Original Article

Comparison of Mean D-Dimer Levels in Preeclampsia Versus Normal Pregnancy

Sidra Bukhari[1], Mudassara Iram[2], Lubna Noor[3], Maryam Arshad[4], Mahreen Afzal[5], Mahnoor Sajjad[6]

[1,2,4,5,6]Postgraduate Resident, [3]Assistant Professor
Dept of Obs & Gynae, Combined Military Hospital, Multan

***Correspondence**: Dr. Sidra Bukhari*
Postgraduate Resident Dept of Obs & Gynae, Combined Military Hospital, Multan
sidrabukhari012@gmail.com

Abstract

Objective: To compare prevalence of high D-dimer levels between pregnant women with or without preeclampsia.
Methodology: This comparative cross-sectional study was conducted in the Department of Obstetrics and Gynecology, CMH Multan, from April 2025, to September 2025. Seventy (70) third-trimester pregnant women aged 20–40 years were enrolled, including 35 women with preeclampsia and 35 normotensive women in the comparison group. Demographic and clinical variables were recorded. D-dimer levels were estimated in platelet-poor plasma using MISPA-i2 reagent, and levels >0.5 µg/mL were considered elevated. Data were analyzed using SPSS version 27. Descriptive statistics were calculated. Quantitative and categorical variables were compared using the independent-samples t-test and chi-square test, respectively. A p-value <0.05 was considered statistically significant.
Results: Mean age was 30.5±5.2 years and 54% of women were obese. Women with preeclampsia were older than normotensive pregnant women (31.8±5.3 vs. 29.2±4.8 years, p=0.039). Mean D-dimer levels were significantly higher among women with preeclampsia compared to without preeclampsia (0.76±0.78 vs. 0.23±0.44 µg/ml, p<0.001). High D-dimer levels were more frequent in preeclampsia (22.9% vs. 2.9%, p=0.028). Stratified analysis showed significantly higher D-dimer levels in younger, urban, and obese women with preeclampsia. Among women aged below 30-years, prevalence of high D-dimer levels remained significantly greater in pregnant females with pre-eclampsia than without preeclampsia (38.5% vs. 0%, p-value = 0.003), in primigravida (46.2% vs. 6.3%, p-value=0.026) and with obesity (28.6% vs. 3.8%, p=0.025).
Conclusion: Pre-eclampsia was associated with elevated D-dimer levels and higher prevalence of abnormal D-dimer positivity during third trimester of pregnancy.
Key Words: Body mass index, D-dimer, Third trimester of pregnancy, Pre-eclampsia

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Introduction

A small amount of protein produced by fibrinolysis, known as D-dimer (DD), dramatically rises during pregnancy.[1] Throughout the prenatal period, its concentrations in the mother's blood rise without any thromboembolic complications. To lower the danger of postpartum hemorrhage, blood coagulation mechanism in pregnant women must be intact.[2] One of the most important markers for diagnosing and treating thrombotic conditions with atrial fibrillation and venous thromboembolism is D-dimer.[3]

The etiology and pathophysiology of hypertensive disorders of pregnancy (HDP), which are reported in around ten percent of pregnancies worldwide and are the 2nd leading reason of maternal deaths, remain unclear.[4] Adverse events for both the mother and the fetus, including maternal stroke, mortality, less weight of babies at birth and the requirement for neonatal critical care, can result from HDP.[5] Preeclampsia (PE), which affects 5–8% of pregnancies, is the most frequent pregnancy complication of HDP.[6] It is identified by two measurements of arterial pressure greater than 140/90 mmHg taken more than four hours apart and more than 300 mg of urinary protein in a 24-hour period following the twentieth week of pregnancy.[7] Higher D-dimer values suggest that there is significant

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amount of thrombi in the blood vessels. This clarifies the process of endothelial injury in PE, where dissolution of fibrin clot is crucial. Endothelial injury promotes tissue factor expression, platelet activation, and thrombin generation, leading to excessive fibrin deposition within the maternal vasculature and intervillous placental space. Consequently, elevated D-dimer levels in preeclampsia reflect enhanced coagulation turnover and secondary fibrinolysis, paralleling disease severity and the hypercoagulable state characteristic of the disorder.[8]

