"ASSOCIATION OF SERUM PLACENTAL GROWTH FACTOR IN PRE-ECLAMPSIA AND FGR WITH MATERNAL AND PERINATAL OUTCOMES-A ONE-YEAR CROSS SECTIONAL STUDY."

BY

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Dissertation

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Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"ASSOCIATION OF SERUM PLACENTAL GROWTH FACTOR IN PRE-ECLAMPSIA AND FGR WITH MATERNAL AND PERINATAL OUTCOMES- A ONE YEAR CROSS SECTIONAL STUDY",** is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee.

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(Dr. Harsha Hegde)
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LIST OF ABBREVIATIONS USED

GLOSSARY	ABBREVIATIONS
ACOG	AMERICAN COLLEGE OF OBSTETRICIANS AND GYNECOLOGIST
AKI	ACUTE KIDNEY INJURY
DIC	DISSEMINATED INTRAVASCULAR COAGULATION
HDP	HYPERTENSIVE DISORDERS OF PREGNANCY
HELLP	HEMOLYSIS, ELEVATED LIVER ENZYMES, LOW PLATELET
ISSHP	INTERNATIONAL SOCIETY FOR THE STUDY OF HYPERTENSION IN PREGNANCY.
PlGF	PLACENTAL GROWTH FACTOR
PT-INR	PROTHROMBIN TIME-INTERNATIONAL NORMALISED RATIO
sFlt-1	SOLUBLE FMS – LIKE TYROSINE KINASE-1
TNF	TUMOR NECROSIS FACTOR
VEGF	VASCULAR ENDOTHELIAL GROWTH FACTORS
WHO	WORLD HEALTH ORGANISATION
ALP	ALKALINE PHOSPHATASE
APLA	ANTI PHOSPHO LIPID ANTIBODY
BMI	BODY MASS INDEX

FGR	FETAL GROWTH RESTRICTION
IL	INTERLEUKIN
LFT	LIVER FUNCTION TEST
PE	PRE- ECLAMPSIA
PPH	POST PARTUM HEMORRHAGE
SGOT	SERUM GLUTAMIC OXALOACETIC TRANSAMINASE
SGPT	SERUM GLUTAMIC PYRUVIC TRANSAMINASE
SLE	SYSTEMIC LUPUS ERYTHEMATOSUS
EO	EARLY ONSET
LO	LATE ONSET
USG	ULTRASONOGRAPHY

ABSTRACT

Background and Objectives

Preeclampsia and fetal growth restriction (FGR) are significant contributors to maternal and perinatal morbidity and mortality. These conditions are often rooted in placental dysfunction, which may be reflected in angiogenic markers such as Placental Growth Factor (PlGF). Low levels of PlGF have been associated with poor placental perfusion, increased disease severity, and adverse maternal and fetal outcomes. The objective of this study was to evaluate the association of serum PlGF levels in pregnant women with preeclampsia and FGR and maternal and perinatal outcomes.

Methods

This was a cross-sectional study conducted at the Department of Obstetrics & Gynaecology, KAHER's Dr. Prabhakar Kore Hospital, Belagavi. A total of 94 pregnant women diagnosed with preeclampsia were included. Detailed clinical data, PlGF levels, Doppler findings, and maternal and perinatal outcomes were studied. PlGF levels were categorized as <12 pg/mL, 12–50 pg/mL, 51–100 pg/mL, and >100 pg/mL. Associations between PlGF levels and various clinical parameters were statistically analyzed using Chi-square and appropriate tests.

Results

The majority of participants were aged between 21–25 years (44.7%) and primigravida (60.6%).

62.8% had abnormal PlGF levels, and 38.3% of the study population was diagnosed with FGR. Abnormal Doppler was noted in 27.7%, and preterm delivery (<37 weeks)

occurred in nearly 70% of cases. Regarding severity, 46.8% had preeclampsia without severe features, 43.6% had severe preeclampsia, and 9.6% had eclampsia. HELLP syndrome (11.7%), placental abruption (6.4%), PPH (2.1%), and renal failure (1.1%) were among the maternal complications observed. Neonatal outcomes included 11.7% ELBW, 18.1% VLBW, and 41.5% NICU admissions. Statistically significant associations were found between low PlGF levels and: Severity of preeclampsia (p=0.001), FGR (p=0.001), Abnormal Doppler (p=0.001). NICU admissions and low birth weight (p=0.001)

Conclusion

The study demonstrates that low serum PlGF levels are significantly associated with increased severity of preeclampsia, fetal growth restriction, abnormal Doppler findings, and adverse neonatal outcomes including low birth weight and NICU admission. The findings underscore the value of PlGF as a reliable biomarker in the prediction and risk stratification of pregnancies complicated by placental insufficiency. Incorporating PlGF testing into routine obstetric care can improve clinical decision-making, allowing for early intervention and better maternal-fetal outcomes.

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INTRODUCTION

The abbreviation HDP refers to hypertension illness of pregnancy, and preeclampsia is one of the subgroups of this condition. In addition to having a significant contributor to the morbidity and death rates of both mothers and their newborns, it is estimated that between two percent and eight percent of all pregnancies encounter this condition[1-3]. Preeclampsia is a complicated sickness process that begins at the interface between the mother and the foetus. It can impact a variety of organ systems, and it usually manifests itself in the first few weeks of pregnancy[4,5]. The hypertension that is characteristic of the syndrome is commonly, but not always, accompanied with proteinuria throughout the course of the ailment. This is the hallmark of the syndrome. There are a number of consequences that can result from severe types of preeclampsia, including renal, cardiac, pulmonary, hepatic, and neurological dysfunction; haematologic abnormalities; foetal growth limitation; stillbirth; and maternal death[3,6].

These pregnancy-related illnesses provide a particularly significant challenge due to the fact that the pathophysiology and therapeutic management of hypertensive diseases of pregnancy, such as chronic hypertension, gestational hypertension, and preeclampsia, impact both the mother and the foetus at the same time with the same symptoms. When it comes to the issues that are most feared during pregnancy, preeclampsia stands out as one of the most alarming of them all. Preeclampsia may continue to exist after delivery and, in some cases, may develop from scratch during the postpartum period. It is possible for the majority of signs and symptoms to diminish after birth; however, preeclampsia may continue to exist after delivery.[7]

Table 29: Highlights of Clinical Features of Preeclampsia[8]

Clinical Feature	Underlying Abnormalities	Clinical Consequences
Hypertension	Increased SVR and afterload Decreased CO and intravascular volumes Activation of RAAS, ET-1, SNS AT1R down-regulated, placental hypoxia, and AT1R autoantibodies Increased vasoconstrictors, decreased vasodilators Increased sFlt-1 and sEng, oxidative stress	Heart failure Pulmonary edema Renal dysfunction Neurological injury
Proteinuria	Glomerular endotheliosis Disruption of filtration barrier Increased tubular permeability	Hypertension Ischemic heart disease Stroke Chronic kidney disease End-stage renal disease
Renal dysfunction	Decreased RBF and GFR Glomerular endotheliosis Increased tissue factor expression Thrombotic microangiopathy	Hypertension Chronic kidney disease End-stage renal disease
Neurological abnormalities	Headache: loss of fenestrae on choroid plexus, periventricular edema, vasogenic edema in posterior cerebral circulation Visual disturbances: retinopathy, retinal detachment, cortical blindness, central serous chorioretinopathy, hypertensive retinopathy, diabetic retinopathy	Seizures PRES Permanent blindness
Eclampsia	Unknown (potentially vasogenic or cytotoxic edema)	Permanent neurological dysfunction
Cardiac dysfunction	Increased SVR, afterload Concentric LV hypertrophy, LA enlargement Increased RVSP, increased LV filling pressures, LV diastolic dysfunction,	Heart failure Peripartum cardiomyopathy
Pulmonary edema	Increased vascular permeability Cardiac dysfunction Corticosteroids/tocolytics Iatrogenic volume overload	Acute hypoxemic respiratory failure
Hepatic dysfunction	Hepatic microcirculatory deterioration, hepatocellular injury	Liver failure, hepatic rupture
Hematologic dysfunction	Procoagulant state	Thrombocytopenia, DIC
Fetal growth restriction	Incomplete spiral artery remodeling Decidual vasculopathy Uterine and placental dysfunction	Fetal growth <10th percentile

PLGF

The placenta is the primary organ in which it is expressed, and it belongs to the family of vascular endothelial growth factor (VEGF). On the other hand, it is also expressed in a wide variety of other tissues, including the heart, lung, thyroid, liver, skeletal muscle, and bone, but only at low levels. The placenta is responsible for the production of placental growth factor. The human PlGF gene, which can be located on chromosome 14q14, is responsible for encoding a total of four distinct isoforms of the protein known as PlGF. PlGF-1 and PlGF-3 are isoforms of the protein that are diffusible, and the protein is secreted as a glycosylated homodimer. The PlGF-2 and PlGF-4 proteins, on the other hand, feature regions that are capable of binding heparin. Among them, the PlGF-1 and PlGF-2 variations are the most frequent, and during pregnancy, they are secreted in a manner that is significantly associated with

one another, which indicates that they share a common regulatory mechanism. Placental growth factor binds to a number of different proteins, including VEGFR-1 (vascular endothelial growth factor-1 receptor-1), FLT-1 (fms-related tyrosine kinase-1), and its soluble form, or sFLT-1 (soluble fms-like tyrosine kinase-1) reducing its bioavailability. On the other hand, it does not bind to VEGFR-2, which is also known as KDR (kinase insert domain receptor) or FLK-1 (foetal liver kinase-1). VEGFR-2 is a receptor for vascular endothelial growth factor. In addition to this, it is associated with neuropilin receptor-1 (NP-1) and -2, both of which are located in neurones. Furthermore, NP-1 was recently found in the placenta; however, the function of this protein has not yet been defined. NP-1 was discovered quite recently.[9,10]

Not only does it participate in the process of angiogenesis, but it also plays a role in the proliferation and differentiation of trophoblasts after it has already been involved in the process. Pre-eclampsia and growth restriction are two complications that can arise later in pregnancy as a consequence of inadequate uteroplacental development[11,12]. For there to be an adequate invasion of extravillous trophoblast cells into the uterine wall and the maternal spiral arteries, it is necessary for a greater blood flow and a lower resistance. This is because it is necessary for there to be a higher blood flow. When a pregnancy is regarded to be normal, the concentrations of PlGF remain low throughout the first trimester of the pregnancy and then gradually peaks after that. This pattern continues until half of the pregnancy has passed. After reaching their highest point, which occurs during the thirty-first week of pregnancy, they begin to decrease after reaching their peak. Women who are in the clinical phase of preeclampsia as well as those who are in the preeclampsia phase have been found to have lower levels of PlGF[12,13]. In order to facilitate the process of diagnosing pre-eclampsia, the serum marker has been the focus of attention. When it comes to the

diagnosis of preeclampsia in women who are suspected of having preeclampsia and who require delivery within 14 days, it has been demonstrated that relatively low circulating maternal PlGF concentrations (below the fifth centile or less than or equal to 100 pg/ml) have a high sensitivity (96%; 95% confidence interval [CI]: 89–99) and a negative predictive value (98%; 93–99 5) in the diagnosis of preeclampsia[14].

It has been proven that women who are already experiencing pre-eclampsia have higher amounts of maternal serum that are circulating in their bodies. The circulating anti-angiogenic protein known as sFLT1 is able to inhibit the activity of both VEGF and PlGF. It is a protein that functions as an antagonist. Due to this, endothelial dysfunction occurs, which can lead to pre-eclampsia as well as growth limitation[15]. An higher ratio of sFlt-1 to PlGF has been reported to be associated with an increased risk of preeclampsia, and it may perform better than PlGF alone[16]. This was discovered through research. It has been observed that a sFlt-1:PlGF ratio cut-off of 38 had a negative predictive value (no pre-eclampsia in the subsequent one week) of 99.3% (95% CI, 97.9–99.9), with 80.0% sensitivity (95% CI, 51.9–95.7) in women with suspected pre-eclampsia between 20 and 36 plus 6 weeks[15]. The National Institute for Health and Care Excellence (NICE) has expressed their recommendation that a sFlt-1/PlGF ratio of 33 can be utilised as a rule-out cutoff between the ages of 33 and 6 weeks[17]. Despite the fact that PlGF and the sFlt-1/PlGF ratio are proposed by the National Institute for Health and Care Excellence (NICE) as ruleout tests for pre-eclampsia, it is not currently recommended for routine adoption to rule in or diagnose pre-eclampsia due to the lack of sufficient evidence. This is because of the fact that further research is required. It is necessary to conduct additional study in order to evaluate the utilisation of repeat PlGF-based testing in the case of women who present with a suspicion of pre-eclampsia and have previously received a negative result.

Furthermore, it is essential to explore the ways in which a positive PlGF-based test result, which is used to rule out pre-eclampsia, may alter management decisions for the date of delivery and the outcomes that are connected with this outcome. [17]

Table 30 : NICE's recommended cut-off values for PlGF testing[18]

Result	Classification	Interpretation
PlGF <12 pg/ml	Test positive – highly abnormal	Suggestive of severe placental dysfunction and at increased risk for preterm delivery
PlGF ≥12 pg/ml and < 100 pg/ ml	Test positive – abnormal	Suggestive of placental dysfunction and at increased risk for preterm delivery
PlGF ≥100 pg/ ml	Test negative – normal	Suggestive of no placental dysfunction and unlikely to progress to delivery within 14 days of the test

Need for the study: Pre-eclampsia is a multi-system pregnancy-specific condition that affects between two and ten percent of pregnant women. It is responsible for more than seventy thousand maternal deaths and five hundred thousand fetal deaths annually[7]. The diagnosis of pre-eclampsia can be difficult because women frequently do not exhibit any symptoms of the condition. Furthermore, the clinical and biochemical parameters are frequently not predictive of unfavorable outcomes experienced by the mother or the postnatal period. This results in an increased demand on resources and concern among the mother. VEGF, which is a member of the vascular endothelial growth factor family, is a protein that is expressed in the placenta and is linked to the process of angiogenesis.

PlGF begins to reach its peak around the thirty-week mark, and it is discovered that it is lower in women before the development of pre-eclampsia and during the clinical phase of the condition. Recently, there has been a considerable amount of focus placed on angiogenic factors, which include placental growth factor (PLGF), among biochemical markers. The most important findings lend credence to the concept that faulty placentation is caused by a pathogenetic model, which is characterised by decreased concentrations of angiogenic growth factors (free PLGF) and increased concentrations of anti-angiogenic factors (sFLT-1). The maternal free PLGF concentrations of preeclamptic patients have been reported to decrease significantly when compared to those of non-preeclamptic patients, according to case-control scientific research. In addition, results from a study conducted by Levine and colleagues demonstrated that the decrease in free PLGF concentrations occurred several weeks before to the clinical diagnosis[40].

AIMS AND OBJECTIVES

- Primary objective- To study the association of Serum PlGF in cases of pre-eclampsia, eclampsia with or without FGR.
- Secondary objective- To study the maternal and foetal outcomes in the cases of pre- eclampsia, eclampsia with or without FGR.

REVIEW OF LITERATURE

Pre-eclampsia is a multisystem progressive illness that is distinguished by the new onset of hypertension and proteinuria or the new onset of hypertension in addition to severe end-organ dysfunction with or without proteinuria. Pre-eclampsia can occur even in the absence of proteinuria. It is common for the signs of this condition to become apparent after twenty weeks of gestation or after the person has given birth. It is estimated that approximately ninety percent of cases take place during the late preterm period (when the baby is between 34 and 37 weeks), during the term period, or during the postpartum process. However, there is a potential that the mother and/or the perinatal period could experience a significant illness or perhaps that they will pass away. The other ten percent of cases are associated with higher chances of serious perinatal morbidity or mortality due to the dangers associated with moderately preterm, very preterm, or extremely preterm birth. This is because of the risks that are connected with these types of birth defects. The early presentation of these cases, which takes place prior to the 34th week of gestation, provides a distinguishing feature. Despite the fact that preeclampsia always wanes off on its own within the first few days or weeks after birth, those who have a history of preeclampsia are at a higher risk for cardiovascular-related morbidity and mortality throughout their lifetime. This is the case even if the sickness always disappears on its own. During pregnancy, a woman is considered to have high blood pressure if she has a systolic blood pressure of at least 140 mmHg and/or a diastolic blood pressure of at least 90 mmHg. The definition of severe hypertension includes having a systolic blood pressure of 160 mmHg or higher and/or a diastolic blood pressure of 110 mmHg or higher. Both of these blood pressure readings must be greater than normal. A lower threshold for diagnosing hypertension is regarded to be present in individuals who are

not pregnant and have a systolic blood pressure of at least 130 mmHg or a diastolic blood pressure of at least 80 mmHg. These individuals are considered to have a lower threshold. Furthermore, the American College of Cardiology and the American Heart Association have also acknowledged the significance of this threshold. However, there are some individuals who have raised the possibility that this particular definition might also be appropriate for patients who are pregnant[20]. Concerning the utilisation of the lower threshold, on the other hand, there has not been comprehensive research conducted. There would be an increase of approximately ten percent in the number of cases of hypertension that develop during pregnancy, according to estimates. In addition to this, it would result in an increase in the number of tests, hospitalisations, and interventions that might not be necessary, despite the fact that there is no evidence that they are beneficial. A meta-analysis came to the conclusion that lowering the blood pressure threshold for high blood pressure at any point after 20 weeks of pregnancy would not be of any use to medical professionals in identifying individuals who are at an elevated risk of maternal or neonatal problems[21].

