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Study of Serum Placental Growth Factor (PlGF) Level in Women of Preeclampsia and its Correlation with Fetomaternal Outcome

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ABSTRACT

Background: Preeclampsia is a hypertensive disorder of pregnancy that affects many pregnancies and is a major cause of maternal and neonatal morbidity and mortality. Early prediction of disease severity and adverse outcomes is difficult due to its variable clinical presentation and lack of reliable biomarkers. Placental Growth Factor (PlGF), an angiogenic marker, has emerged as a promising predictor of preeclampsia severity and fetomaternal outcomes. This study aimed to evaluate serum PlGF levels in women with preeclampsia and examine their correlation with maternal and fetal outcomes.

Methods: This prospective study was conducted involving 160 pregnant women between 20- and 40-weeks' gestation, equally divided into preeclampsia and normotensive control groups. Serum PlGF was measured by using ELISA. Participants were monitored till one week postpartum to assess fetomaternal outcomes.

Results: Median serum PlGF levels were significantly lower in preeclamptic women (226.44 pg/ml) compared to controls (282.45 pg/ml, p=0.0001). Both severe and non-severe preeclampsia groups showed reduced PlGF, especially after 29 weeks gestation. Nulliparity, advanced maternal age, and previous stillbirth were linked to lower PlGF levels. Preeclampsia cases had higher rates of fetal growth restriction, preterm birth, caesarean delivery, low Apgar scores, and neonatal intensive care admissions. Low PlGF correlated significantly with these adverse outcomes (p<0.05).

Conclusion: Serum PlGF levels are significantly associated with preeclampsia severity and adverse fetomaternal outcomes in the later stages of pregnancy. Single-point PlGF measurement may serve as a cost-effective biomarker to predict disease severity and improve clinical management in preeclamptic pregnancies.

Key-words: Preeclampsia, Placental Growth Factor, Serum biomarker, Hypertensive Disorders

INTRODUCTION

Preeclampsia is a common hypertensive disorder of pregnancy and a leading cause of maternal and neonatal morbidity and mortality [1]. It affects approximately 3% to 8% of all pregnancies and is associated with serious complications that can result in adverse maternal and perinatal outcomes.

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Clinical prediction of these complications may enable timely management to reduce or avert such outcomes. However, preeclampsia remains a challenging diagnosis due to its variable presentation, unpredictable course, and limited therapeutic options. Despite extensive research, incomplete understanding of its pathophysiology and reliance on established norms continue to hinder progress [2].

The lack of reliable tools to identify high-risk pregnancies has led to conservative clinical approaches, increasing healthcare costs and unnecessary interventions, including iatrogenic preterm births [3]. The emotional and financial burden on affected women and their families is also substantial. Advancing preeclampsia management requires objective tools for accurate diagnosis and risk

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stratification. Over the past 15 years, angiogenic factors such as PlGF and soluble fms-like tyrosine kinase-1 (sFlt-1) have emerged as key placental biomarkers [4,5]. PlGF, a proangiogenic member of the vascular endothelial growth factor (VEGF) family, is predominantly expressed in the placenta and normally increases with gestational age, peaking around 30 weeks [6].

In preeclampsia, a marked reduction in PlGF levels is observed before clinical onset, indicating placental dysfunction [7]. The human PlGF gene, located on chromosome 14q14, encodes four isoforms. PlGF-1 and -2 are the most abundant and are secreted as glycosylated homodimers. These isoforms show correlated secretion patterns during pregnancy, suggesting a shared regulatory mechanism. Although PlGF varies by gestational age, many studies have adopted absolute cutoffs rather than gestational age-specific percentiles, with similar test performance [8]. Current biomarkers such as proteinuria and uric acid have shown limited predictive value for adverse outcomes in preeclamptic women. PlGF, produced exclusively by the trophoblast, has been consistently found to be reduced in such patients. Given its pregnancy-specific expression and association with placental health, this study was conducted to evaluate the predictive performance of serum PlGF in preeclamptic women and its correlation with fetomaternal outcomes.