Khawaja U et al enrolled 60 women aged between 18 to 40 years (30 pre-eclamptic and 30 normotensive women). High d-dimer levels were more common in the case group (pre-eclampsia - 46.67%) than in the normotensive (control group - 13.33%).[9]

The purpose of this study is to examine the mean plasma D-dimer levels in preeclampsia and controls in our local setting because studies done in different populations have revealed variances in its values. D-dimer would be a useful marker for identifying people at risk for serious outcomes, encouraging closer monitoring or preventative measures, if it is demonstrated to be significantly raised in preeclamptic pregnancies. This study will provide contemporary comparative data from a Pakistani tertiary-care setting, where local evidence is limited despite the high burden of preeclampsia. By directly comparing D-dimer levels in preeclamptic and normotensive pregnancies using a uniform methodology, the study will contribute population-specific evidence that may improve understanding of the coagulation abnormalities associated with preeclampsia. To differentiate between preeclamptic and normotensive pregnancies, particularly when symptoms overlap, the study would support the use of D-dimer as a regular marker to monitor pregnancy.

Methodology

This comparative cross-sectional study was performed at Obstetrics and Gynecology department, CMH Multan, from April 2025 to September 2025. This study was approved by the institutional ethical review committee (ERC# 93/2024, dated: 06-01-2025). A minimum sample size of 70 participants was calculated using OpenEpi software by applying the formula for comparison of two proportions, assuming prevalence of high D-dimer levels among pre-eclamptic women as 46.67% and among normal pregnant women as 13.3%,[9] with 80% power and 95% confidence level. Pregnant women 20-40 years of age in 3rd trimester presenting for routine antenatal checkup were screened for eligibility. Thirty-five women each with or without preeclampsia were enrolled after written informed consent through non-probability consecutive sampling. Women with chronic hypertension, smoking history, liver disease, or renal disease were excluded from the study. AT the time of enrollment none of the participant had multiple pregnancy and clinical evidence of venous thromboembolism (DVT, pulmonary embolism), placental abruption, disseminated intravascular coagulation, intrauterine fetal demise were also excluded from the study.

After enrollment, demographic and clinical characteristics including age, gravida, obesity status, and gestational age were recorded. Preeclampsia was defined as pregnancy ≥ 20 weeks gestation based on last menstrual period with blood pressure of ≥140/90 mmHg along with proteinuria of +1 or higher on spot urine dipstick examination. Three milliliters of venous blood was drawn under aseptic conditions in tubes containing trisodium citrate (3.2%). Platelet-poor plasma was prepared immediately by centrifugation at 3000 rpm for 15 minutes at room temperature. D-dimer estimation was performed on freshly separated plasma within 4 hours of sample collection using D-dimer latex kit methodology using MISPA-i2 reagent, and levels > 1.7 µg/ml (normal third trimester range 0.13-1.7) were labeled as high D-dimer levels. The mean BMI of the participants was 29.1 ± 2.2 kg/m², with 77.1% (n=54) classified as obese. The mean BMI was comparable between women with pre-eclampsia and those without pre-eclampsia (29.2 ± 2.2 vs. 28.9 ± 2.3 kg/m², p=0.731).

Data were analyzed using SPSS version 27. Normality of quantitative variable was determined through Shapiro-Wilk test. Age, gestational age, BMI and D-dimers levels were presented as mean ± standard deviation, whereas residence, gravida, obesity, and high D-dimer levels were presented as frequencies and percentages. Comparison of D-dimer levels and prevalence of high D-dimer levels between women with or without preeclampsia was done utilizing independent sample t-test and chi square test respectively, while Fisher's exact test was used where expected cell count was below five. Stratification was carried out for residence, age, gravida, obesity and gestational age to assess their effect on distribution of D-dimer levels and prevalence of high D-dimer between the groups. A p-value < 0.05 was taken as statistically significant.