Table 31 : Diagnostic criteria for preeclampsia (ACOG 2020) [22]

Systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg on at least 2 occasions at least 4 hours apart after 20 weeks of gestation in a previously normotensive patient AND the new onset of 1 or more of the following*:
Proteinuria ≥0.3 g in a 24-hour urine specimen or protein/creatinine ratio ≥0.3 (30 mg/mmol) in a random urine specimen or dipstick ≥2+ if a quantitative measurement is unavailable
Platelet count <100,000/microL
Serum creatinine >1.1 mg/dL (97.2 micromol/L) or doubling of the creatinine concentration in the absence of other kidney disease
Liver transaminases at least twice the upper limit of the normal concentrations for the local laboratory
Pulmonary edema
New-onset and persistent headache not accounted for by alternative diagnoses and not responding to usual doses of analgesics¶
Visual symptoms (eg, blurred vision, flashing lights or sparks, scotomata)

"**Preeclampsia** refers to the new onset of hypertension and proteinuria or the new onset of hypertension plus significant end-organ dysfunction with or without proteinuria in a previously normotensive patient, typically after 20 weeks of gestation or postpartum". There are multiple subtypes of preeclampsia that can be found, each characterised by a diverse range of pathophysiological pathways that can result in mortality and morbidity in both the mother and the foetus[23]. Early onset, which occurs before 34 weeks of gestation, and late onset, which occurs beyond 34 weeks of gestation, are the subtypes that are described the most frequently. Although there is some resemblance in the clinical symptoms, the range of disease and the

consequences are different: Early-onset disease has been linked to an increased severity of placental and maternal/fetal clinical findings, which in turn has been linked to a decrease in the outcomes for both the mother and the child[24,25]. Therefore, it has been hypothesised that the two phenotypes have different origins and pathophysiologies[24,26,27]. This is because of the reason stated above.

"Preeclampsia with severe features (formerly severe preeclampsia) is the subset of patients with preeclampsia who have severe hypertension and/or specific signs or symptoms of significant end-organ dysfunction that signify the severe end of the preeclampsia spectrum."

Table 32 : Preeclampsia with severe features

In a patient with preeclampsia, the presence of one or more of the following indicates a diagnosis of "preeclampsia with severe features"

ACOG 2020[22]

Severe blood pressure elevation
Systolic blood pressure ≥160 mmHg and/or diastolic blood pressure ≥110 mmHg on 2 occasions at least 4 hours apart while the patient is on bedrest; however, antihypertensive therapy generally should be initiated upon confirmation of severe hypertension, in which case criteria for severe blood pressure elevation can be satisfied without waiting until 4 hours have elapsed
Symptoms of central nervous system dysfunction:
New-onset cerebral or visual disturbance, such as: Photopsia, scotomata, cortical blindness, retinal vasospasm and/or Severe headache (ie, incapacitating, "the worst headache I've ever had") or headache that persists and progresses despite analgesic therapy with acetaminophen and not accounted for by alternative diagnoses
Hepatic abnormality:
Impaired liver function not accounted for by another diagnosis and characterized by serum transaminase concentration >2 times the upper limit of the normal range and/or Severe persistent right upper quadrant or epigastric pain unresponsive to medication and not accounted for by an alternative diagnosis
Thrombocytopenia:
Platelet count <100,000 platelets/microL

Kidney function impairment:
Serum creatinine >1.1 mg/dL [97.2 micromol/L] and/or Doubling of the serum creatinine concentration in the absence of other kidney disease
Pulmonary edema

"Preeclampsia superimposed upon chronic hypertension is diagnosed when preeclampsia occurs in a patient with preexisting chronic hypertension (hypertension that precedes pregnancy or is present on at least two occasions before the 20th week of gestation or persists longer than 12 weeks postpartum). It is characterized by worsening or resistant hypertension (especially acutely), the new onset of proteinuria or a sudden increase in proteinuria, and/or significant new end-organ dysfunction after 20 weeks of gestation or postpartum in a patient with chronic hypertension."[22]

"HELLP syndrome (Hemolysis, Elevated Liver enzymes, Low Platelets) appears to be a subtype of preeclampsia with severe features in which hemolysis, elevated liver enzymes, and thrombocytopenia are the predominant features". This condition may also be accompanied by hypertension, dysfunction of the central nervous system, and/or dysfunction of the kidneys. Both hypertension (82 to 88 percent, although in certain cases the increase in blood pressure may be mild initially) and/or hypertension (86 to 100 percent) are present in the majority of patients, although not all of them[28]. Before making a diagnosis of HELLP in these atypical patients, it is important to distinguish between other diseases that are linked with comparable laboratory abnormalities and rare persons who have neither of these conditions.

"Eclampsia refers to the occurrence of a tonic-clonic seizure in a patient with preeclampsia in the absence of other neurologic conditions that could account for the seizure".

"Gestational hypertension refers to hypertension without proteinuria or other signs/symptoms of preeclampsia-related end-organ dysfunction that develops after 20 weeks of gestation in a patient with a previously normal blood pressure". There is a possibility that approximately fifty percent of these patients will eventually exhibit signs and symptoms of preeclampsia.

INTERNATIONAL SOCIETY FOR THE STUDY OF HYPERTENSION IN PREGNANCY — The International Society for the Study of Hypertension in Pregnancy (ISSHP) classification system for hypertensive disorders of pregnancy is slightly different[29]:

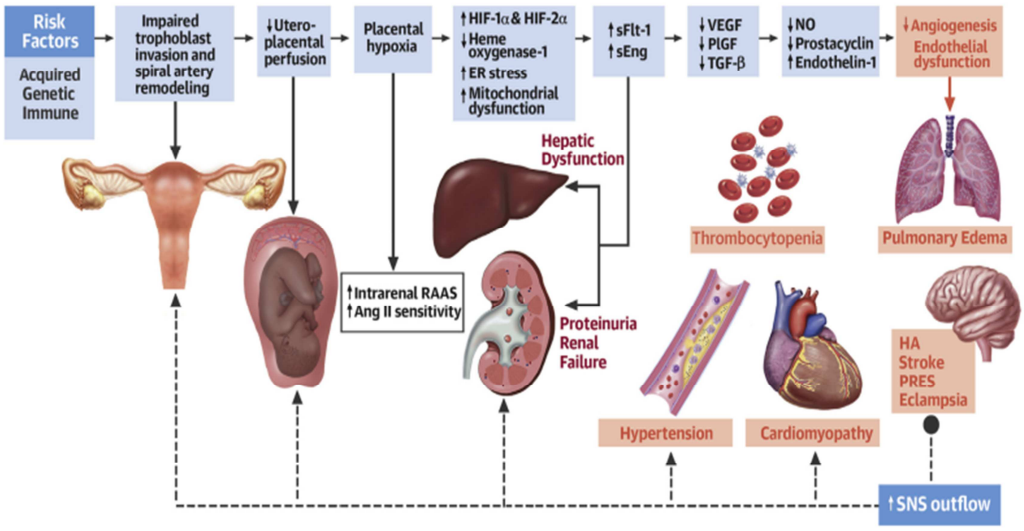
- **Prepregnancy or <20 weeks of gestation**
- **White-coat hypertension:** Systolic pressure ≥140 and/or diastolic pressure ≥90 mmHg when measured in the office or clinic, and blood pressure <135/85 mmHg using home or ambulatory 24-hour blood pressure monitoring readings.
- **Masked hypertension:** Blood pressure <140/90 mmHg at a clinic/office visit, but ≥135/85 mmHg at other times outside the clinic/office.
- **Chronic hypertension:** Hypertension detected prepregnancy or before 20 weeks of gestation. Chronic hypertension may be essential hypertension (ie, without a known secondary cause) or Secondary hypertension (ie, with a known secondary cause, eg, kidney disease).
- **Pregnancy ≥20 weeks of gestation**
- **Gestational hypertension:** Hypertension de novo ≥20 weeks of gestation without proteinuria or other features suggestive of preeclampsia

RISK FACTORS: [22]

Table 33: Clinical factors that have been associated with an increased risk of developing preeclampsia

Nulliparity
Preeclampsia in a previous pregnancy
Age >40 years or <18 years
Family history of preeclampsia
Chronic hypertension
Chronic kidney disease
Autoimmune disease (eg, antiphospholipid syndrome, systemic lupus erythematosus)
Vascular disease
Diabetes mellitus (pregestational and gestational)
Multifetal gestation
Obesity
Minority racial or ethnic group
Hydrops fetalis
Poorly controlled hyperthyroidism
Patient themself was small for gestational age
Fetal growth restriction, abruption, or fetal demise in a previous pregnancy
Prolonged interpregnancy interval if the previous pregnancy was normotensive; if the previous pregnancy was preeclamptic, a short interpregnancy interval increases the risk of recurrence
Male partner-related factors (new male partner, limited sperm exposure [eg, previous use of barrier contraception])
In vitro fertilization
Post traumatic stress disorder

Diagram 1 : PREECLAMPSIA: PATHOGENESIS



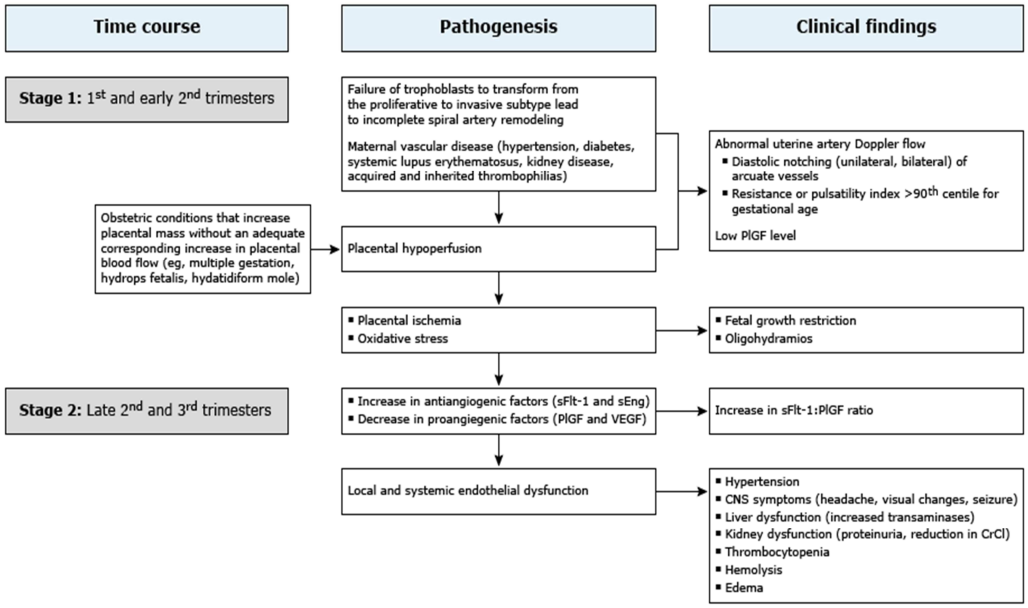


Diagram 1: Pathogenesis of preeclampsia[8]

ROLE OF ANGIOGENIC AND ANTIANGIOGENIC FACTORS

The developing placenta is responsible for the secretion of a number of proangiogenic factors, including vascular endothelial growth factor (VEGF) and placental growth factor (PlGF), amongst others. There are additional antiangiogenic factors that are secreted, such as soluble fms-like tyrosine kinase-1 (sFlt-1), which is an example of one of these factors. Establishing a healthy equilibrium between these elements is absolutely necessary in order to guarantee that its development will be typical. The disruption of this equilibrium is brought about by the increased synthesis of antiangiogenic markers in the placenta, which ultimately results in the systemic endothelial dysfunction that is characteristic of preeclampsia during pregnancy. It is likely that the absence of abnormal quantities of these substances in the foetuses of mothers who have preeclampsia is the reason why these foetuses do not exhibit the same clinical characteristics as their mothers, such as hypertension and proteinuria. Additionally, it is possible that these foetuses do not exhibit the same clinical characteristics as their mothers.[30]

PLGF AND ANGIOGENESIS

One of the most significant processes that occurs during embryonic development is known as angiogenesis. The regulation of this process is controlled by a complex interaction between a wide range of molecules, one of which is the VEGF family. The process by which new blood vessels are generated begins with a remodelling of the existing vasculature, which is then followed by the sprouting of new branches. After that, non-branching angiogenesis, which is marked by extension and expansion, occurs. Angiogenesis is a process that occurs typically in the endometrium during the monthly cycle, in the healing of wounds, and as an adaptive process in the heart and skeletal muscle of adult humans. It is also a process that occurs in the process of

wound healing. One of the reasons that placental growth factor is believed to be pro-angiogenic is that it has the capacity to enhance the activity of VEGF by means of competitive binding to the VEGFR-1 receptor. This is due to the fact that it makes it possible for VEGF to attach to VEGFR-2, which comes equipped with a more potent tyrosine kinase capability. However, PlGF also exerts its impact through other processes, such as the intermolecular transphosphorylation of VEGFR-2 after the activation of VEGFR-1, which amplifies the response of VEGFR-2 to the binding of VEGF. PlGF is a protein that has been shown to have a number of different functions. The formation of a heterodimer between PlGF and VEGF results in the formation of a heterodimer, which has the capacity to either stimulate or prevent the creation of new blood vessels. Pluripotent growth factor (PlGF) is responsible for the principal function of angiogenesis in tissues other than the placenta. This process takes place in response to pathological arechaemia or injury. The presence of hypoxia in cells that are not trophoblasts leads to an increase in the synthesis of plGF among those cells. On the other hand, the transcriptional activity of PlGF in trophoblast is decreased by hypoxia3 and promoted by a normoxic environment. This suggests that these cells have a specific regulatory mechanism and function. Hypoxia is the factor that inhibits PlGF transcription. [31]

PLGF IN REPRODUCTION

PlGF, on the other hand, has been shown to be produced by endometrial tissue following the secretory phase of the human menstrual cycle. This has been established by empirical research. [32] The presence of PlGF during this window of time lends support to the hypothesis that PlGF plays a role in influencing embryo implantation; however, specifics about this subject have not yet been identified. PlGF has been present throughout this window of time. The initial findings suggest that there are

some minor abnormalities in the development of the blood arteries in the brain among the children of women who are pre-eclamptic. It is presumed that these discrepancies are connected to the occurrence of events that take place inside the uterus[33]. In light of this, it is highly probable that PlGF has a considerable influence on the development of vascular structures and pregnancy, despite the fact that it might not be required for reproduction.

THE ROLE OF PLGF IN PLACENTAL DEVELOPMENT

It is through the placenta that much greater levels of circulating PlGF are known to occur during pregnancy. These levels are known to be significantly higher. In the placenta, there is a high possibility that the aim of PlGF is to facilitate the growth and maturation of the placental vascular system. This is a hypothesis that has been studied extensively. Furthermore, there is a decrease in the branching of the uteroplacental arteries, despite the fact that decidual invasion is not altered. It is also possible to see an unusual development in the lymphatic vessels that are found in the mouse uterus. The expression of PlGF in the human placenta is correlated with the different stages of placental development. This association can be broken down into two categories. To be more specific, the maturation of the utero-placental circulation and the non-branching angiogenesis of the feto-placental circulation occur at the same time as the rising production of PlGF in later stages of gestation. PlGF may play a more broad role in the development of human placentas, as this is a potential that has been explored. During the second trimester of pregnancy, while the utero-placental circulation is expanding, placental expression of PlGF begins to establish itself as the predominant form of expression. This occurs at the same time as the remodelling of myometrial spiral arteries, which takes place during a "second wave" of invasion that starts between 16 and 18 weeks of gestation. On the other hand, there have been

papers that debate whether or not PlGF has a role in trophoblast invasion. These publications are in direct opposition to one another. [34,35] In response to an increase in oxygen tension, trophoblasts develop invasive capabilities, and PlGF expression also increases with an increase in placental oxygenation. However, it is unknown if these two occurrences have a regulatory mechanism that is directly linked to each other due to the fact that there is no known connection between them. PlGF has a part in the differentiation of uterine natural killer cells, both of which have the ability to act as mediators for the invasion of trophoblasts into the decidua. PlGF also plays a role in the differentiation of uterine natural killer cells. The levels of PlGF are responsible for the acceleration of the proliferation of trophoblast cells. By starving trophoblast cells, it is also possible to minimise the amount of apoptosis that takes place in these cells. On the other hand, this does not take place when the cells are exposed to inflammatory cytokines. On the other hand, the precise function of PlGF-mediated reduction of apoptosis in placental development is not completely understood. It is possible that this will manifest itself as an increase in the quantity of circulating trophoblast debris that is found in cases of pre-eclampsia, which is typically characterised by a deficiency in PlGF. [36-38]

PLGF LEVELS IN NORMAL PREGNANCY

The concentrations of PlGF are low throughout the first trimester of a pregnancy that is not disturbed by any complications. On the other hand, they start to increase from week 11 to week 12, and they continue to increase until they reach their peak point at week 30, after which they start to decrease. This is in contrast to the fact that sFLT-1 levels have a tendency to increase as the pregnancy proceeds, which is contrary to the statement. The binding of PlGF to sFLT-1 results in a reduction in the bioavailability of PlGF, which in turn causes the typical divergence of angiogenic factors levels to

occur in a normal manner. The normal PlGF concentrations will range from a high of approximately 141 pg ml -1 at approximately 30 weeks of gestation to a low of 23 pg ml -1 at term this range is determined by the gestational age. These levels are roughly where the lower limit of normal, which is referred to as the fifth centile, falls within the range of values. [39]