MATERIALS AND METHODS

Study Design and Ethical Approval- The study was conducted in the Department of Obstetrics and Gynaecology at King George's Medical University, Lucknow, over one year after obtaining ethical clearance from the institutional ethical committee (ethical number ECR/262/Inst/UP/2013/RR19). In this prospective observational study, Participants were recruited from antenatal women attending Queen Mary Hospital between 20 and 40 weeks of gestation. Informed written consent was taken from each participant.

Inclusion criteria- Inclusion criteria for the study group consisted of women aged 18 to 40 years with singleton pregnancies at 20 weeks gestation or more, who fulfilled the diagnostic criteria for preeclampsia at the time of delivery and provided informed consent. Women were excluded if they had multiple pregnancies, gestational age below 20 weeks, declined to participate, or had chronic hypertension, cardiovascular disease, or any other chronic illness.

Exclusion criteria- Exclusion criteria were multiparity, gestational <20 weeks pts not willing to give consent, chronic hypertension, cardiovascular disease and any other chronic illness.

Methodology- A total of 160 women were enrolled in the study and divided into two groups: 80 normotensive pregnant women as the control group and 80 women with preeclampsia as the study group. Women in the control group had singleton pregnancies with a gestational age of 20 to 40 weeks and remained normotensive throughout. The study group included women with singleton pregnancies and preeclampsia, defined as blood pressure≥140/90 mmHg on two occasions at least four hours apart, with or without proteinuria ≥300 mg/24 hours or persistent proteinuria ≥30 mg/dL (>1+ on dipstick) in random urine samples, as per criteria outlined in Williams Obstetrics, 24th edition [9]. After enrollment, all participants underwent detailed history taking and clinical examination. A 5 ml venous blood sample was collected from each woman and the samples were allowed to clot, serum was separated by centrifugation at room temperature. Serum samples were stored at −20°C until analysis. PlGF levels in the serum were estimated using a commercially available ELISA kit (BT LAB–Bioassay Technology Laboratory). For analysis, a standard curve was generated by plotting the average optical density (OD) for each standard on the vertical (Y) axis against concentration on the horizontal (X) axis. A best-fit curve was drawn using computer-based software, and results were calculated using regression analysis. All participants were followed for up to one week postpartum for outcome assessment.

Statistical Analysis- The statistical analysis was done using SPSS Version 24.0. As PlGF levels were non-normally distributed (Kolmogorov-Smirnov test significant), median and interquartile ranges were used. Mann-Whitney test compared PlGF between cases and controls, while the Kruskal-Walli's test was used for NSPE, SPE, and control groups. The chi-square test assessed associations between categorical variables. A p-value<0.05 was considered statistically significant.

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RESULTS

Eighty women with preeclampsia were categorized as Group A, including 46 with non-severe (NSPE) and 34 with SPE. An equal number of normotensive pregnant women were included in Group B. The mean ages in Groups A and B were 28.21±4.34 and 28.30±5.07 years, respectively, with no significant difference. Nulliparity was significantly higher in Group A (53.8%) compared to Group B (30.0%). Most participants (68.1%) were vegetarian, and socioeconomic, education, and booking status were comparable across groups. BMI ranged from 20.0 to 31.4 kg/m² and showed no significant difference. Gestational age at specimen collection was also similar (p=0.12) (Table 1).