Results

The mean age of the patients was 30.5±5.2 years and 51.4% were below 30-year. Participants were from rural areas in 61.4%, had mean BMI of 29.1±2.2 kg/m^2 and 54% were obese. The mean gestational age was 32.7±2.3 weeks. The participants were multigravida in 58.6%. The mean D-dimer levels were 0.5±0.7 µg/ml and 12.9% (n=9) of women had high D-dimer levels. Residence, BMI, obesity, gestational age and gravidity were comparable between pregnant women with or without pre-eclampsia. Pregnant women with pre-eclampsia were older than without pre-eclampsia [31.8±5.3 vs. 29.2±4.8, p-value=0.039, ≥30 years: 62.9% vs. 34.3%, p-value=0.017]. Mean d-dimer levels were significantly higher in pregnant women with pre-eclampsia compared to without pre-eclampsia (0.76±0.78 vs. 0.23±0.44, p-value< 0.001). The prevalence of elevated D-dimer levels was significantly higher among pregnant women with preeclampsia than among those without preeclampsia (22.9% vs. 2.9%, p = 0.028; Table I).

After stratification on demographic characteristics, significantly high d-dimer levels were observed in pregnant females with PE if < 30-years of age (0.8 ± 0.8 vs. 0.2 ± 0.3, p-value = 0.015), urban resident (0.8 ± 0.8 vs. 0.1 ± 0.1, p=0.007) and in obese women (0.9 ± 0.8 vs. 0.3 ± 0.5, p-value < 0.001), in contrast to women without pre-eclampsia. Table II. Compared to women without pre-eclampsia, prevalence of high d-dimers (>1.7 µg/ml) was markedly high in pregnant females with PE in the strata of age < 30-years (38.5% vs. 0%, p-value = 0.003), in primigravida (46.2% vs. 6.3%, p-value=0.026) and with obesity (28.6% vs. 3.8%, p=0.025). Table III

Discussion

Fibrin buildup in the walls of tiny blood vessels is a hallmark of preeclampsia. D-dimer was employed as a marker for fibrin formation and breakdown in vivo. Because its plasma concentration has a strong predictive value for the evaluation of venous thromboembolism, it has become a valuable indicator in the diagnosis of thrombotic disorders.[10]

In the present study, mean d-dimer levels and prevalence of high-d-dimers were significantly high in pregnant females with PE in contrast to those without PE. A total of 154 pregnant women were enrolled by Shams N et al. Preeclampsia was present in 37.7% cases, while 62.3% had normal blood pressure. Pre-eclamptic and normal subjects had mean plasma d-dimer levels of 1.02±0.07 and 0.18±0.04 (ng/ml),

Table I: Characteristics of pregnant women during third trimester presenting in antenatal clinic. (N=70)

Characteristics	All (N=70)	With Pre-eclampsia (n=35)	Without Pre-eclampsia (n=35)	p-value
Age (years)	30.5 ± 5.2	31.8 ± 5.3	29.2 ± 4.8	0.039*
<30-year	36 (51.4)	13 (37.1)	23 (65.7)	0.017**
≥30-year	34 (48.6)	22 (62.9)	12 (34.3)	
Residence- Rural	43 (61.4)	21 (60)	22 (62.9)	0.806**
Urban	27 (38.6)	14 (40)	13 (37.1)	
Body Mass Index (kg/m^2)	29.1 ± 2.2	29.2 ± 2.2	28.9 ± 2.3	0.731*
Obesity – Yes	54 (77.1)	28 (80)	26 (74.3)	0.569**
No	16 (22.9)	7 (20)	9 (25.7)	
Gestation (week)	32.7 ± 2.3	32.8 ± 2.3	32.7 ± 2.3	0.917*
Gravida - Primigravida	29 (41.4)	13 (37.1)	16 (45.7)	0.628**
Multigravida	41 (58.6)	22 (62.9)	19 (54.3)	
d-dimer levels (µg/ml)	0.5 ± 0.7	0.76 ± 0.78	0.23 ± 0.44	< 0.001*
High d-dimer – Yes	09 (12.9)	08 (22.9)	01 (2.9)	0.028***
No	61 (87.1)	27 (77.1)	34 (97.1)	

Table II: Effect of demographic characteristics on d-dimer levels between pregnant women with or without pre-eclampsia. (N=70)