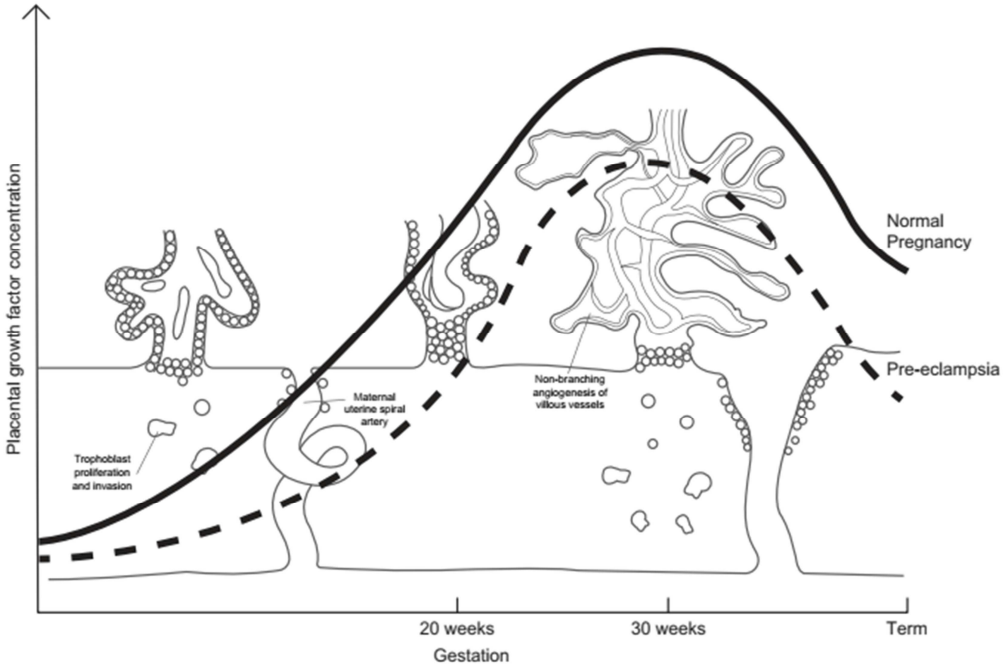


Diagram 2: Circulating PlGF

PLGF IN PRE-ECLAMPSIA

The levels of PlGF in the blood and the urine were shown to be lower in women who were diagnosed with pre-eclampsia and in women who were diagnosed with the condition a significant amount of time before the onset of the syndrome. A combination of decreased expression of PlGF and reduced free PlGF as a result of binding with sFLT-1, which is greater in women who are affected by the disease, is thought to be the cause of the shortfall in PlGF. Women who are affected by the condition are more likely to have this problem. [40] PlGF concentrations are lower in

women who ultimately develop preclampsia than they are in normal pregnant women throughout the early stages of pregnancy. This happens because preclampsia is a condition that can be fatal. On the other hand, there is no change in the levels of sFLT-1, which supports the hypothesis that PlGF expression in the placenta is reduced. Towards the conclusion of pregnancy, however, there is a reciprocal relationship that occurs between sFLT-1 and PlGF. This link occurs. This association is described by increasing levels of total sFLT-1 (both free and bound to VEGF or PlGF) and decreasing amounts of free PlGF inside the body. Together, these two factors serve to characterise the relationship. [41] Taking this into consideration, it is possible to draw the conclusion that the fundamental reason for the drop in PlGF concentrations that occurs during the second part of pregnancy is the sequestration of PlGF by sFLT-1. There is a high probability that low circulating PlGF is both a consequence of defective early events in placentation and a contributing factor to continuous aberrant growth throughout the latter part of pregnancy. Both of these outcomes are likely to occur simultaneously. PlGF is a marker of abnormal placentation, and the fact that women who do not have preeclampsia and who give birth to babies that are small for their gestational age also have low PlGF early in pregnancy gives support to the idea that PlGF reflects improper placentation. PlGF is an indication of abnormal placentation. [42] When it comes to the evidence addressing the expression of PlGF in placental tissue, however, there is a lack of consistency. The expression of PlGF is thought to be diminished as a result of the suppression that is brought about by prolonged placental hypoxia, which is brought about by an underdeveloped uteroplacental circulation. This idea is supported by the fact that there is a hypothesis that implies this. Some experiments, on the other hand, have indicated that the expression of PlGF in pre-eclamptic placental tissue either increased or remained stable. In spite of the fact that there is a lack of understanding about the

regulation of PlGF expression, several strategies and mechanisms have been examined. A number of processes, including as endoplasmic reticulum stress and epigenetic modifications, are responsible for influencing the functioning of the transcription factor hypoxia-inducible factor-1α (HIF1-α). Nevertheless, the extent to which HIF1-α plays a role in the formation of trophoblasts continues to be a contentious issue. [31,43-45]

PLGF FOR THE PREDICTION AND DIAGNOSIS OF PREECLAMPSIA

The identification of the disparities in the levels of circulating angiogenic factors that exist between pre-eclamptic and normal pregnancies, research has been carried out to investigate whether or not these factors have the potential to identify women who require close monitoring. The condition can only be alleviated by the delivery of the placenta once it has been determined that pre-eclampsia has arrived. This is the only way that the condition may be ameliorated. Women who are at risk for developing pre-eclampsia have a low PlGF level throughout the first trimester of pregnancy. This occurs well before the syndrome becomes clinically apparent. Pre-eclampsia is a disorder that can be quite dangerous. PlGF has a sensitivity of 32% and a false-positive rate of 5%, which indicates that individual angiogenic factors do not contribute to accurate prediction. In spite of the fact that there are disparities between the groupings, this is the position that has been taken. The development of multifactorial prediction tools is feasible through the combination of angiogenic factors, such as the ratio of sFLT-1 to PlGF, with characteristics of the patient's medical history or the results of an ultrasound. However, despite the fact that certain combinations have the potential to be valuable, they are not currently being utilised to a significant degree. As an illustration, the predictive algorithm that was developed by the Foetal Medicine Foundation at 11–13 weeks of gestation, which uses a

combination of maternal characteristics, mean arterial pressure, uterine artery pulsatility index, PAPP-A, and PlGF, manages to detect 95 percent of women with early pre-eclampsia and 46 percent of women with late pre-eclampsia, respectively, with a false-positive rate of ten percent.[46-48] It is possible that the application of prediction algorithms in particular subgroups, such as women who have antiphospholipid syndrome and systemic lupus erythematosus, will be more successful. This is because the baseline risk of adverse pregnancy outcome is higher in these patients, and the potential significance of angiogenic factors in the pathogenesis of disease is potentially greater. This is due to the fact that these patients have a greater risk of experiencing unfavourable outcomes during pregnancy.[49]

It is believed that the utility of PlGF and other angiogenic factors, such as VEGF and sFLT-1, in the prediction of pre-eclampsia is limited. This is because the pathology that causes the range of clinical presentation of pre-eclampsia will vary from patient to patient. Some of the women who are affected have mild symptoms that manifest themselves at term, while others have severe intrauterine growth restriction and early-onset abnormalities. There is a wide range of women who are impacted by this condition. Abnormal placentation appears to have a stronger link with early and severe disease, and abnormalities in angiogenic factors are more prevalent in people who have this disorder. This condition is characterised by abnormalities in the placenta. There is a group of women that exhibit an earlier and more severe presentation of the condition, and this subset is indicated by the presence of consistently low levels of PlGF during pregnancy, in conjunction with an abnormal ratio of sFLT-1 to PlGF. In order to conduct research that is more specific to the many subtypes of pre-eclampsia, the classification of pre-eclamptic patients may make use of angiogenic factors. This would allow for more targeted research that encompasses

the specific characteristics of each subtype. When it comes to women who are suspected of having pre-eclampsia but have not yet met the diagnostic criteria for the illness, the sFLT-1: PlGF ratio or plasma PlGF alone can be helpful as a 'rule out' test that has a good negative predictive value. In the process of diagnosing women with pre-eclampsia who needed to deliver within two weeks, maternal plasma PlGF that was lower than the fifth centile for gestation at the time of presentation performed better than a five-factor combination of commonly used clinical parameters (systolic and diastolic blood pressure, alanine transferase, uric acid, and dipstick proteinuria) (ROC area 0.87 versus 0.70, P=0.001). This was determined by comparing the results of the five medical parameters. It was discovered that low PlGF had the most sensitivity when it was applied to the delivery of a newborn that was tiny for gestational age. This finds additional evidence to support the notion that low PlGF is symptomatic of placental disease. It has been determined through analyses of the costs and benefits of these tests that angiogenic factor testing will be of assistance in determining the most effective way to allocate resources. The reason for this is because it will make it possible to observe women who are thought to have a lower chance of developing pre-eclampsia than other women. This will allow for a reduced frequency of observation. By utilising the sFLT-1: PlGF ratio, it is possible to identify between individuals who have preeclampsia and those who have illnesses that may present their symptoms in a similar manner, such as glomerulonephritis. In addition, this ratio is advantageous for classification purposes. [50]

FIRST TRIMESTER PREDICTION OF PE UTILIZING PLGF

The levels of PlGF in the serum might vary substantially depending on the characteristics of the mother and any comorbidities she may have. Women who are not pregnant, who smoke cigarettes, and who are of Afro-Caribbean, South Asian, or

East Asian heritage have greater levels of testosterone than women who are not pregnant. In contrast, levels are lower in women who are obese or who have diabetes mellitus that requires insulin. These women also have a lower risk of developing diabetes.[51-53] Consequently, in order to standardise the data and make it feasible to compare the findings of various research projects, it is necessary to make adjustments for these variables as well as other changes in the process of testing biomarkers and performing experimentation. This is necessary in order to make it possible to compare the findings of different research projects.[54] The PlGF values can be expressed as multiples of the expected median (MoM) in order to successfully achieve the objective of standardising the results through the utilisation of this method. Using Bayes' theorem, the application of a competing risks model to the prediction of preeclampsia in the first trimester requires the combination of the a priori risk from the maternal history with measurements of the uterine artery Doppler pulsatility index (UtPI), mean arterial blood pressure (MAP), and the PlGF MoM result in order to calculate a patient-specific risk for preeclampsia. This is done in order to determine the likelihood of the patient developing preeclampsia. One of the most important steps in the process is this one. [55,56] Before producing an adjusted estimate of the chance of delivery with PE, the previous risk that was estimated based on maternal characteristics and history is modified. This allows for the production of an adjusted estimate. An modified estimate of the chance of delivery with PE is obtained through this process in order to reach the desired result. It is based on the assumption that if pregnancy were to continue indefinitely, every woman would develop preeclampsia (PE), and the experience of this outcome prior to a particular gestation is dependent on the competition between delivery occurring before or after the onset of PE. This survival time strategy operates under the assumption that if pregnancy were to continue indefinitely, every woman would develop preeclampsia[55,56] There is a

change in the mean distribution of gestational age at birth with PE as a consequence of the integration of individual maternal characteristics and biomarker levels (MAP, UtPI, and PlGF). There is a movement to the right in the distribution of gestational age in pregnancies that are deemed to be low risk. This shift implies that it is likely that delivery will take place before a diagnosis of preeclampsia is made. Women who are at a high risk for preterm birth have a distribution of gestational age that is shifted to the left, which indicates that the birth is likely to occur after the commencement of preterm labour. Those two things are related to one another in some way. [55,56] Taking into account not only maternal risk factors but also protective characteristics, such as having a previous pregnancy that was normal, this makes it possible to evaluate an individual's risk in a manner that is tailored to their own circumstances.[55] Taking this method has a number of advantages, one of which is that it makes it possible to calculate patient-specific risks based on partial combinations, even in circumstances in which not all biomarkers are available. Numerous studies have shown that women who develop preterm preeclampsia have significantly lower levels of plenocyte growth factor (PlGF) in the first trimester of pregnancy compared to women who have normal pregnancies. This is the case in women who have preterm preeclampsia. In addition, there is a significant correlation between the concentrations of PlGF in the serum and the severity of PE, which is determined by the gestational age of the mother at the time of iatrogenic delivery as well as the percentile of the neonatal birthweight. [54] As an independent biomarker, PlGF has detection rates (DR) of 55% and 33% for the identification of early-onset and late-onset PE, respectively (with a fixed false-positive rate (FPR) of 10%). These rates are based on the assumption that FPR is fixed at 10%.[54] Despite this, there is currently a substantial body of evidence suggesting that the DR of preterm PE can be improved through the application of the competing risks model that was originally mentioned earlier. This particular model is

referred to as the Foetal Medicine Foundation (FMF) triple test, and it is carried out between the ninth and thirteenth week of gestation. [57] In the beginning, the model was constructed based on a study that involved 58,884 women. The study demonstrated that the model was accurate in detecting preterm PE (DR 77.3% at a fixed FPR of 10%).[56] In addition, this algorithm took into account the levels of pregnancy-associated plasma protein A (PAPP-A) in the serum, which are also known to be decreased during the first trimester of pregnancies that are later affected by PE. Additionally, maternal factors, MAP, UtPI, and PlGF were also incorporated into this algorithm.[57] Including PAPP-A resulted in a DR of 76.6%, which was comparable. In order to update the original algorithm and evaluate its effectiveness in the diagnosis of preterm and term preterm birth, prospective data from the first trimester screening of 35,948 women was utilised. The rates of detection for preterm and term preterm birth were 75% and 47%, respectively, with an FPR of 10%.[57] The introduction of PAPP-A in this combination model that also included PlGF did not result in an improvement in the screening performance, which is a conclusion that Tan et al. have also confirmed. [58] Moreover, a later systematic review and meta-analysis has showed that PlGF is superior to PAPP-A in terms of its ability to predict PE during the first trimester of pregnancy. [59] It is possible to use PAPP-A in the FMF model in the event that PlGF is not available; however, this approach leads to a decrease in the detection of preterm PE, with DRs being reported to be up to 7.1% (95% CI, 3.8–10.6%) lower when using PAPP-A as opposed to When using PlGF. [60,61]

Second and Third Trimester Prediction of PE

Screening for preeclampsia (PE) is performed throughout the second and third trimesters of pregnancy with the objective of calculating the patient-specific risk. This, in turn, dictates the frequency and substance of an individual's future antenatal

surveillance throughout the duration of their pregnancy. It is not primarily aimed at preventing PE because the window of opportunity for preventative intervention that is currently available is smaller than it was in the past. [62] A predictive value that is comparable to, if not superior to, screening for early and preterm preterm birth in the first trimester is provided by the combination of maternal risk factors, mean UtPI, mean papillomaglobulins, and plGF. This combination is responsible for providing this predictive value. According to studies, this combined screening that takes place between 19 and 24 weeks of gestation is able to accurately predict 99% of early preterm births, 85% of preterm preterm births, and 46% of term preterm births with a 90% false positive rate. In comparison to the DRs that are achieved only by maternal factors, which are 54 percent, 47 percent, and 37 percent, respectively, this is an improvement. [63] Even while screening during the third trimester, between 30 and 34 weeks, may predict 98% of preterm preterm births, it is still unable to detect more than half of the cases of term preterm births. [64] Because the addition of UtPI does not increase detection at this gestation, the best DR of term PE by combined testing is achieved between 35 and 37 weeks of gestation (70%) and includes sFlt-1 levels in addition to MAP, PlGF, and maternal variables. Because of this, the best DR of term PE is achieved at this gestation. [65]

USE OF PLGF-BASED TESTING TO PREDICT ADVERSE MATERNAL AND PERINATAL OUTCOMES

The "real-world" data from the PARROT trial exhibited significant decreases in serious unfavourable maternal outcomes with revealed PlGF testing. Additionally, there were apparent clinical benefits in the use of angiogenic biomarkers as diagnostic adjuncts to focus intensified antenatal surveillance to high-risk mothers. [66] Recent evidence suggests that individuals who experience potentially life-threatening

complications such as haemolysis, elevated liver enzymes and low platelets (HELLP) syndrome, disseminated intravascular coagulation, severe renal involvement, or eclampsia have the most significant alterations in serum sFlt-1 and PlGF levels. They also have the highest levels of PlGF. When compared to women who experience PE without severe features, similar consequences are not experienced by those women under the same circumstances. Women with HELLP syndrome appear to acquire the greatest derangement in angiogenic factor levels with a 74% reduction in PlGF levels and a ten-fold increase in the sFlt-1/PlGF ratio, followed by those who experience eclampsia with 57% lower PlGF levels and a four-fold increase in the sFlt-1/PlGF ratio. When compared to women who do not have severe features, those who have perinatal encephalopathy (PE) and at least two adverse outcomes, such as severe hypertension, left ventricular failure, stroke, and coma, in addition to those that have been detailed above, have significantly elevated levels of sFlt-11/PlGF (5.70-fold change; 95% confidence interval: 3.37–9.63, $p<0.001$). The fact that this is the case demonstrates that the degree of dysregulation of angiogenic markers is also associated with the amount of difficulties that arise when breastfeeding a child. [67]

It has also been discovered that abnormal levels of sFlt-1 and PlGF are associated with foetal abnormalities such as sudden death of pregnancy, premature birth, and death that happens within the uterus. These are all situations that can occur during pregnancy. [68-72] With a sensitivity of 93.2% and an NPV of 89.7% in women who come with suspected preeclampsia, low maternal PlGF concentrations appear to have an outstanding predictive performance for the diagnosis of SGA foetuses. This is as evidenced by the fact that they have a high sensitivity. When compared to ultrasound examination alone, which has a sensitivity of 71.2% and an NPV of 78.5 percent, this suggests that PlGF may have a role in the detection of supposed growth restriction.

