptic patients than controls, likely reflecting endothelial cell damage in these patients.[10] This finding is consistent with Nagori et al.[21] and Wasan et al.[22], both of whom reported higher CRP in preeclampsia. However, this study found no significant difference in CRP between mild and severe preeclampsia (P = 0.271), consistent with Mohammed et al.[23] and Mishra et al.[24], but in contrast to Wasan et al.[22] and Nagori et al.[21], who found significant differences; this discrepancy may reflect differences in sample size across studies. CRP also showed no significant correlation with albuminuria (in contrast to Nagori et al.[21]) or with blood pressure parameters (consistent with Mohammed et al.[23]), which may relate to variability in the severity of inflammation and renal involvement across patients.

4.3 Plasma Fibrinogen in Preeclampsia

Plasma fibrinogen was significantly higher in preeclamptic women than controls, consistent with Ren et al.[20] and Wasan et al.[22], but in contrast to Meena et al.[16] and Han et al.[19], who reported lower fibrinogen in preeclampsia, possibly reflecting differences in gestational age and insufficient hepatic fibrinogen production in late pregnancy under increased hepatic load. Fibrinogen was significantly higher in severe than mild preeclampsia, consistent with Ren et al.[20] and Wasan et al.[22], but again in contrast to Meena et al.[16] and Han et al.[19]. These discrepancies across studies likely reflect differences in gestational timing, sample size, and assay methodology.

4.4 Correlations with Clinical Parameters

Fibrinogen level increased significantly with the degree of albuminuria, plausibly because endothelial damage to the renal vasculature increases permeability to protein and activates the coagulation system at the site of injury, raising fibrinogen consumption and turnover.[25] No previous study was identified for direct comparison of this specific relationship. There was a significant direct correlation between fibrinogen and SBP/MAP, consistent with Alwan et al.[18], who also reported a significant correlation between fibrinogen and MAP. No relationship was found between CRP and fibrinogen levels and no relationship between symptoms/signs and either marker, for which no prior comparative data were identified.

4.5 Strengths and Limitations

This study benefits from a case-control design with standardized clinical assessment and laboratory measurement of both markers in the same cohort, allowing direct comparison of their diagnostic behavior. However, the relatively small number of mild preeclampsia cases (n = 14) may have limited the power to detect differences between severity subgroups, and the single-center design may limit generalizability. Larger multicenter studies are recommended to confirm these findings and to further evaluate the combined predictive value of fibrinogen and CRP in preeclampsia.

5. Conclusion and Recommendations

Serum CRP and plasma fibrinogen levels are increased in pregnant women with preeclampsia compared with normotensive pregnant women. Plasma fibrinogen level rises further with increasing disease severity and can help differentiate mild from severe preeclampsia, whereas CRP level does not distinguish between the two. Plasma fibrinogen also showed a direct correlation with systolic blood pressure and mean arterial pressure. These findings support consideration of plasma fibrinogen as a useful adjunct marker for assessing preeclampsia severity, while CRP appears more useful for identifying the presence of preeclampsia than for grading its severity. Early screening for preeclampsia is recommended in women with recognized risk factors, including nulliparity, multiple gestation, chronic hypertension, advanced maternal age, obesity, diabetes mellitus, and autoimmune disease. Larger studies with increased sample size are recommended to strengthen the statistical power of these findings, and pregnant women should be encouraged to attend regular antenatal care.

Declaration

This thesis is submitted in partial fulfillment of the requirements for the Iraqi Board for Medical Specializations in Obstetrics and Gynecology.