Table 1: Socio-Demographic Parameters of Patients among Groups

Socio-demographics		Total (n=160)	Group A (n=80)		Group B (n=80)		p-value
			No.	%	No.	%	
Age Group (years)	18-25 yrs	44(27.5%)	21	26.3	23	28.8	χ^2=1.58; p=0.45
	26-35 yrs	102(63.8%)	54	67.5	48	60.0	
	>35 yrs	14(8.8%)	5	6.3	9	11.3	
Parity	G1	67	43	53.8	24	30.0	χ^2=9.27; p=0.01
	G2	43	17	21.3	26	32.5	
	G3+	50	20	25.0	30	37.5	
Socio-economic status	Lower	60	29	36.3	31	38.8	χ^2=0.10; p=0.74
	Middle	100	51	63.8	49	61.3	
Educational Status	Illiterate	23	13	16.3	10	12.5	χ^2=0.51; p=0.77
	School	101	50	62.5	51	63.8	
	College	36	17	21.3	19	23.8	
Nutritional Status (BMI kg/m2)	Normal (18.5-24.9)	64	32	40.0	32	40.0	χ^2=0.34; p=0.84
	Overweight (25-29.9)	93	46	57.5	47	58.8	
	Obese (≥30)	3	2	2.5	1	1.3	
Gestational Age (weeks)	20-28 weeks	23	16	20.0	7	8.8	χ^2=4.15; p=0.12
	29-32 weeks	63	30	37.5	33	41.3	
	33 weeks-Term	74	34	42.5	40	50.0	

The median PlGF level was significantly lower in Group A (226.44) as compared to Group B (282.45). Within Group A, NSPE and SPE showed similar PlGF levels (228 and 222.89, respectively), both significantly lower than controls (Table 2).

Table 2: Comparison of PlGF (pg/ml) among Groups.

Group	n	Median	IQR	p-value*
Group A	80	226.44	124.32	0.0001
Subgroup A1 (NSPE)	46	228.0	132.68	0.001
Subgroup A2 (SPE)	34	222.89	121.87	
Group B	80	282.45	94.9	

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Between 20–28 weeks, PlGF levels did not differ significantly among subgroups. However, at 29–32 and 33–38 weeks, PlGF levels were significantly lower in preeclampsia subgroups compared to controls (Table 3).

Table 3: Comparison of PlGF among the Cases and Control Groups Based on the Gestational Age.

Gestational Age	PIGF Median (IQR)			p-value*
	SPE	NSPE	Control	
20-28 wks	195.85 (97.1)	210.05 (105.34)	292.53 (53.85)	0.46
29-32 wks	220.12 (96.19)	233.0 (112.75)	290.05 (66.78)	0.04
33-38 wks	226.73 (92.4)	241.71 (124.62)	276.69 (107.42)	0.04

**Krusal wallis test*

Fetal growth restriction was significantly higher in SPE (2.4%) compared to NSPE (17.4%) and controls (12.5%). Advanced maternal age (>35 years), nulliparity, and previous stillbirth were significantly associated with lower PlGF levels. Risk factors such as GDM, BMI >30, and ART were not significantly associated. Vaginal delivery was more common in controls (43.8%) than in NSPE (32.6%) and SPE (17.6%), while caesarean rates were significantly higher in Group A. HELLP syndrome was more frequent in NSPE (8.7%) and was significantly associated with low PlGF. Preterm birth was significantly more common in SPE and NSPE than in controls. Low Apgar scores at 5 minutes and NNU admissions were also higher in Group A, particularly SPE. Stillbirth (n=9) and neonatal death (n=3) were more common in Group A but not statistically significant. PlGF levels were significantly associated with preterm delivery, low Apgar, NNU admission, and neonatal death ($p<0.05$) (Tables 4 and 5).

Table 4: Comparison of Risk Factors, Maternal, and Neonatal Outcomes among Subgroups of Group A and Group B

Variables		Group A				Group B (n=80)		Significance of difference	
		Subgp A1(n=46)		SubgpA2(n=34)					
		No.	%	No.	%	No.	%	χ^2	'p'
Risk Factors	H/o pre-eclampsia	1	2.2	1	2.9	0	0.0	2.11	0.34
	GDM (Gestational Diabetes Mellitus)	2	4.3	4	11.8	9	11.3	1.92	0.38
	Advanced age (>35 yrs)	4	8.7	1	2.9	9	11.3	2.06	0.35
	BMI >30 kg/m^2	1	2.2	1	2.9	1	1.3	0.40	0.81
	Nullipara	31	67.4	23	67.6	34	42.5	10.10	0.006
	FGR (Fetal growth restriction)	8	17.4	11	32.4	10	12.5	6.36	0.04
	Asstt. Reprod Tech.	2	4.3	0	0.0	0	0.0	5.01	0.08
	Prev. Still birth	3	6.5	3	8.8	3	3.8	1.25	0.53