This is in comparison to the results of the ultrasound assessment. [72] Furthermore, placenta-derived growth factor (PlGF) has been suggested as a possible discriminator between foetuses that are intrinsically small and those who are undergoing placentally mediated foetal growth restriction (FGR). The fact that a substantial independent correlation has been discovered between PlGF levels below the fifth percentile and histological placental markers of malperfusion lends credence to the notion that this is the case. [73] When it came to predicting FGR, low PlGF levels were superior to gestational age, belly circumference measurement, and umbilical artery resistance index in a study that was carried out across a number of different centres. The area under the curve (AUC) was 0.96, the net present value (NPV) was 99.2%, the sensitivity was 98.2%, and the specificity was 75.1% according to the findings of the study. When comparing individuals with PlGF levels below the fifth percentile to those with normal levels, there was a significant increase in the probability of intrauterine fatalities among those with PlGF levels below the fifth percentile ($p<0.05$). [71] Sherell and colleagues conducted a systematic review that investigated maternal PlGF levels as a predictor of adverse intrapartum and perinatal outcomes. The review further demonstrated that lower concentrations of PlGF are consistently associated with foetal compromise, which necessitates Caesarean delivery, admission to neonatal intensive care, and stillbirth. This was demonstrated by the fact that the review was conducted. [70]

In addition, abnormally high ratios of sFlt-1 to PlGF have been reported in pregnancies that have been made more difficult by early-onset FGR. These ratios appear to have a strong correlation with ultrasonography Doppler measurements, which either show that there is no end-diastolic flow or flow in the opposite direction in the umbilical arteries. [74] As the severity of FGR grew, Garcia-Manau and

colleagues found that there were statistically significant fluctuations in Elecsys® sFlt-1/PlGF levels. This was revealed in the course of their research. A significant degree of resistance in the umbilical artery Doppler was used to determine the severity of this condition ($p<0.05$).[74] Quantification of appropriate sFlt-1/PlGF thresholds was performed in order to ascertain the degree of severity associated with early-onset FGR. A value that was lower than 97.46 (area under the curve = 0.852, 95% confidence interval = 0.772, 0.932) was utilised in order to eliminate the possibility of the presence of absent or reversed end-diastolic flow. This was accomplished with a sensitivity of 78.3% and a specificity of 97.1%, respectively. The identification of reversed end-diastolic flow was accomplished with a ratio that was more than or equal to 523.78 (area under the curve = 0.751; 95% confidence interval = 0.578–0.924). This ratio had a sensitivity of 70.6% and a specificity of 87.2%. It was also found that there were significant associations between higher levels of sFlt-11/PlGF and negative outcomes, such as preterm birth ($p<0.001$), time-to-delivery ($p<0.001$), lower birthweight ($p=0.026$), and hospitalisation in the neonatal critical care unit ($p=0.006$). These findings were made possible by analysing the correlations between these variables. [74] There are no sFlt-1/PlGF cut-offs that have been recommended; nevertheless, connections between low blood levels of PlGF and elevated sFlt-1 levels have been discovered in a few studies that have investigated late-onset FGR. It has been discovered that these linkages might be interpreted in both positive and negative modes. The use of biomarker testing as an adjuvant to ultrasound for the aim of detecting foetal growth restriction (FGR) is not recommended outside of clinical research at the present time. This is because there is a limited availability of data from prospective interventional trials. As an alternative, larger studies that involve many centres are required in order to investigate the role these biomarkers play in monitoring foetal growth. [75,76] The link between higher sFlt-1/PlGF values and

unfavourable perinatal outcomes has been validated by additional research through their findings. The first of these studies was a retrospective, "real world" study that included 1117 pregnant women who were less than 36 weeks and 6 days along in their pregnancy. With an area under the curve (AUC) of 0.887, the research discovered that integrating sFlt-1/PlGF with maternal information assists in the prediction of undesirable outcomes such as preterm delivery, respiratory distress syndrome, and foetal death. This was found to be useful in the prediction of these events. [77] Significantly, it has been demonstrated that the sFlt-1/PlGF ratio is capable of accurately predicting a composite of other adverse perinatal outcomes. These outcomes include intraventricular haemorrhage, hypoxic-ischemic encephalopathy, necrotising enterocolitis, and retinopathy of prematurity (area under the curve = 0.87, 95% confidence interval = 0.81–0.93). A big discovery has been made here. This finding is significant because it may be more clinically meaningful than the prediction of time-to-delivery, which may be influenced by a different threshold to deliver with increasing gestational age. This is a finding that deserves attention since it is important. It is noteworthy that decreased levels of PlGF and an increased ratio of sFlt-1 to PlGF are associated with unfavourable outcomes throughout the perinatal period. [78]

LITERATURE FROM PREVIOUS STUDIES:

Côté et al. (2025) conducted a prospective study with the objective of determining whether or not there is a correlation between the levels of placental growth factor (PlGF) and pregnancy-associated plasma protein A (PAPP-A) in the maternal serum during the first trimester and the likelihood of placenta-mediated problems. Specifically, the intention of this study was to determine whether or not there is a correlation between these two factors. During the course of this study, which was a

secondary analysis of the PREDICTION trial, a total of 7,262 women who had never given birth were recruited. The gestational age range for these women was between 11 and 14 weeks. After taking into account maternal variables and the advancement of gestational age, the levels of PlGF and PAPP-A in the serum were expressed as multiples of the median (MoM). Additionally, these levels were corrected for progress. In order to categorise the participants into distinct groups, it was determined whether or not they possessed low PlGF (less than 0.4 MoM) and/or low PAPP-A (less than 0.4 MoM). A composite unfavourable pregnancy outcome analysis was performed on deliveries that took place before 34 weeks, before 37 weeks, and at or after 37 weeks of pregnancy. Pre-eclampsia, foetal growth restriction (FGR), foetal death, and placental abruption were all outcomes that were associated with this result. A combination of low PAPP-A and low PlGF was related with the highest probability of poor outcomes, with 21% before 37 weeks and 12% before 34 weeks. This was in comparison to isolated low PlGF (7% and 3%), isolated low PAPP-A (2% and 1%), and neither biomarker aberrant (1% and 0.4%). The results demonstrated that a combination of these two biomarkers was statistically significant. When the probability of bad effects against isolated low PlGF and low PlGF was compared, this was the situation that was seen. When compared to low PlGF alone (6%), low PAPP-A alone (0.5%), or neither marker abnormal (0.7%), the combination of low PlGF and low PAPP-A was also significantly related with preterm pre-eclampsia (12%). This was the case when comparing the results of the three different combinations. On the other hand, the low PlGF alone (12%) and the low PAPP-A alone (0.5%) were both significantly lower than this. A low PAPP-A score on its own should not be regarded a significant risk factor for adverse pregnancy outcomes, as indicated by the findings of the study. However, a low PlGF and its combination with a low PAPP-A have a substantial link with premature pre-eclampsia and other placental issues. This is the

case even when these two factors are considered together. Furthermore, the findings underscore the necessity of conducting biomarker screenings at an early stage in order to provide information that may be used to drive risk assessment and the implementation of preventative measures.[79]

A retrospective study was carried out by Palmrich et al. (2024) with the purpose of determining the prognostic value of angiogenic markers, specifically soluble fms-like tyrosine kinase-1 (sFlt-1) and placental growth factor (PlGF), as well as their ratio, in the context of predicting pre-eclampsia (PE) in pregnancies that were complicated by foetal growth restriction (FGR). This analysis comprised 93 singleton pregnancies with FGR. All of these pregnancies met the criteria for inclusion. Because there was a chance of PE, the levels of sFlt-1 and PlGF that were found in the maternal serum were investigated. During the time that we were collecting the sample, we did not include any women who had previously been diagnosed with PE before. The findings of the study indicate that forty-nine percent of the women eventually developed pulmonary embolism (PE). Among the patients, fourteen (15.1%) were diagnosed within one week, twenty-one (22.6%) patients were identified within two weeks, and forty-nine percent of the patients were diagnosed at any time after sampling. With an area under the curve (AUC) of 0.87, the sFlt-1/PlGF ratio was a more accurate predictor of PE than either sFlt-1 or PlGF alone when it came to predicting PE within one week. This was the case when it came to whether or not PE would occur. Using a cutoff value of sFlt-1/PlGF <38, which had a negative predictive value (NPV) of 0.933 and a sensitivity of 0.952, it was possible to entirely eliminate the chance of pulmonary embolism (PE) within a duration of two weeks. In contrast to the cutoffs that had been created in the past for ruling in PE, which were less successful, this was a significant improvement. Using the sFlt-1/PlGF ratio is superior to using PlGF alone

when it comes to forecasting the risk of PE in FGR pregnancies, according to the findings of the study, which came to the conclusion for this particular purpose. In addition, the research discovered that established thresholds have the potential to direct clinical decision-making by enhancing early diagnosis and risk classification.[80]

The purpose of the prospective longitudinal noninterventional study that Verma et al. (2022) conducted was to determine whether or not there is a correlation between the levels of placental growth factor (PlGF) in the first trimester of pregnancy and the occurrence of poor outcomes for both the mother and the prenatal period in Indian women. The study was carried out with the intention of determining whether or not there is a connection between the two. One hundred eighty singleton pregnancies that were between eleven and thirteen and six weeks of gestation were included in the study. The enzyme-linked immunosorbent assay (ELISA) was utilised to determine the levels of PlGF in the serum, and patients were tracked throughout the entirety of their pregnancies. The presence of pre-eclampsia (PE) was discovered in 9.3% of the instances (15/161), whilst foetal growth restriction (FGR) was discovered in 19.8% of the cases (32/161). Women who had previously suffered PE were shown to have a significantly higher risk of experiencing a recurrence of the condition ($p < 0.04$) after experiencing PE. In the cases of pregnancies that were impacted by foetal growth restriction (FGR) and peritoneal effusion (PE), the levels of plGF were found to be considerably lower in comparison to pregnancies that were considered to be normal ($p < 0.04$). Furthermore, the Doppler pulsatility index (PI) of the uterine artery during the first trimester was shown to be considerably higher in cases with peritoneal effusion (PE) ($p < 0.0001$). Seven point five percent of the pregnancies that were affected by the condition ended in stillbirths, and ten percent of those pregnancies required admission to the neonatal intensive care unit (NICU). According to the

findings of the study, the measurement of PlGF during the first trimester of pregnancy has the potential to be an excellent screening tool for detecting pregnancies that are at risk for PE and FGR. This would allow for early surveillance and intervention, which would be beneficial for all parties involved. [81]

For the purpose of assessing the maternal and perinatal outcomes associated with extremely high values (>655) of the sFlt-1 (soluble fms-like tyrosine kinase 1)/PlGF (placental growth factor) ratio in pregnancies that were complicated by suspected or confirmed placental dysfunction, Villalaín et al. (2020) took the initiative to conduct a study. A multicenter retrospective cohort was used for the research that was carried out. Two hundred and thirty-seven singleton pregnancies that were between twenty-five and thirty-seven weeks of gestation were included in the study. Despite the fact that the clinicians were aware of the results of the biomarker, they continued to follow the procedures that had been established for delivery indications. It was shown that a ratio of sFlt-1 to PlGF that was greater than 655 had a substantial link with pre-eclampsia (PE) or foetal growth restriction (FGR). Furthermore, it was discovered that 38 percent of women who did not have preeclampsia at the beginning of their pregnancy had PE at a later time. In preeclamptic pregnancies, the median time to delivery was found to be four days, but in non-preeclamptic pregnancies, the median time to birth was reported to be seven days. The perinatal mortality rate was 62.1% during the first 24 weeks of pregnancy, and the severe neonatal morbidity rate was higher than 50% before 29 weeks, although it significantly dropped after 34 weeks. Both of these rates were higher during the first 24 weeks of pregnancy. At every stage of pregnancy, there was a significant prevalence of maternal complications, such as severe hypertension, HELLP syndrome, and abruptio placentae difficulties. These complications were related with a high incidence. The findings of the study indicate

that a rapid course of the disease is indicated by levels of sFlt-11/PlGF that are abnormally high. As a consequence of this, early monitoring and proper care are essential in order to achieve the best possible outcomes for both women who are breastfeeding and neonates. [82]

Placental growth factor (PlGF) was used in the screening process for preeclampsia (PE) in women who did not display any symptoms. The objective of the meta-analysis that was carried out by Agrawal et al. (2019) was to evaluate the predictive accuracy of PlGF in this process. The research was carried out with the intention of assessing various PlGF cutoffs, its effectiveness across a variety of gestational ages, 14 weeks to term, and its accuracy in predicting early-onset (EO) and late-onset (LO) preeclampsia in populations that contained both low-risk and high-risk individuals. A total of forty papers were included in the investigation, wherein 3,189 instances of preeclampsia and 89,498 controls were analysed. The findings showed that PlGF levels were much lower in women who later developed PE, with predictive values varying across several thresholds. This was indicated by the way that the findings were presented. Regardless of the specific criterion, this was always the case. With a predictive odds ratio (POR) of 25 (7–88), a sensitivity of 0.78 (95% confidence range, 0.67–0.86), and a specificity of 0.88 (95% confidence interval, 0.75–0.95), it was shown that PlGF levels between 80 and 120 pg/mL had the highest predictive accuracy. This was the condition that was found to have the highest predictive accuracy. The results of the PlGF test were also more accurate when they were performed after 14 weeks of gestation (odds ratio: 10 [7–15]), and they were also more accurate when they were used to predict preeclampsia with an early onset (odds ratio: 18 [9–37]). Despite the fact that its clinical utility should be carefully evaluated, the study came to the conclusion that PlGF is a valuable screening tool for

preeclampsia, particularly for early-onset disease. However, additional randomised controlled trials are required to determine whether or not its implementation improves maternal and neonatal outcomes. [83]

Duhig et al. (2019) conducted a multicenter, pragmatic, stepped-wedge cluster-randomized controlled study with the objective of evaluating the clinical usefulness of placental growth factor (PlGF) testing in terms of improving the diagnosis and management of pre-eclampsia (PE) that is suspected. The study was designed to be pragmatic and stepped-wedge cluster of randomised controlled trials. A total of 1,023 pregnant women from 11 different maternity hospitals in the United Kingdom took part in the trial with a gestational age between 20 weeks zero days to 36 weeks 6 days. The purpose of the study was to compare the outcomes of disclosed PlGF testing in conjunction with a clinical treatment algorithm to those of concealed PlGF testing in conjunction with normal care. The primary outcome was the amount of time that passed between the beginning of the patient's presentation and the confirmation of a PE diagnosis. It was discovered during the course of the research that PlGF testing significantly reduced the median time to diagnosis from 4.1 days to 1.9 days (time ratio 0.36, 95% confidence interval 0.15–0.87; p=0.027). This results in a significant reduction in waiting time. The therapeutic intervention that was supposed to take place sooner was ultimately made possible as a result of this. In addition, the rates of serious unfavourable outcomes for mothers were lower in the PlGF testing group (4%), in comparison to the control group (5%; adjusted odds ratio: 0.32, 95% confidence interval: 0.11–0.96; p=0.043). Although there were some differences between the two groups, the perinatal unfavourable outcomes and the gestational age at birth were comparable. Using PlGF testing is connected with a reduction in severe maternal difficulties and speeds up the process of identifying preeclampsia (PE),

according to the findings of the study. This is the conclusion that can be drawn from the researchers' findings. The inclusion of this testing into clinical practice for high-risk pregnancies is given more credence by this study. [66]

Sung et al. (2017) conducted a prospective observational study with the objective of determining whether or not there is a correlation between maternal serum levels of placental growth factor (PlGF) and pregnancy-associated plasma protein A (PAPP-A) during the first trimester of pregnancy and pregnancies that were complicated by pre-eclampsia (PE) or small-for-gestational-age (SGA) infants throughout the course of the pregnancy. In total, there were 175 pregnant women who took part in the experiment, and 155 of them were able to complete the follow-up. The levels of PlGF and PAPP-A in the serum were measured between 11 and 13 weeks of gestation. These levels were expressed as multiples of the median (MoM), and they were connected with the outcomes of the pregnancy after the measurements were taken following the completion of the measures. The use of logistic regression analysis was employed for the goal of establishing the overall prediction accuracy of the models. According to the findings, lower levels of PlGF and PAPP-A were found to be significant predictors of SGA in infants (PlGF: odds ratio 0.143, 95% confidence interval 0.025–0.806; PAPP-A: odds ratio 0.191, 95% confidence interval 0.051–0.718). Lower levels of PlGF were found to be associated with a lower risk of SGA in newborns. PlGF and PAPP-A were not significant predictors of PE like maternal age and body mass index (BMI) were. The only exceptions to this were maternal age and BMI. Maternal age and body mass index were the only characteristics that were found to have a significant relationship with PE. During the second trimester of pregnancy, the study found that there was a substantial association between PAPP-A and the uterine artery systolic/diastolic ratio. Additionally, the study found that there was a

positive correlation between PlGF and PAPP-A ($r = 0.467$, $P < 0.001$). The appropriate cutoff parameters for predicting SGA babies were showed to be 0.885 for PlGF MoM and 1.06 for PAPP-A MoM. This was demonstrated by the findings of the study. The results of the study showed that PAPP-A was a better predictor of SGA than PlGF. At the end of the study, the researchers arrived to the realisation that first-trimester PlGF and PAPP-A are useful markers for diagnosing SGA babies; however, they have very limited prognostic value for PE disorders. There is a need for future study with larger sample sizes in order to evaluate their potential involvement in the assessment of placentation and hypertensive problems that occur during pregnancy. [84]