Characteristics		With Pre-eclampsia	Without Pre-eclampsia	p-value*
Age Groups	< 30-years	0.8 ± 0.8	0.2 ± 0.3	0.015
	≥ 30-years	0.7 ± 0.7	0.4 ± 0.6	0.221
Residence	Rural	0.7 ± 0.7	0.3 ± 0.5	0.064
	Urban	0.8 ± 0.8	0.1 ± 0.1	0.007
Gravida	Primigravida	1.3 ± 0.8	0.4 ± 0.6	0.002
	Multigravida	0.4 ± 0.6	0.1 ± 0.1	0.013
Obesity	Yes	0.9 ± 0.8	0.3 ± 0.5	< 0.001
	No	0.1 ± 0.04	0.1 ± 0.1	0.281

Table III: Factors affecting the high d-dimer distribution between pregnant women with or without pre-eclampsia. (N=70)

Factors		Study Group	High d-dimer levels		p-value*
			Yes	No	
Age Groups	< 30-year	With pre-eclampsia	5 (38.5)	9 (61.5)	0.003
		Without pre-eclampsia	0 (0.0)	23 (100)	
	≥ 30-year	With pre-eclampsia	3 (13.6)	19 (86.4)	>0.999
		Without pre-eclampsia	1 (8.3)	11 (91.7)	
Residence	Rural	With pre-eclampsia	4 (19.0)	17 (81.0)	0.185
		Without pre-eclampsia	1 (4.5)	21 (95.5)	
	Urban	With pre-eclampsia	4 (28.6)	10 (71.4)	0.098
		Without pre-eclampsia	0 (0.0)	13 (100)	
Gravida	Primigravida	With pre-eclampsia	6 (46.2)	7 (53.8)	0.026
		Without pre-eclampsia	1 (6.3)	15 (93.8)	
	Multigravida	With pre-eclampsia	2 (9.1)	20 (90.9)	0.490
		Without pre-eclampsia	0 (0.0)	19 (100)	
Obesity	Yes	With pre-eclampsia	8 (28.6)	20 (71.4)	0.025
		Without pre-eclampsia	1 (3.8)	25 (96.2)	
	No	With pre-eclampsia	-	7 (100)	--
		Without pre-eclampsia	-	9 (100)	

**Fischer's exact test*

respectively (p<0.001).[11] Our findings are comparable to these results. This hypercoagulable state is probably explained by increased fibrin production and endothelial damage in pregnancy. D-dimer levels were substantially high in the case group compared to controls, according to prospective cohort research by Rodríguez-Peña et al (19.3% vs. 2.3%, odds ratio [OR] 10.30 95% confidence interval [CI] 1.32-80.14, P=0.004).

The severity of preeclampsia was substantially correlated with elevated d-dimer levels.[12] Godtfredsen et al examined fibrinolytic alterations in preeclampsia and found that, in comparison to normal pregnancy, women with preeclampsia had higher circulating fibrin degradation products, such as d-dimer, increased fibrin turnover, and endothelial dysfunction.

D-dimer was significantly increased and plasminogen/Plasmin inhibitor together with fibrin clot lysability time decreased in the PE-group, p = 0.0004 p = 0.04, p = 0.03, p < 0.0001 respectively.[13] This validates our results mechanistically. The marked difference observed in our cohort supports the concept that preeclampsia is associated with pathological activation of the coagulation–fibrinolytic pathway beyond the physiological hypercoagulability of normal pregnancy.

Keepanasseril et al examined biomarkers in severe preeclampsia and discovered that women with severe maternal morbidity had considerably higher levels of coagulation and endothelial dysfunction markers, such as d-dimer. BNP levels were higher among women who developed pulmonary oedema (55.4 pg/mL vs 42.0 pg/mL, p = 0.008).[14] This agreement suggests that increasing D-dimer concentrations reflect progressive activation of the coagulation–fibrinolytic system and endothelial injury in preeclampsia. Although our study did not stratify patients according to disease severity, the significantly higher D-dimer levels support its potential role as an adjunctive biomarker of disease burden.

Xu et al evaluated changes in plasma d-dimer during pregnancy in a prospective study. They showed that whereas d-dimer normally increases with increasing gestation, it increases significantly in pregnancies affected by hypertensive diseases.[15] This validates our findings and highlights the need to take gestational age into account when interpreting d-dimer during pregnancy. Beyond typical pregnancy-related alterations, women with preeclampsia show increased coagulation activation.