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Maternal Outcome	Vaginal Normal delivery	15	32.6	6	17.6	35	43.8	7.30	0.02
	Instrumental delivery.	0	0.0	0	0.0	2	2.5	2.02	0.36
	Caesarean section	31	67.4	28	82.4	43	53.8	8.81	0.01
	HELLP syndrome	4	8.7	0	0.0	1	1.3	6.74	0.03
	Placental abruption	0	0.0	2	5.9	3	3.8	2.44	0.29
	Eclampsia	1	2.2	2	5.9	0	0.0	4.51	0.10
	Pulmonary edema	0	0.0	1	2.9	1	1.3	1.37	0.50
	Maternal death	1	2.2	0	0.0	0	0	1.006	0.60
Neonatal Outcome	Preterm delivery	19	41.3	21	61.8	19	23.8	15.30	<0.001
	FGR	8	17.4	11	32.4	10	12.5	6.36	0.04
	Apgar <7 at 5 min	4	8.7	6	17.6	3	3.8	6.20	0.04
	NNU Adm.	2	4.3	5	14.7	0	0.0	12.30	0.002
	Still Birth	3	6.5	4	11.8	2	2.5	3.95	0.13
	Neonatal death	1	2.2	1	2.9	1	1.3	1.25	0.53

Table 5: Comparison of Risk Factors, Maternal, and Neonatal Outcomes Between Cases and Controls with Corresponding Median (IQR) Values.

Variables			Cases	Controls	p-value
Risk Factors	History of Pre-eclampsia	n	3	0	-
		Median (IQR)	139.0(86.3)	-	
	GDM	n	13	2	0.305
		Median (IQR)	194.3(146.6)	250.5(88.6)	
	Advanced age >35 yrs	n	5	56	0.0001
		Median (IQR)	225.3(118.6)	291.7(89.3)	
	BMI>30	n	1	2	0.063
		Median (IQR)	211.9	231.4	
	Nulliparity	n	57	31	0.019
		Median (IQR)	179.9(133.1)	284.2(91.3)	
	FGR	n	21	8	0.059
		Median (IQR)	227.1(84.01)	281.95(108.6)	
	Assisted Reproduction Technique	n	0	2	-
		Median (IQR)	-	253.6(89.6)	
	Previous still birth	n	6	3	0.048
		Median (IQR)	208.5(83.2)	318.6(53.64)	

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Maternal Outcome	Vaginal delivery	n	32	24	0.476
		Median (IQR)	228.9(100.9)	272.7(92.5)	
	Instrumental delivery	n	2	2	0.047
		Median (IQR)	226.4(125.6)	284.3(95.9)	
	Caesarean delivery	n	66	36	0.002
		Median (IQR)	228.9(117.7)	279.6(102.7)	
	HELLP	n	2	4	0.027
		Median (IQR)	226.01(126.7)	214.5(132.0)	
	Placenta abruption	n	4	1	-
		Median (IQR)	226.01(126.7)	-	
	Eclampsia	n	3	0	-
		Median (IQR)	227.1(128.5)	-	
	Pulmonary oedema	n	2	0	-
		Median (IQR)	226.9(126.9)	-	
Neonatal Outcome	Preterm	n	36	23	0.049
		Median (IQR)	182.71(106.9)	264.6(58.9)	
	Term	n	44	57	0.289
		Median (IQR)	233.1(90.7)	294.35(101.28)	
	FGR	n	21	8	0.059
		Median (IQR)	227.1(84.01)	281.95(108.6)	
	No FGR	n	59	72	0.195
		Median (IQR)	226.1(132.1)	282.5(90.37)	
	APGAR <7 at 5 mins	n	53	21	0.026
		Median (IQR)	209.2(137.3)	284.3(123.8)	
	APGAR >7 at 5 mins	n	27	59	0.518
		Median (IQR)	226.9(126.79)	280.6(104.3)	
	NNU admission	n	5	2	0.039
		Median (IQR)	226.8(137.9)	206.85(124.5)	
	No NNU admission	n	75	78	1.000
		Median (IQR)	226.01(126.7)	286.5(86.11)	
	Still Birth	n	5	3	0.143
		Median (IQR)	219.06(123.7)	264.6(112.3)	
	Live Birth	n	75	77	0.085
		Median (IQR)	226.4(129.6)	284.3(95.01)	
	Neonatal Death	n	1	2	0.033
		Median (IQR)	95.35	-	