Benton et al. (2016) conducted a prospective multicenter study with the objective of determining the role of placental growth factor (PlGF) as a marker for foetal growth restriction (FGR) caused by placental dysfunction and distinguishing it from foetuses that were fundamentally undersized. This was done in order to distinguish between the two types of foetuses. In this particular research project, participants included women from Canada, New Zealand, and the United Kingdom who were suspected of having foetal growth restriction (FGR), which is defined as foetal abdominal circumference that is lower than the tenth percentile for gestational age respectively. After birth, measurements of maternal plasma PlGF were taken, and placental histology was analysed. The outcomes of the study indicated that a low PlGF, which was defined as being less than the fifth percentile for gestational age, was capable of reliably predicting placental FGR to a high degree. The performance of this prediction was superior to that of traditional foetal ultrasonography readings, as evidenced by the fact that it had an area under the receiver-operating characteristic curve (AUC) of 0.96, a sensitivity of 98.2%, and a specificity of 75.1%. As an additional point of interest, it was observed that a PlGF level that was much lower than 12 pg/mL was

associated with significantly shorter sampling-to-delivery intervals (13 days as opposed to 29.5 days, $p < 0.0001$). This suggests that it has the capability to predict the need for early delivery. There was a correlation between low levels of PlGF and severe placental pathology in each and every stillbirth that took place within the cohort. According to the findings of the study, PlGF is a significant biomarker that has the potential to be utilised in the identification of placental dysfunction in FGR pregnancies. This biomarker has the ability to differentiate between high-risk instances that require close monitoring and early intervention and fundamentally small but healthy foetuses. [71]

The objective of the prospective multicenter study that Chappell et al. (2013) conducted was to investigate the diagnostic accuracy of placental growth factor (PlGF) levels in predicting preeclampsia (PE) in women who were suspected of having hypertensive disorders of pregnancy. Specifically, the researchers wanted to determine whether or not PlGF levels were significant in predicting PE. A total of six hundred and twenty-five pregnant women who presented with clinical suspicion of preeclampsia between the ages of twenty and thirty-five weeks of gestation were included in the study. For the purpose of determining the levels of PlGF in maternal serum, the Alere Triage test was employed. In accordance with the primary objective, the delivery of demonstrated PE had been accomplished within a span of fourteen days. The outcomes of the study indicated that PlGF levels that were lower than the fifth percentile for gestational age had a superior sensitivity of 96% and a negative predictive value of 98% when it came to predicting delivery due to PE within 14 days. This was indicated by the fact that the levels were able to predict birth due to PE. On the other hand, the specificity of these levels was insufficient to reach 55%. It was shown that the area under the receiver operating characteristic (ROC) curve produced

a value of 0.87, which was higher than the performance of standard diagnostic tests such blood pressure, proteinuria, uric acid, and liver enzymes (ROC range: 0.58–0.76, $p<0.001$). A low PlGF was found to be an incredibly effective rule-out test for preeclampsia, according to the findings of the study, which proved to be the basis for the conclusion. This makes it possible to better risk classification and decreases the requirement for therapies that are neither necessary nor necessary in women who are at a lower risk. [50]

The research that was conducted by Sibiude et al. (2012) aimed to investigate the predictive value of circulating placental growth factor (PlGF) levels in the diagnosis of preeclampsia (PE) and unfavourable pregnancy outcomes in women who had a suspicion of having PE or intrauterine growth restriction (IUGR). Specifically, the researchers wanted to determine whether or not PlGF levels were useful in predicting what would happen during pregnancy. The research was carried out in a prospective fashion while conducting it in a double-blind fashion. In order to determine the levels of plasma PlGF, the researchers utilised the Triage test on 96 pregnant women who had been admitted to the hospital after 22 weeks of gestation. For the purposes of this investigation, adverse outcomes were defined as severe preeclampsia, newborns that were undersized for their gestational age (less than the tenth centile), or elective delivery due to complications in either the mother or the foetus. Severe unfavourable outcomes, on the other hand, included HELLP syndrome, eclampsia, extremely small for gestational age (less than the third centile), or preterm delivery (less than 34 weeks). A comparison was made between the levels of PlGF in women who had PE and those who had negative outcomes. The findings revealed that the former group had significantly lower levels (2.9 compared 3.7, $p=0.02$), while the latter group had even lower levels (2.9 versus 4.3, $p<0.001$). A 13-fold greater risk of poor outcomes

was related with the lowest tertile of PlGF (odds ratio 13, 95% confidence interval [3–50]), and a 216-fold increased risk of severe adverse outcomes before 34 weeks (odds ratio 216, 95% confidence interval [18–2571]) was associated with its presence. After 15 days, only five percent of women whose PlGF levels were higher than the fifth centile suffered a major unfavourable outcome. On the other hand, ninety-six percent of women whose PlGF levels were lower than 12 pg/mL experienced a severe adverse event. This was the situation with those individuals who enrolled before to the 34th week. PlGF was found to be a powerful indication of adverse pregnancy outcomes, which can be helpful in risk stratification and the management of both PE and IUGR. This conclusion was reached based on the findings of the study, which were presented in the previous sentence. [19]

MATERIALS AND METHODS

STUDY AREA: The present study was carried out in the Department of Obstetrics & Gynaecology, KAHER's Dr. Prabhakar Kore Hospital, Belagavi

STUDY POPULATION: Antenatal patients admitted to the labor room of KAHER's Dr. Prabhakar Kore Hospital, Belagavi

STUDY DESIGN: Cross sectional study

SAMPLE SIZE:

p=51.7%

q=100-51.7=48.3

Sample size at 95% confidence interval

20% tolerable error and 5% attrition

n=$Z^2_{1-\frac{\alpha}{2}}.pq$ x 1.05

$(20\%\ of\ p)^2$

n=94

TIME FRAME TO ADDRESS THE STUDY: One year

INCLUSION CRITERIA:

- Singleton pregnancy
- Gestational age > 20 weeks
- First or Second trimester ultrasound for dating
- Preeclampsia.
- Eclampsia.

EXCLUSION CRITERIA

- Multifetal Pregnancy.
- Gestational hypertension.
- Known case of epilepsy.
- Women not willing to provide consent.

METHODOLOGY:

Women with gestational age >20 weeks and identified as having pre-eclampsia or eclampsia admitted to the labor room for delivery were included in the study.

Blood samples (3ml) were drawn in a plain vacutainer and sera extracted by centrifugation and PlGF test was done on serum samples using brand Revvitys: DELFIA-R with a principle of time -resolved fluorescence. Maternal and new born complication were recorded post deliver or till discharge.

Diagnostic criteria-

Pre-eclampsia= Pre-eclampsia (de novo) is gestational hypertension accompanied by one or more of the following new- onset conditions at ≥20 weeks' gestation:

1. Proteinuria

2. Other maternal end-organ dysfunction, including:

• Neurological complications (e.g., eclampsia, altered mental status, blindness, stroke, clonus, severe headaches, or persistent visual scotomata)

• Pulmonary oedema

• Haematological complications

(e.g., platelet count < 150,000/μL, DIC, haemolysis)

• AKI (such as creatinine ≥ 90 μmol/L or 1 mg/dL)

• Liver involvement (e.g., elevated transaminases such as ALT or AST > 40 IU/L) with or without right upper quadrant or epigastric abdominal pain)

3. Uteroplacental dysfunction (e.g., placental abruption, angiogenic imbalance, fetal growth restriction, abnormal umbilical artery Doppler waveform analysis, or intrauterine fetal death)

FGR-

Defined as estimated fetal weight (EFW) or abdominal circumference (AC) $<10^{th}$ centile for gestational age

Outcome measures PlGF based testing to identify maternal and newborn adverse outcomes early

Maternal adverse outcomes

- Eclampsia
- HELLP
- PPH
- Renal failure
- DIC
- Abruption
- Sepsis
- Cerebrovascular accidents

Fetal adverse outcomes

- Still birth
- IUFD

- Perinatal death
- Birth asphyxia
- Early neonatal death
- LBW(<2.5 kg)

EARLY NEONATAL DEATH

STATISTICAL ANALYSIS:

Data entry was done using M.S. Excel and statistically analysed using Statistical package for social sciences (SPSS Version 16) for M.S Windows.

Descriptive statistical analysis was carried out to explore the distribution of several categorical and quantitative variables. Categorical variables were summarized with n (%), while quantitative variables were summarized by mean ± S.D. All results were presented in tabular form and are also shown graphically using bar diagram or pie diagram as appropriate. The difference in the two groups was tested for Statistical Significance using Parametric tests such as t-test and categorical variables tested by chi square test. P-value <0.05 was considered statistically significant after assuming all the rules of statistical tests.

ETHICAL ISSUES

1. The objectives and procedure of the study was explained to all patients.
2. Informed consent was taken from all patients willing to participate in the study.
3. The option to opt out of the study was kept open without any clause.
4. Complete confidentiality regarding patient information was maintained through all stages of the study.

RESULTS

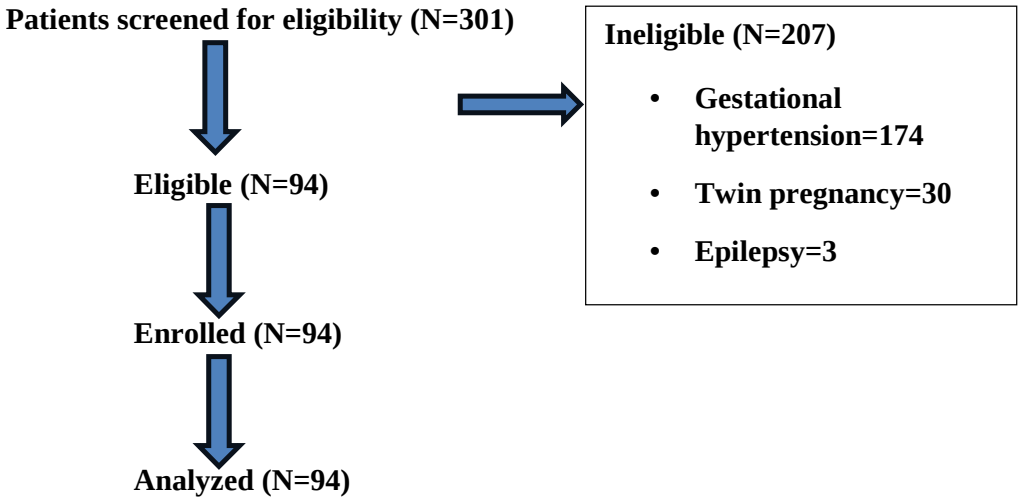
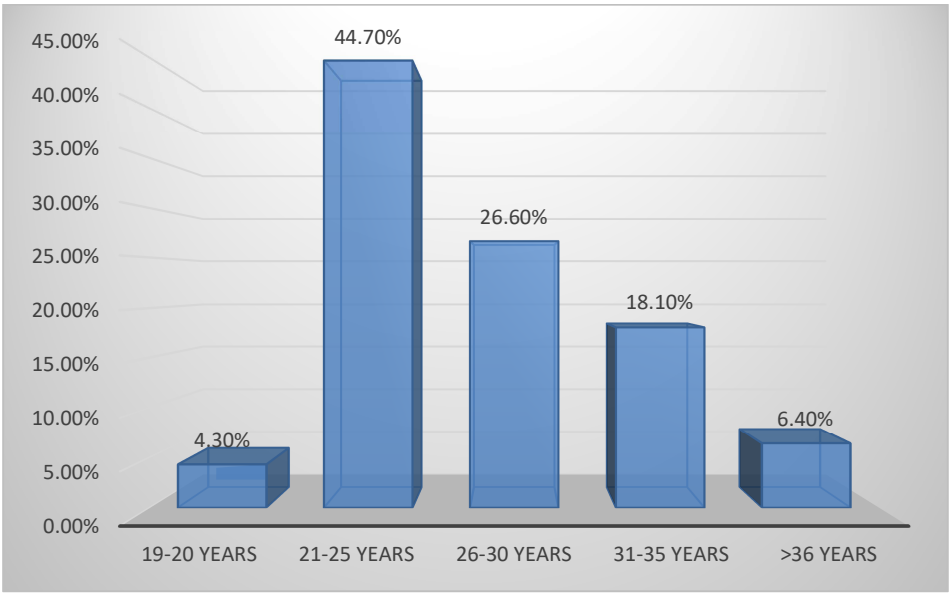

Patients screened for eligibility (N=301)
Ineligible (N=207)
• Gestational hypertension=174
• Twin pregnancy=30
• Epilepsy=3
Eligible (N=94)
Enrolled (N=94)
Analyzed (N=94)

Table 1: Distribution of Maternal Age Group

		n=94	Percent
Age Group	19-20 years	4	4.3%
	21-25 years	42	44.7%
	26-30 years	25	26.6%
	31-35 years	17	18.1%
	>36 years	6	6.4%
	Total	94	100.0%

The largest proportion of participants (44.7%) belonged to the 21–25 years age group, making up 42 of the 94 participants. The 31–35 years age group included 17 participants (18.1%). The least represented age groups were 19–20 years (4.3%, n = 4) and above 36 years (6.4%, n = 6).

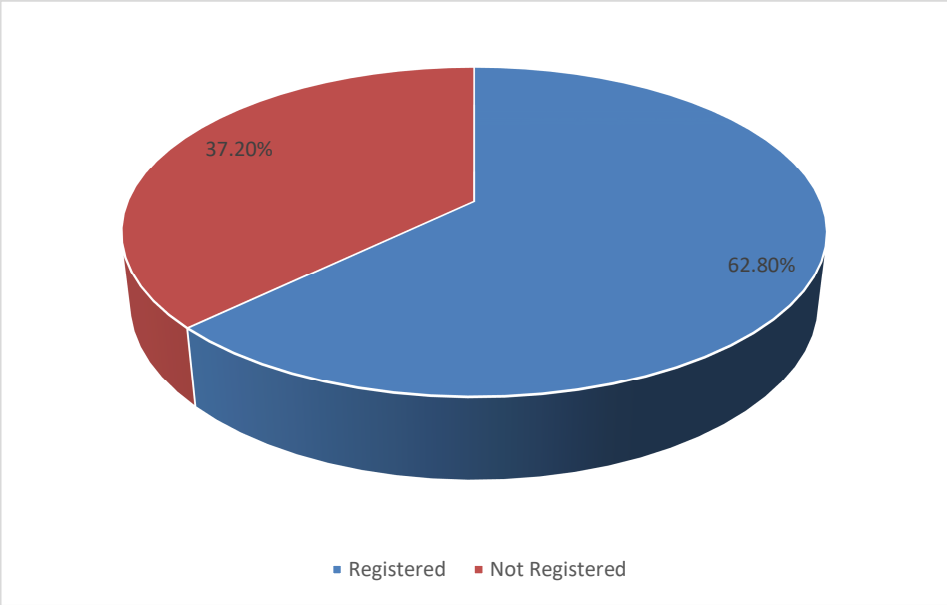


Graph 1: Distribution of Maternal Age Group

Table 2: Registration Status of Participants

		n=94	Percent
Registration status	Registered	59	62.8%
	Not Registered	35	37.2%
	Total	94	100.0%

A total of 94 women were included in the study, of whom 59 participants (62.8%) were registered, while 35 participants (37.2%) were not registered.

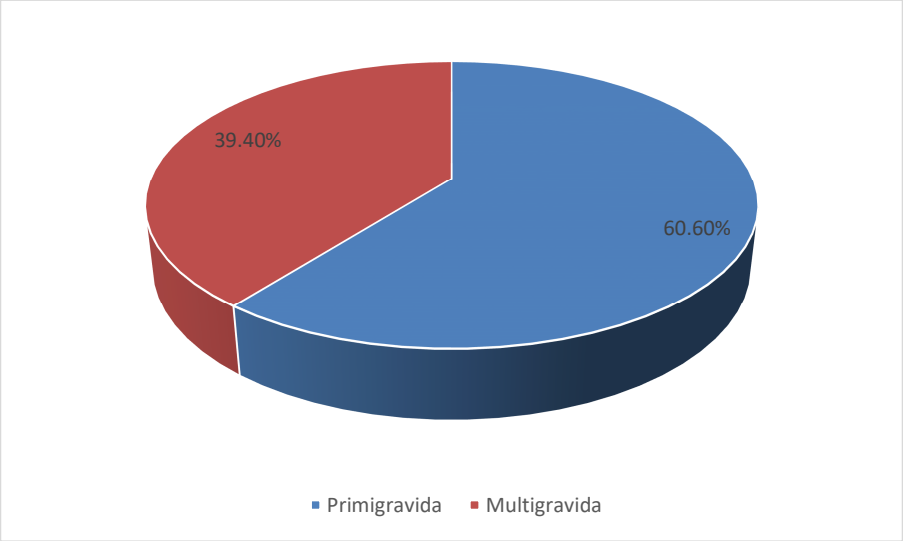


Graph 2: Registration Status of Participants

Table 3: Distribution of Gravida Status

Gravida	n=94	Percent
Primigravida	57	60.6%
Multigravida	37	39.4%
Total	94	100.0%

57 participants (60.6%) were primigravida, while 37 participants (39.4%) were multigravida.