Elevated serum d-dimer levels were substantially linked to the later development of preeclampsia, according to Chen et al's analysis of first-trimester predictors of hypertensive disorders in pregnancy. When the cut-off values of DD for the gestational hypertension, PE, and severe PE groups were 0.725, 0.815, and 0.945 MoM (Multiple of the Median), respectively, the corresponding sensitivities were 0.992, 0.962, and 1.000, respectively. This implies that coagulation problems may start early in pregnancies that would eventually result in preeclampsia.[16]

Palomo et al showed significant activation of thrombo-inflammatory pathways, including increase in coagulation indicators such as d-dimer, by comparing endothelial and angiogenic profiles in preeclampsia.[17] These results reinforce the current investigation by emphasizing the inflammatory and thrombotic aspects of preeclampsia. The markedly elevated d-dimer levels seen in affected pregnancies are likely caused by increased endothelial damage and placental hypoperfusion.

Placental ischemia and extensive endothelial dysfunction, which trigger the coagulation cascade and cause excessive fibrin production in the mother's blood, are the hallmarks of preeclampsia.[18] D-dimer levels rise because of secondary fibrinolysis caused by this enhanced fibrin turnover. Therefore, rather than causing preeclampsia, elevated d-dimer reflects its hypercoagulable state.[19] According to the present study, compared with women without pre-eclampsia, the prevalence of elevated D-dimer levels was significantly higher among pregnant women with pre-eclampsia in the age group <30 years and among those with obesity.

The MaTOPs study, conducted by Mandlebe et al, examined coagulation markers in obese pregnant women and found that these women had considerably greater levels of d-dimer and thrombin production.[20] In preeclampsia, obesity may increase fibrin production and fibrinolysis by promoting endothelial dysfunction, chronic inflammation, and hypercoagulability. Inflammatory biomarkers were assessed by Jancsura et al, in obese and overweight pregnant women who subsequently experienced preeclampsia. When compared to obese controls without preeclampsia, the authors discovered that obese women who later developed preeclampsia had considerably higher levels of pro-inflammatory cytokines and endothelium activation indicators.[21] This relates to our results because inflammation associated with obesity probably exacerbates endothelial damage and coagulation activation, which raises d-dimer levels considerably in obese preeclamptic women.

In another study, Shao et al examined coagulation anomalies in preeclamptic pregnancies and discovered a strong correlation between severe maternal problems and raised prenatal d-dimer values. D-dimer levels were markedly greater in women with severe preeclampsia and postpartum hemorrhage (2.02 µg/ml vs. 1.37 µg/ml, p=0.001).[22] This implies that once the condition manifests, the hypercoagulable and endothelial dysfunction pathways of preeclampsia may happen regardless of the mother's age.

The strengths of this study included its comparative design and clearly defined control group of normotensive pregnant women, which strengthened the validity of between-group comparisons Objective biochemical assessment of d-dimer using standardized laboratory method enhanced measurement reliability. Inclusion of both mean d-dimer levels and categorical threshold (>0.5 µg/ml) improved clinical interpretability. Stratified analysis (age, obesity, residence) added depth and identified high-risk subgroups.

The study had a few limitations. Small sample size limited statistical power and generalizability. Single-center study may not reflect broader population diversity. Cross-sectional design precluded causal inference between elevated d-dimer and development of preeclampsia. Lack of severity classification (mild vs severe preeclampsia) limited assessment of dose–response relationship.

In future, larger multi-center studies are needed to validate findings across diverse populations. Prospective longitudinal studies should assess whether elevated d-dimer can predict onset or severity of preeclampsia. Stratification by severity of preeclampsia and gestational age-specific d-dimer reference ranges is recommended.

Conclusion

Preeclampsia in third-trimester pregnancy is associated with elevated mean d-dimer levels and a higher prevalence of abnormal d-dimer positivity, particularly among younger and obese women. These findings highlight the role of enhanced coagulation and fibrinolytic activity in preeclampsia and suggest that d-dimer may be used as an adjunct biomarker for identifying high-risk pregnancies.

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