REFERENCES

1. Ndwiga C, Odwe G, Pooja S, et al. Clinical presentation and outcomes of preeclampsia and eclampsia at a national hospital, Kenya: A retrospective cohort study. PLoS One, 2020; 15(6): 1-15.
2. Espinoza J, Vidaeff A, Pettker CM, et al. Gestational Hypertension and Preeclampsia - Clinical Management Guidelines for Obstetrician-Gynecologists. Obstet Gynecol, 2019; 133(76): 168-186.
3. Opichka MA, Rappelt MW, Gutterman DD, et al. Vascular dysfunction in preeclampsia. Cells, 2021; 10(11): 3055.
4. Leeman L, Dresang LT, Fontaine P, et al. Hypertensive disorders of pregnancy. Am Fam Physician, 2016; 93(2): 121-127.
5. Pankiewicz K, Szczerba E, Maciejewski T, et al. Non-obstetric complications in preeclampsia. Przeglad Menopauzalny, 2019; 18: 99-109.
6. Peres GM, Mariana M, Cairrão E. Pre-eclampsia and eclampsia: An update on the pharmacological treatment applied in Portugal. J Cardiovasc Dev Dis, 2018; 5(1): 3.
7. Al-Jameil. A Brief Overview of Preeclampsia. J Clin Med Res, 2013; 6(1): 1-7.
8. Jena MK, Sharma NR, Petitt M, et al. Pathogenesis of preeclampsia and therapeutic approaches

targeting the placenta. Biomolecules, 2020; 10(6): 953.
9. Barjat TR, Edebiri O, Ainle FN, et al. Preeclampsia and venous thromboembolism: pathophysiology and potential therapy. Front Cardiovasc Med, 2022; 9: 856923.
10. Gharib M, Mostafa M, Harira M, et al. Predictive value of maternal serum C-reactive protein levels with severity of preeclampsia. Zagazig Univ Med J, 2016; 22(2): 1-12.
11. Nehring S, Goyal A, Bhupendra C. C Reactive Protein, 2022; 18: 35-40.
12. Kattula S, Byrnes JR, Wolberg AS. Fibrinogen and fibrin in hemostasis and thrombosis. Arterioscler Thromb Vasc Biol, 2017; 37: e13-e21.
13. Kaur J, Jain A. Fibrinogen, 2020; 992: 181-192.
14. Pinheiro MB, Gomes KB, Dusse LM. Fibrinolytic system in preeclampsia. Clin Chim Acta, 2013; 416: 67-71.
15. Shao H, Gao S, Dai D, et al. The association of antenatal D-dimer and fibrinogen with postpartum hemorrhage and intrauterine growth restriction in preeclampsia. BMC Pregnancy Childbirth, 2021; 21(1): 1-7.
16. Meena P, Gupta A, Meena S, et al. A comparative study of coagulation profile in pre-eclamptic and normotensive pregnant females, 2018; 17(3): 49-58.
17. Grum T, Seifu A, Abay M, et al. Determinants of pre-eclampsia/eclampsia among women attending delivery services in selected public hospitals of Addis Ababa, Ethiopia: A case control study. BMC Pregnancy Childbirth, 2017; 17(1): 1-7.
18. Alwan AF. Study of plasma fibrinogen in pregnant women with severe preeclampsia. IOSR J Dent Med Sci, 2013; 8(5): 55-59.
19. Han L, Liu X, Li H, et al. Blood coagulation parameters and platelet indices: changes in normal and preeclamptic pregnancies and predictive values for preeclampsia. PLoS One, 2014; 9(12): 1-14.
20. Ren H, Liu W, Niu A, et al. Fibrinogen to albumin ratio, a novel serum indicator for evaluating the severity of preeclampsia: A single-center retrospective study. Medicine (Baltimore), 2023; 102(13): e33419.
21. Nagori M, Chauhan M. Serum CRP levels in pre-eclampsia and its association with microalbuminuria. Indian J Obstet Gynecol Res, 2020; 7(4): 553-558.
22. Wasan W, Al-Assaly R, Saeed Al-Haddad N. CA-125, plasma fibrinogen and C-reactive protein in correlation with severity of preeclampsia. J Fac Med, 2017; 59(1): 31-35.
23. Mohammed C, Adze JA, Bature SB, et al. Serum C-reactive protein in preeclamptic women in Jos, Nigeria, 2022; 1(1): 1-7.
24. Mishra N, Pradhan K, Sahoo GD. Can hsCRP be the sole investigation for predicting the severity and outcome in women with preeclampsia preceding late in pregnancy? Trop J Obstet Gynaecol, 2019; 36: 54-60.
25. Boeldt DS, Bird IM. Vascular adaptation in pregnancy and endothelial dysfunction in preeclampsia. J Endocrinol, 2017; 232(1): R27-R44.