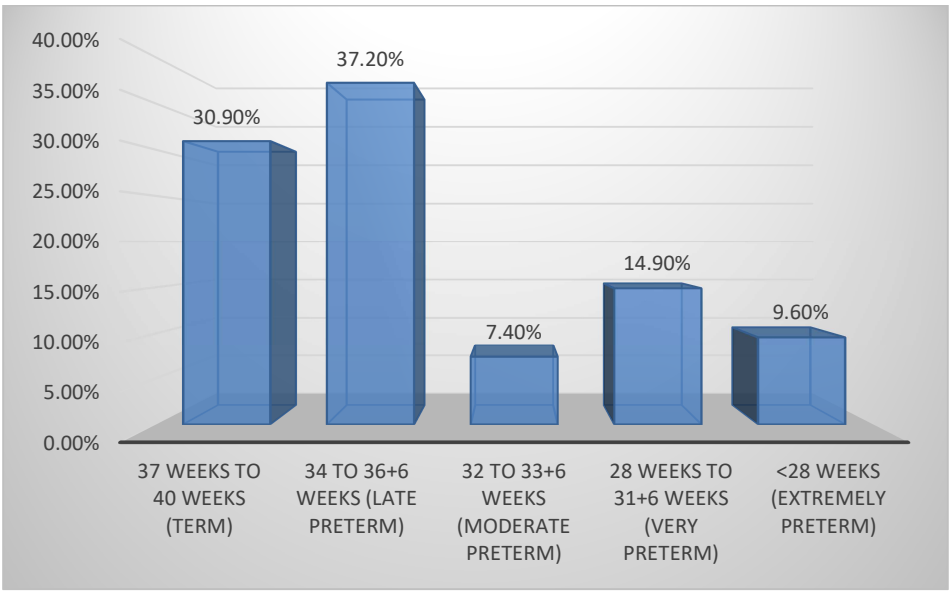


Graph 3: Distribution of Gravida Status

Table 4: Distribution of Gestational Age at Delivery

		n=94	Percent
Gestational Age	37 weeks to 40 weeks (Term)	29	30.9%
	34 to 36+6 weeks (Late Preterm)	35	37.2%
	32 to 33+6 weeks (Moderate Preterm)	7	7.4%
	28 weeks to 31+6 weeks (Very Preterm)	14	14.9%
	<28 weeks (Extremely Preterm)	9	9.6%
	Total	94	100.0%

The largest proportion of deliveries occurred in the late preterm group (34 to 36+6 weeks), accounting for 35 cases (37.2%).Delivery at term (37 to 40 weeks) was reported in 29 cases (30.9%), Deliveries in the moderate preterm category (32 to 33+6 weeks) were less frequent, with only 7 cases (7.4%).



Graph 4: Distribution of Gestational Age at Delivery

Table 5: Maternal Height, Weight, and BMI

	Mean	Std. Deviation
Height	152.67	6.55
Weight	64.29	14.11
BMI	29.27	10.76

The mean height of the participants was 152.67 cm with a standard deviation of 6.55 cm.

The mean weight of the participants was 64.29 kg with a standard deviation of 14.11 kg.

The mean BMI of the participants was 29.27 with a standard deviation of 10.76.

Table 6: Maternal Symptoms at presentation

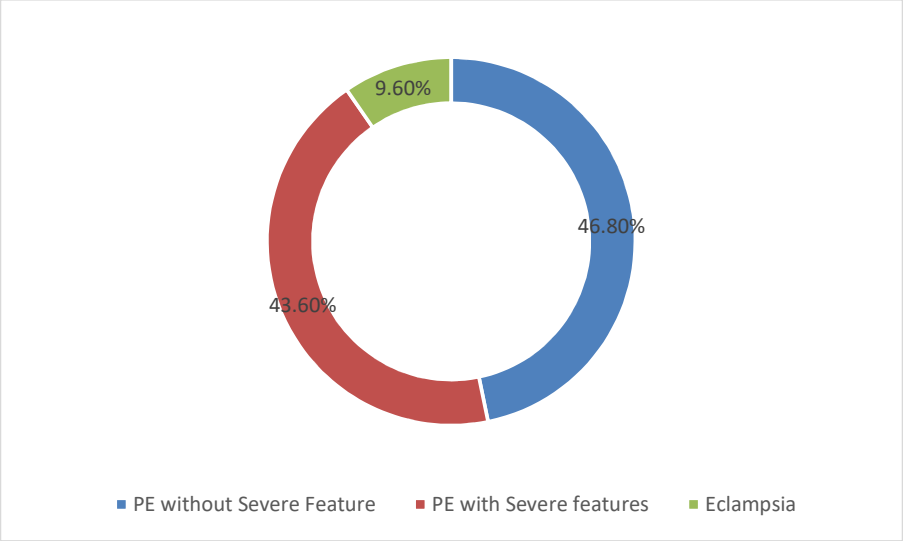
Maternal Symptoms at presentation	n=94	Percent
Headache	17	18.1%
Blurring of vision	12	12.7%
Epigastric pain	6	6.4%
Seizure	9	9.6%
Oliguria	0	0%
No Maternal Symptoms	50	53.2%
Total	94	100%

The most common symptom was headache (18.1%), while blurring of vision was reported by 12.7% of participants.

Table 7: Severity of Preeclampsia

CLINICAL PROFILE	n=94	Percent
PE without Severe Feature	44	46.8%
PE with Severe features	41	43.6%
Eclampsia	9	9.6%
Total	94	100.0%

46.8% had preeclampsia without severe features, while 43.6% had severe preeclampsia, and 9.6% had eclampsia.

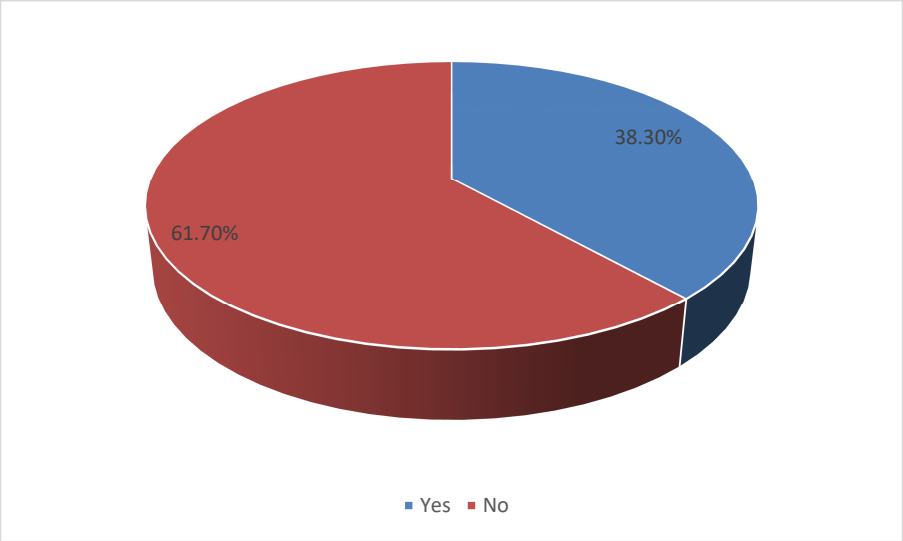


Graph 5: Severity of Preeclampsia

Table 8: Presence of Fetal Growth Restriction (FGR)

		n=94	Percent
Fetal growth restriction	Yes	36	38.3%
	No	58	61.7%
	Total	94	100.0%

38.3% of pregnancies were affected by FGR.

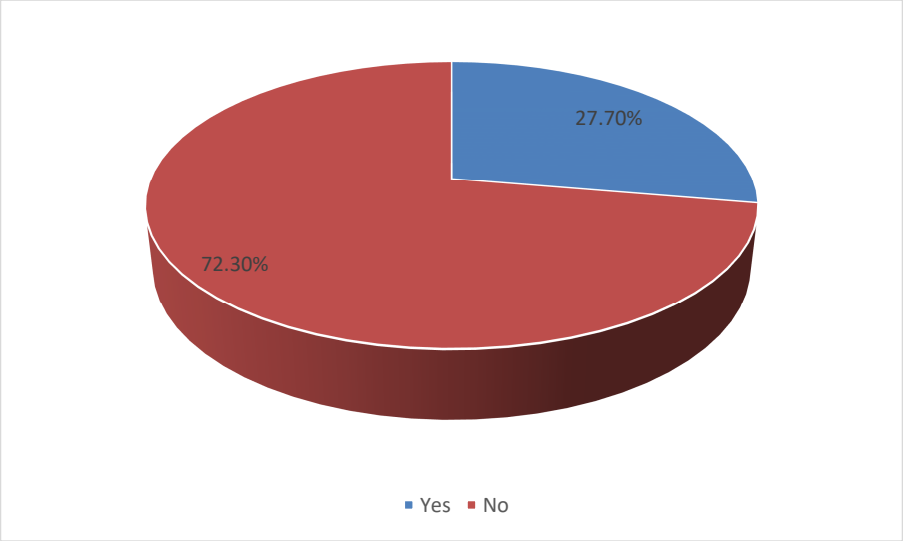


Graph 6: Presence of Fetal Growth Restriction (FGR)

Table 9: Umbilical artery doppler

		n=94	Percent
Umbilical artery doppler	Yes	26	27.7%
	No	68	72.3%
	Total	94	100.0%

26 cases (27.7%) showed abnormal Umbilical artery doppler findings, while 68 cases (72.3%) had normal Doppler results.

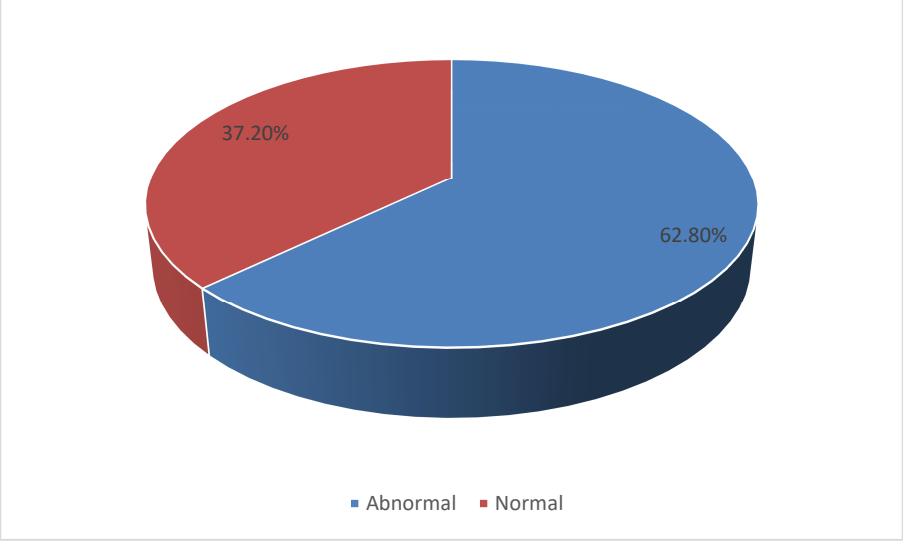


Graph 7: Umbilical artery doppler

Table 10: PlGF Classification

		n=94	Percent
PIGF Classification	Abnormal	59	62.8%
	Normal	35	37.2%
	Total	94	100.0%

59 cases (62.8%) were classified as having abnormal PlGF levels, while 35 cases (37.2%) had normal PlGF levels.



Graph 8: PlGF Classification

Table 11: Preceding Medical History

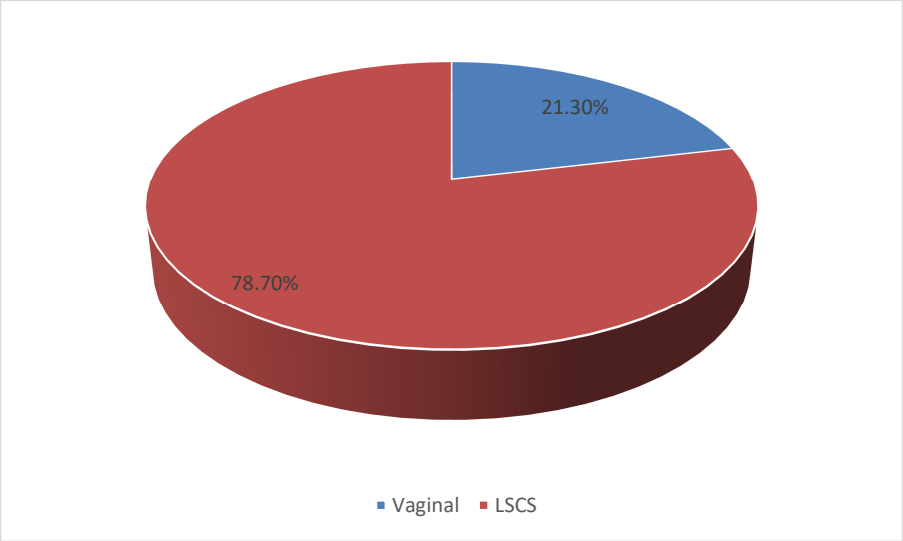
Preceding Medical History	n=94	Percent
Heart disease	1	1.1%
Hypothyroidism	21	22.3%
Chronic hypertension super imposed with PE	12	12.8%
Diabetes mellitus	15	16.0%
No preceding medical history	45	47.8%
Total	94	100%

22.3% of participants had hypothyroidism, and 16% had diabetes. Heart disease was reported in only 1 participant (1.1%).

Table 12: Mode of Delivery

		n=94	Percent
Mode of Delivery	Vaginal	20	21.3%
	LSCS	74	78.7%
	Total	94	100.0%

74 cases (78.7%) were delivered by lower segment caesarean section (LSCS), while 20 cases (21.3%) were delivered vaginally.

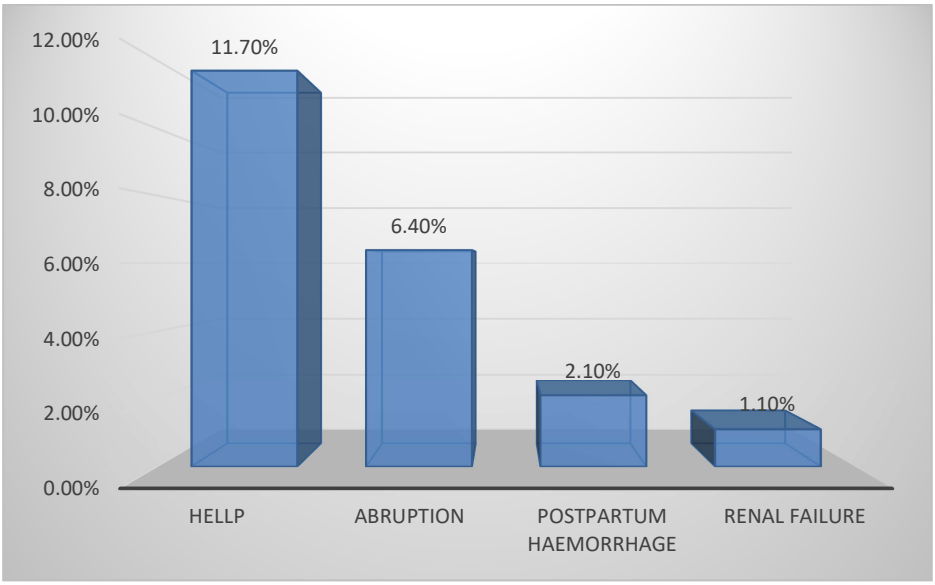


Graph 9: Mode of Delivery

Table 13: Maternal complications

Maternal complications	n=94	Percent
HELLP	11	11.7%
Abruption	6	6.4%
Postpartum haemorrhage	2	2.1%
Renal failure	1	1.1%

11 cases (11.7%) were complicated by HELLP syndrome (Haemolysis, Elevated Liver enzymes, and Low Platelet count), Placental abruption was reported in 6 cases (6.4%), Postpartum haemorrhage (PPH) occurred in 2 cases (2.1%), Renal failure was observed in 1 case (1.1%).

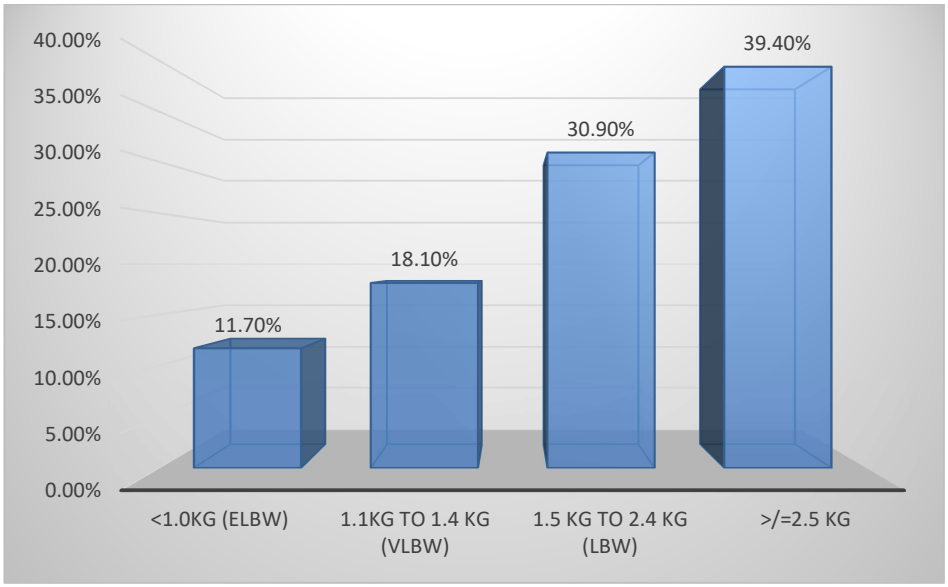


Graph 10: Maternal Complications

Table 14: Birth Weight Distribution

		n=94	Percent
Birth Weight	<1.0kg (ELBW)	11	11.7%
	1.1kg to 1.4 kg (VLBW)	17	18.1%
	1.5 kg to 2.4 kg (LBW)	29	30.9%
	>/=2.5 kg	37	39.4%
	Total	94	100.0%

Out of 94 newborns, 37 cases (39.4%) had a birth weight of ≥2.5 kg (normal birth weight), while 29 cases (30.9%) were classified as low birth weight (LBW). 17 cases (18.1%) had very low birth weight (VLBW), and 11 cases (11.7%) had extremely low birth weight (ELBW).