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DISCUSSION

Preeclampsia is a hypertensive disorder affecting approximately 3% of singleton pregnancies and significantly contributes to maternal and perinatal morbidity and mortality [10]. It lies on a spectrum of hypertensive disorders that include HELLP syndrome and eclampsia [11]. Around 10% of all pregnancies are affected by hypertension, leading to complications such as preterm birth and perinatal death [10]. PlGF, an angiogenic marker, has shown promise in predicting disease severity [12].

This study, which was conducted at Queen Mary Hospital in collaboration with the Department of Pathology, KGMU, investigated serum PlGF levels in preeclamptic women and their correlation with fetomaternal outcomes, addressing the limitations of conventional markers like proteinuria and uric acid.

The mean age of participants was 28.26±4.70 years (range 18–42), with no significant difference between cases and controls. Similar age distributions were reported by Agarwal *et al.* [13] and others [14], likely due to early marriage and conception in India. A Finnish study also reported similar mean ages (controls 29±5, PE 28±6) [15], while higher means were seen in studies from Iran and Canada [16,17], possibly due to nulliparity and regional differences. Nulliparity was significantly more common among cases (53.8%), especially in NSPE (67.4%) and SPE (67.6%), compared to controls (42.5%). Similar findings were observed by Amsaveni *et al.* [14] confirming nulliparity as a major risk factor. Maeda *et al*. [18] further noted multiparity as a protective aOR: 0.08).

Socioeconomic status plays a vital role in pregnancy outcomes. In our study, 62.5% belonged to middle SES, and 58.8% of cases were unbooked. Similarly Low SES and unbooked status were linked to increased risks of preterm birth, and preeclampsia in the study done by Lee *et al.* [19]. Amsaveni *et al.* [14] also reported higher PE rates in rural, low-SES, and unbooked women. Ross *et al.* [20] echoed these findings.

Most participants (57.5%) had a BMI between 25–29.9 kg/m², and 40% were in the 18.5–<23 range. The mean BMI among cases was 25.54±1.92 kg/m². Similar distributions were reported by Agarwal *et al.* [13] and Keikkala *et al.* [15]. High BMI is a known risk factor for PE, as noted by Zhang *et al.* [21]. Median PlGF levels were significantly lower in cases (226.44) than in controls (282.45), with NSPE and SPE subgroups showing medians of 228 and 222.89, respectively (p=0.0001). PlGF levels normally peak at 30 weeks before declining, but are consistently lower in PE, as shown by Nabweyambo *et al.* [22] and others [13,15].