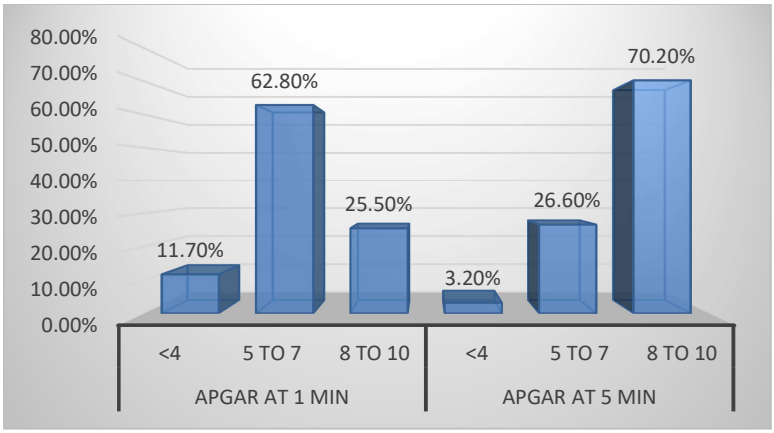
Graph 11: Birth Weight Distribution

Table 15: APGAR Scores at 1 and 5 Minutes

		n=94	Percent
APGAR at 1 min	<4	11	11.7%
	5 to 7	59	62.8%
	8 to 10	24	25.5%
APGAR at 5 min	<4	3	3.2%
	5 to 7	25	26.6%
	8 to 10	66	70.2%

At 1 minute, the majority of newborns (59 cases; 62.8%) had an APGAR score between 5 and 7, indicating mild to moderate respiratory or circulatory depression. 24 cases (25.5%) had an APGAR score between 8 and 10, reflecting good physical condition at birth, while 11 cases (11.7%) had a score of <4, suggesting severe depression and requiring immediate resuscitation.

At 5 minutes, the percentage of newborns with an APGAR score between 8 and 10 increased to 66 cases (70.2%), indicating that most newborns showed significant improvement with medical support. 25 cases (26.6%) maintained scores between 5 and 7, while only 3 cases (3.2%) had scores below 4, suggesting ongoing respiratory or circulatory compromise.

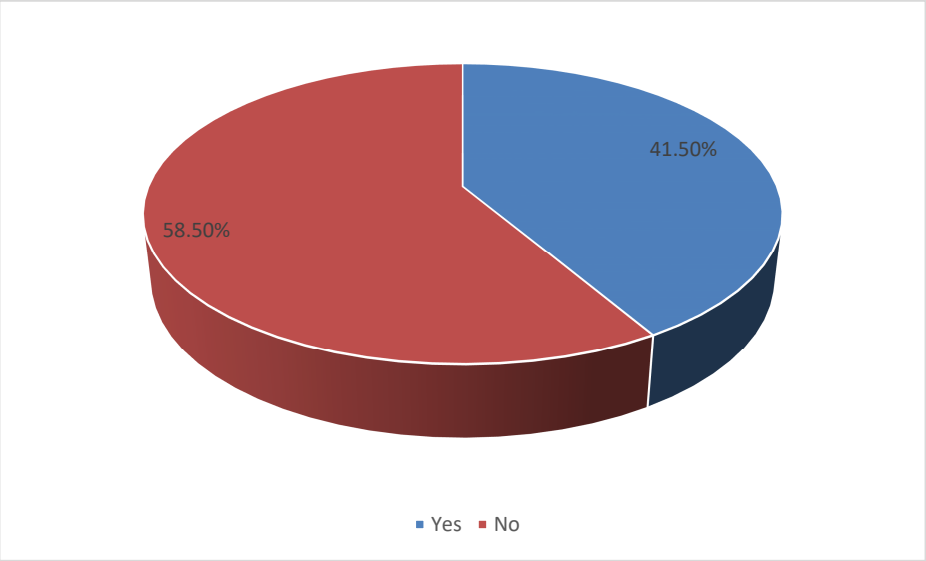


Graph 12: APGAR Scores at 1 and 5 Minutes

Table 16: NICU Admissions

		n=94	Percent
NICU Admission	Yes	39	41.5%
	No	55	58.5%
	Total	94	100.0%

Out of 94 newborns, 39 cases (41.5%) required NICU admission, while 55 cases (58.5%) did not require intensive care.

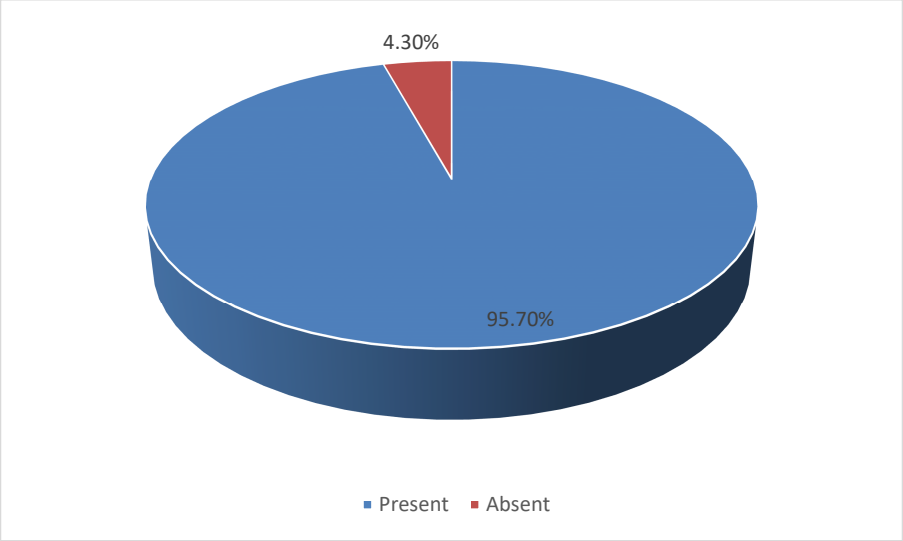


Graph 13: NICU Admissions

Table 17: Live Births at delivery

		n=94	Percent
Live birth	Live birth	90	95.7%
	Still birth	4	4.3%
	Total	94	100.0%

Out of 94 deliveries, 90 cases (95.7%) resulted in live births, while 4 cases (4.3%) resulted in stillbirths.



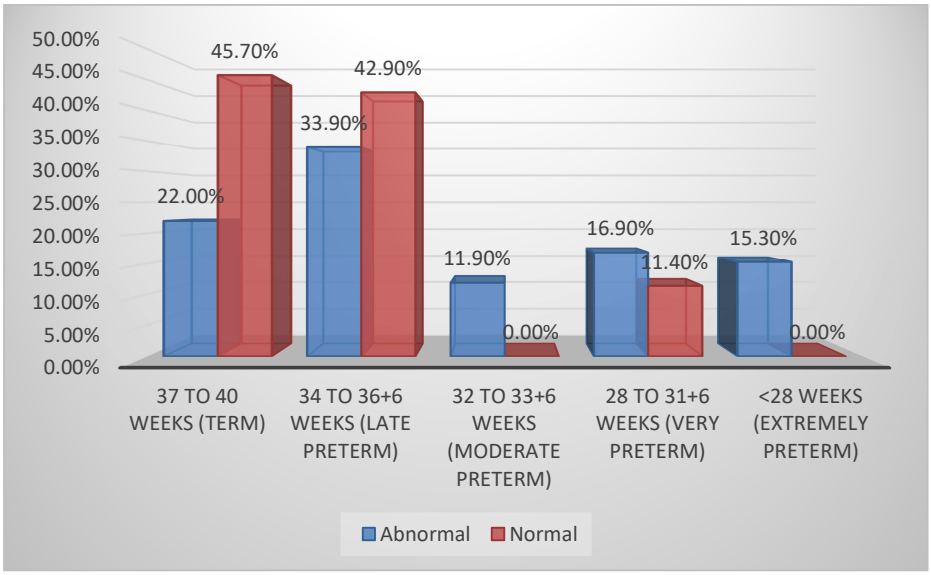
Graph 14: Live Births at delivery

Table 18: PlGF Classification with Gestational age at delivery

			PIGF Classification		Total	P value
			Abnormal	Normal		
Gestational Age at delivery	37 to 40 weeks (Term)	n	13	16	29	0.02
		%	22.0%	45.7%	30.9%	
	34 to 36+6 weeks (Late Preterm)	n	20	15	35	0.51
		%	33.9%	42.9%	37.2%	
	32 to 33+6 weeks (Moderate Preterm)	n	7	0	7	0.08
		%	11.9%	0.0%	7.4%	
	28 to 31+6 weeks (Very Preterm)	n	10	4	14	0.66
		%	16.9%	11.4%	14.9%	
	<28 weeks (Extremely Preterm)	n	9	0	9	0.03
		%	15.3%	0.0%	9.6%	
Total		n	59	35	94	0.006
		%	100.0%	100.0%	100.0%	

Chi-Square: 14.40, P Value: 0.006, Statistically significant

Out of 94 pregnancies, 59 cases (62.8%) were classified as having abnormal PlGF levels, while 35 cases (37.2%) had normal PlGF levels. Among the pregnancies with abnormal PlGF levels, 20 cases (33.9%) were delivered between 34 and 36+6 weeks (late preterm), and 13 cases (22.0%) were delivered at term (37 to 40 weeks). However, 16.9% of cases with abnormal PlGF were delivered at <32 weeks (very preterm), and 15.3% were delivered at <28 weeks (extremely preterm), reflecting a strong link between low PlGF levels and early delivery. The association was found to be statistically significant.



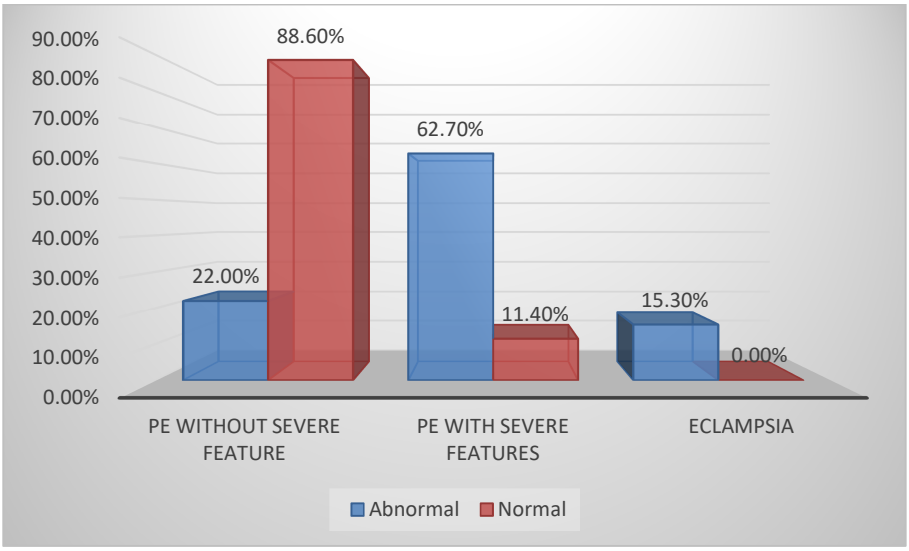
Graph 15: PlGF Classification vs Gestational Age

Table 19: Association Between PlGF Classification and Severity of Preeclampsia

			PIGF Classification		Total	P value
			Abnormal	Normal		
Severity	PE without Severe Feature	n	13	31	44	0.001
		%	22.0%	88.6%	46.8%	
	PE with Severe features	n	37	4	41	0.001
		%	62.7%	11.4%	43.6%	
	Eclampsia	n	9	0	9	0.03
		%	15.3%	0.0%	9.6%	
Total		n	59	35	94	0.001
		%	100.0%	100.0%	100.0%	

Chi-Square: 39.36, P Value: 0.001, Statistically significant

Out of 94 pregnancies, 59 cases (62.8%) were classified as having abnormal PlGF levels, while 35 cases (37.2%) had normal PlGF levels. Among cases with abnormal PlGF levels, the majority (37 cases; 62.7%) had severe preeclampsia, while 9 cases (15.3%) progressed to eclampsia, highlighting a strong correlation between low PlGF levels and the severity of disease. In contrast, only 13 cases (22.0%) with abnormal PlGF levels had preeclampsia without severe features. On the other hand, among cases with normal PlGF levels, 31 cases (88.6%) had preeclampsia without severe features, while only 4 cases (11.4%) developed severe preeclampsia, and none progressed to eclampsia. The Chi-square value of 39.36 and the highly significant p-value of 0.001 confirm a strong and statistically significant association between low PlGF levels and disease severity.



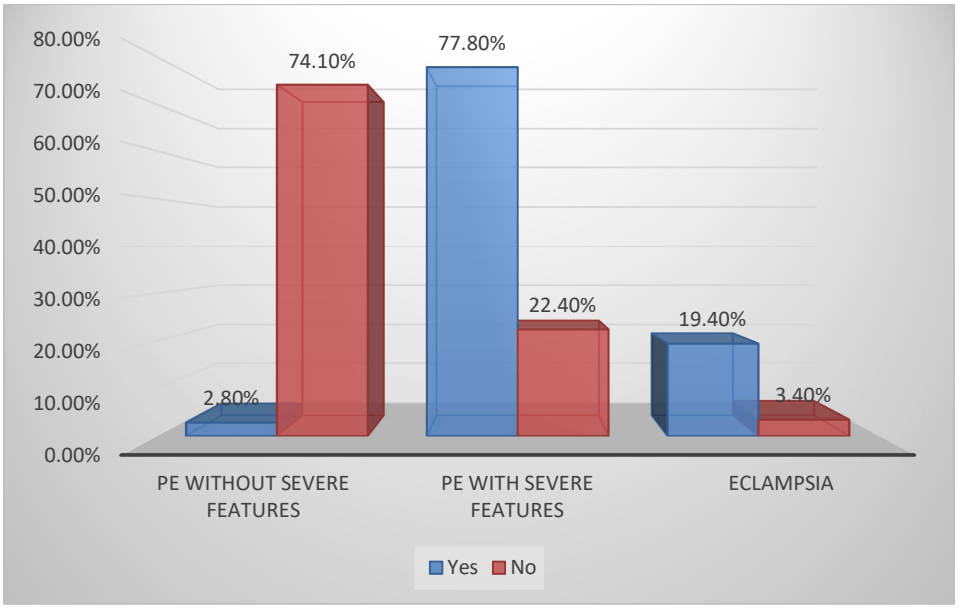
Graph 16: Association Between PlGF Classification and Severity of Preeclampsia

Table 20: Association Between Fetal Growth Restriction (FGR) and Severity of Preeclampsia

			Fetal growth restriction		Total
			Yes	No	
Severity	PE without Severe Features	n	1	43	44
		%	2.8%	74.1%	46.8%
	PE with Severe features	n	28	13	41
		%	77.8%	22.4%	43.6%
	Eclampsia	n	7	2	9
		%	19.4%	3.4%	9.6%
Total		n	36	58	94
		%	100.0%	100.0%	100.0%

Chi-Square: 45.71, P Value: 0.001, Statistically significant

Out of 94 pregnancies, 36 cases (38.3%) were complicated by FGR, while 58 cases (61.7%) had normal fetal growth. Among cases of severe preeclampsia, 28 cases (77.8%) were associated with FGR. Among cases of preeclampsia without severe features, only 1 case (2.8%) was complicated by FGR, while 43 cases (74.1%) had normal fetal growth. Among cases of eclampsia, 7 cases (19.4%) were complicated by FGR.



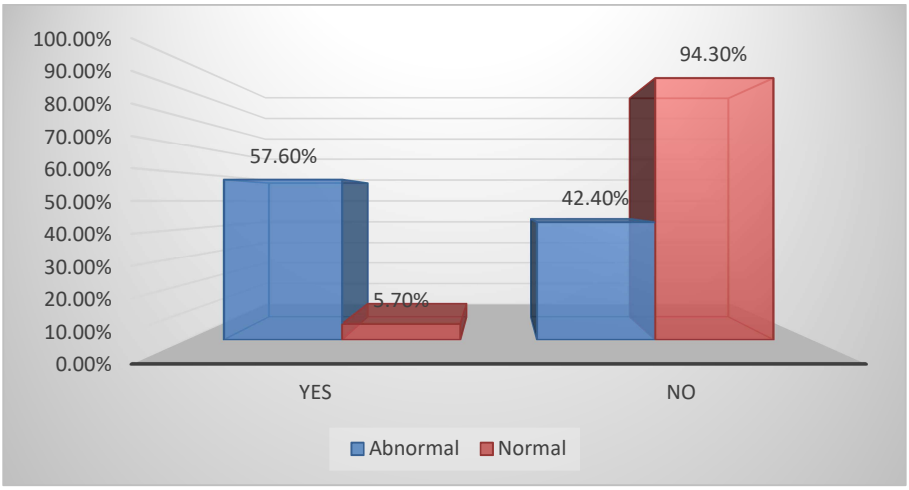
Graph 17: Association Between Fetal Growth Restriction (FGR) and Severity of Preeclampsia

Table 21: Association Between PlGF Classification and Fetal Growth Restriction (FGR)

			PIGF Classification		Total
			Abnormal	Normal	
Fetal growth restriction	Yes	n	34	2	36
		%	57.6%	5.7%	38.3%
	No	n	25	33	58
		%	42.4%	94.3%	61.7%
Total		n	59	35	94
		%	100.0%	100.0%	100.0%

Chi-Square: 25.05, P Value: 0.001, Statistically significant

Out of 94 pregnancies, 36 cases (38.3%) were complicated by FGR, while 58 cases (61.7%) had normal fetal growth. Among the pregnancies with abnormal PlGF levels, 34 cases (57.6%) were associated with FGR. Only 2 cases (5.7%) with normal PlGF levels were complicated by FGR. Among cases without FGR, 33 cases (94.3%) had normal PlGF levels. The Chi-square value of 25.05 and the highly significant p-value of 0.001.