Preeclampsia is linked to maternal complications like seizures, cerebral hemorrhage, DIC, liver rupture, and obstetric issues such as placental abruption and cesarean delivery [11]. Fetal complications include IUGR, oligohydramnios, and stillbirth, with PE contributing to 15–20% of fetal growth issues and 20% of preterm births [23]. Long-term maternal risks include stroke and hypertension. NICE [11] guidelines classify women with chronic kidney disease or prior hypertensive pregnancies as high-risk, while nulliparity, age >40, BMI ≥35, family history, and long inter-pregnancy intervals fall under moderate risk [24]. Bartsch *et al.* [25] confirmed these risk factors in a review of over 25 million pregnancies. Wadhwani *et al.* [26] identified nulliparity and prior preeclampsia (PE) as risk factors for late-onset preeclampsia (PE). In our study, significant risk factors for low PlGF were age >35, nulliparity, and history of stillbirth (p=0.0001, 0.019, 0.048). Maternal outcomes associated with PlGF included vaginal delivery, cesarean section, and HELLP syndrome (p=0.026, 0.012, 0.034).

Cesarean rates were higher among cases (73.75%) than controls (53.8%). Similar trends were noted by Amsaveni *et al.* [14] and others [27]. Being a tertiary referral center likely contributed to the high cesarean rate. McLaughlin *et al.* [28] also linked low PlGF to low birth weight, fetal complications, and emergency C-sections. Our results may reflect the gestational age at sample collection (33–38 weeks), as PlGF levels plateau in late pregnancy.

Indian studies assessing PlGF in PE are limited. Ghosh *et al*. [29] found that while both uterine artery Doppler (UADV) and second-trimester PlGF predicted PE individually, the combination was not beneficial. Fetal complications significantly associated with PlGF included preterm delivery, FGR, low 5-minute APGAR (<7), and NNU admission (p<0.001, 0.04, 0.04, 0.002), with preterm birth being the most common. Similar rates were seen in Amsaveni *et al.* (14) (20.5%), while Sultana *et al.* [30] and Ahmed *et al.* [31] reported higher rates. Causes included spontaneous labor, induction, or LSCS. In our study, 23.75% of neonates born to pre-eclamptic women had FGR, consistent with Amsaveni *et al.* [14] (18.5%), while higher rates were reported by Sultana *et al.* [30] (50%). FGR in PE is due to placental insufficiency.

Fetal outcomes significantly associated with low PlGF included preterm labor, APGAR <7 at 5 minutes, NNU admission, and neonatal death (p=0.04, 0.026, 0.03, 0.03). Amsaveni et al [14] noted 10.5% perinatal mortality, compared to 8% in our study. Singh *et al.* [32] reported higher rates (12.5%). A lower rate was seen in Al-Mulhim *et al.* [33] (3.36%). Differences likely reflect healthcare access. Common causes were asphyxia, prematurity, and IUGR. McLaughlin *et al.* [28] found that low PlGF increased the odds of birth weight <10th percentile (6.4x) and APGAR <6 at 5 minutes (5.8x). Audette *et al.* [17] showed that second-trimester low PlGF (<72 pg/ml) led to higher risks of preterm birth (5.75x), SGA (2.6x), and PE (4.3x), with significantly lower mean birth weight. While many studies used multiple markers, our study relied solely on PlGF, measured once during pregnancy, to differentiate severe from non-severe PE—offering a more cost-effective approach.

CONCLUSIONS

This study demonstrates the significant association between serum PlGF levels and the severity of preeclampsia during the second and third trimesters of pregnancy. Assessment Low PlGF was significantly associated with instrumental delivery, cesarean section, and HELLP syndrome (p=0.047, 0.002, 0.027). of PlGF effectively differentiated between severe and non-severe preeclampsia, highlighting its potential as a cost-effective biomarker for predicting fetomaternal outcomes. Furthermore, by

exploring the relationship between PlGF levels and key maternal risk factors, the study contributes valuable evidence to the limited body of research in this area. However, the study has certain limitations. As the data were collected from a single tertiary care center, the generalizability of the findings may be limited. Future multicentric studies with larger sample sizes and serial PlGF assessments are recommended to validate these findings and further explore the utility of PlGF in preeclampsia screening and management.

CONTRIBUTION OF AUTHORS

Research concept- Dr Vandana Solanki
Research design- Dr Vandana Solanki
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