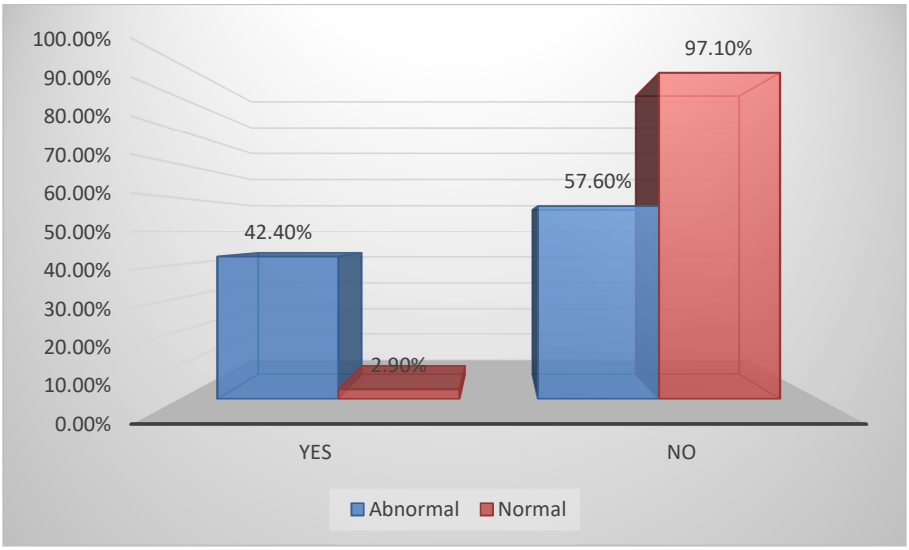
Graph 18: Association Between PlGF Classification and Fetal Growth Restriction (FGR)

Table 22: Association Between PlGF Classification and umbilical artery Doppler Findings

			PIGF Classification		Total
			Abnormal	Normal	
Umbilical artery doppler	Yes	n	25	1	26
		%	42.4%	2.9%	27.7%
	No	n	34	34	68
		%	57.6%	97.1%	72.3%
Total		n	59	35	94
		%	100.0%	100.0%	100.0%

Chi-Square: 17.14, P Value: 0.001, Statistically significant

Out of 94 pregnancies, 26 cases (27.7%) were complicated by abnormal Doppler findings, while 68 cases (72.3%) had normal Doppler results. Among cases with abnormal PlGF levels, 25 cases (42.4%) were associated with abnormal Doppler findings. Only 1 case (2.9%) with normal PlGF levels had abnormal Doppler results. Among cases with normal Doppler findings, 34 cases (97.1%) had normal PlGF levels. The Chi-square value of 17.14 and the highly significant p-value of 0.001 confirm a statistically significant association between low PlGF levels and abnormal Doppler findings.



Graph 19: Association Between PlGF Classification and umbilical artery Doppler Findings

Table 23: Association Between PlGF Classification and Maternal Complications

			PIGF Classification		Total	P Value
			Abnormal	Normal		
Abruption	Present	n	6	0	6	0.05
		%	10.2%	0.0%	6.4%	
	Absent	n	53	35	88	
		%	89.8%	100.0%	93.6%	
		%	100.0%	100.0%	100.0%	
Renal failure	Present	n	1	0	1	0.62
		%	1.7%	0.0%	1.1%	
	Absent	n	58	35	93	
		%	98.3%	100.0%	98.9%	
Postpartum haemorrhage	Present	n	1	1	2	0.60
		%	1.7%	2.9%	2.1%	
	Absent	n	58	34	92	
		%	98.3%	97.1%	97.9%	
HELLP	Present	n	7	4	11	0.61
		%	11.9%	11.4%	11.7%	
	Absent	n	52	31	83	
		%	88.1%	88.6%	88.3%	

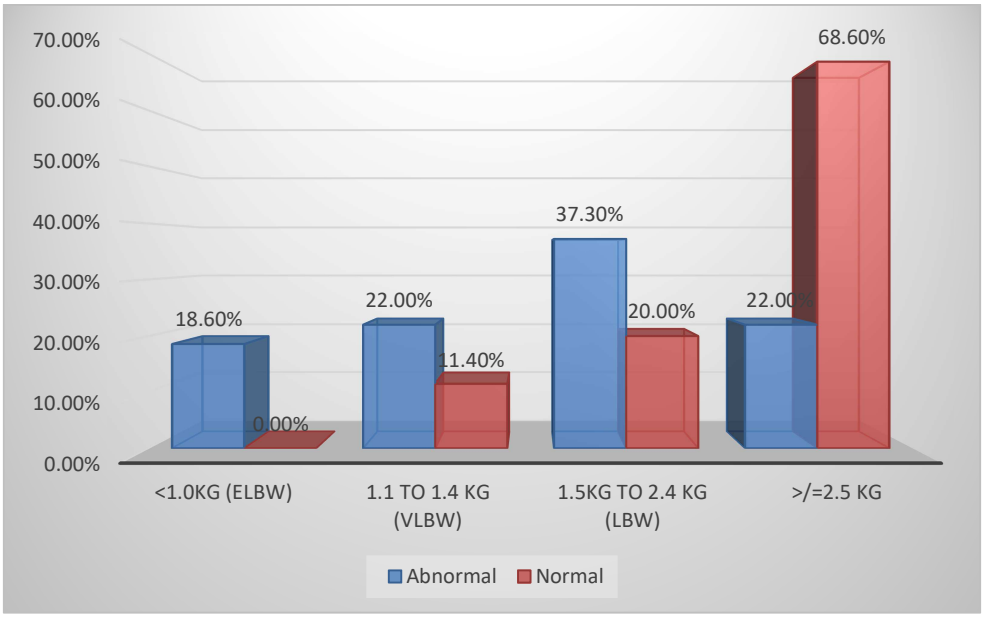
Placental abruption was reported in 6 cases (6.4%), and all were associated with abnormal PlGF levels (10.2%), with a statistically significant p-value of 0.05. Renal failure was observed in 1 case (1.1%) and was linked to abnormal PlGF levels, though the association was not statistically significant (p = 0.62). Postpartum hemorrhage

occurred in 2 cases (2.1%), one in the abnormal PlGF group and one in the normal PlGF group, with no significant difference between the two groups (p = 0.60). HELLP syndrome was reported in 11 cases (11.7%), with 7 cases (11.9%) in the abnormal PlGF group and 4 cases (11.4%) in the normal PlGF group, indicating no significant difference between groups (p = 0.61). Notably, there were no reported cases of disseminated intravascular coagulation (DIC), cerebrovascular accident, or respiratory distress in either group.

Table 24: Association Between PlGF Classification and Birth Weight

			PIGF Classification		Total	P Value
			Abnormal	Normal		
Birth Weight	<1.0kg (ELBW)	n	11	0	11	0.006
		%	18.6%	0.0%	11.7%	
	1.1 to 1.4 kg (VLBW)	n	13	4	17	0.19
		%	22.0%	11.4%	18.1%	
	1.5kg to 2.4 kg (LBW)	n	22	7	29	0.07
		%	37.3%	20.0%	30.9%	
	>/=2.5 kg	n	13	24	37	0.001
		%	22.0%	68.6%	39.4%	
Total		n	59	35	94	0.001
		%	100.0%	100.0%	100.0%	

Among cases with abnormal PlGF levels, 11 cases (18.6%) had extremely low birth weight (ELBW), while none of the cases with normal PlGF levels were in this category. Similarly, 13 cases (22.0%) with abnormal PlGF levels had very low birth weight (VLBW) compared to 4 cases (11.4%) with normal PlGF levels. Low birth weight (LBW) was reported in 22 cases (37.3%) with abnormal PlGF levels, compared to 7 cases (20.0%) with normal PlGF levels. In contrast, normal birth weight (≥2.5 kg) was more common among cases with normal PlGF levels (24 cases; 68.6%) compared to those with abnormal PlGF levels (13 cases; 22.0%). The Chi-square value of 22.10 and the highly significant p-value of 0.001 confirm a statistically significant association between low PlGF levels and poor fetal growth.



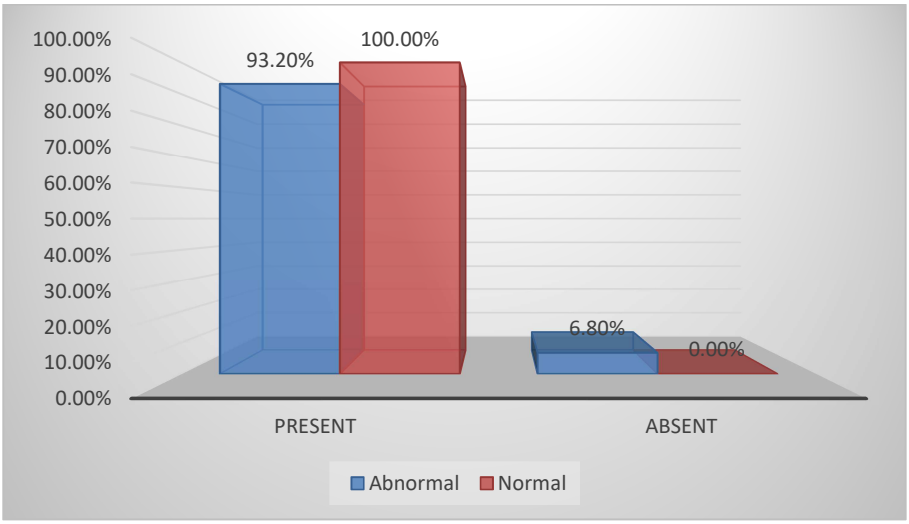
Graph 20: Association Between PlGF Classification and Birth Weight

Table 25: Association Between PlGF Classification and Live Birth

			PIGF Classification		Total
			Abnormal	Normal	
Live birth	Live birth	n	55	35	90
		%	93.2%	100.0%	95.7%
	Still birth	n	4	0	4
		%	6.8%	0.0%	4.3%
Total		n	59	35	94
		%	100.0%	100.0%	100.0%

Chi-Square: 2.47, P Value: 0.14, Statistically not significant

Among cases with abnormal PlGF levels, 55 cases (93.2%) resulted in live births, whereas all cases with normal PlGF levels (100%) resulted in live births. 4 cases (6.8%) with abnormal PlGF levels resulted in stillbirth, while none of the cases with normal PlGF levels had stillbirths. The Chi-square value of 2.47 and the p-value of 0.14 indicate that the association between PlGF levels and live birth outcomes is not statistically significant.



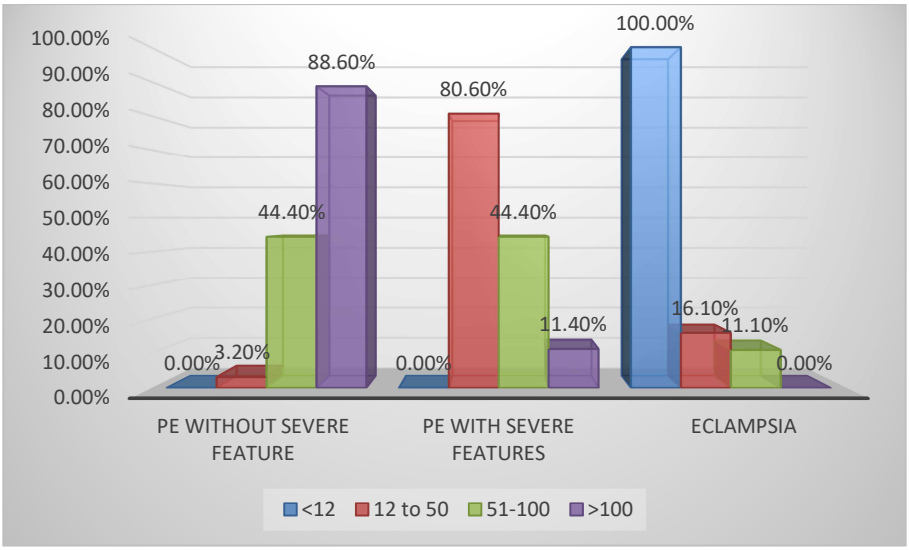
Graph 21: Association Between PlGF Classification and Live Birth

Table 26: Association Between PlGF Levels and Severity of Preeclampsia

Clinical profile		PIGF Classification				Total
		<12	12-50	51-100	>100	
PE without Severe Feature	n	0	1	12	31	44
	%	0.0%	3.2%	44.4%	88.6%	46.8%
PE with Severe features	n	0	25	12	4	41
	%	0.0%	80.6%	44.4%	11.4%	43.6%
Eclampsia	n	1	5	3	0	9
	%	100.0%	16.1%	11.1%	0.0%	9.6%
Total	n	1	31	27	35	94
	%	100.0%	100.0%	100.0%	100.0%	100.0%

Chi-Square: 57.96, P Value: 0.001, Statistically significant

Among cases of preeclampsia without severe features, the majority (31 cases; 88.6%) had PlGF levels above 100 pg/mL, and none of these cases had PlGF levels below 12 pg/mL. 12 cases (44.4%) had PlGF levels between 51–100 pg/mL, while only 1 case (3.2%) had PlGF levels between 12–50 pg/mL. Among cases of severe preeclampsia, the largest proportion of cases (25 cases; 80.6%) had PlGF levels between 12–50 pg/mL. Only 4 cases (11.4%) with severe preeclampsia had PlGF levels above 100 pg/mL, and none of the severe cases had PlGF levels below 12 pg/mL. Among cases of eclampsia, 1 case (100%) had PlGF levels below 12 pg/mL. Additionally, 5 cases (16.1%) of eclampsia had PlGF levels between 12–50 pg/mL, and 3 cases (11.1%) had levels between 51–100 pg/mL. The Chi-square value of 57.96 and the highly significant p-value of 0.001 confirm a statistically significant association between low PlGF levels and the severity of preeclampsia.



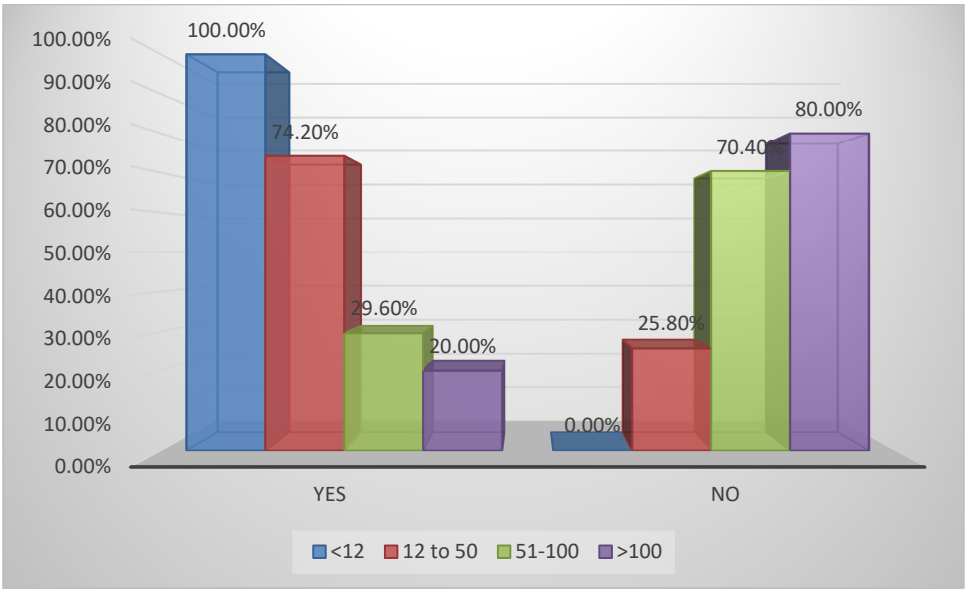
Graph 22: Association Between PlGF Levels and Severity of Preeclampsia

Table 27: Association Between PlGF Levels and NICU Admission

			PIGF Classification				Total
			<12	12-50	51-100	>100	
NICU Admission	Yes	n	1	23	8	7	39
		%	100.0%	74.2%	29.6%	20.0%	41.5%
	No	n	0	8	19	28	55
		%	0.0%	25.8%	70.4%	80.0%	58.5%
Total		n	1	31	27	35	94
		%	100.0%	100.0%	100.0%	100.0%	100.0%

Chi-Square: 23.29, P Value: 0.001, Statistically significant

Among newborns requiring NICU admission (39 cases; 41.5%), the majority (23 cases; 74.2%) had PlGF levels between 12–50 pg/mL, and 8 cases (29.6%) had levels between 51–100 pg/mL. Notably, the only case with PlGF levels below 12 pg/mL resulted in NICU admission. Only 7 cases (20.0%) with PlGF levels above 100 pg/mL required NICU admission. Among cases not requiring NICU admission (55 cases; 58.5%), the majority (28 cases; 80.0%) had PlGF levels above 100 pg/mL, and 19 cases (70.4%) had levels between 51–100 pg/mL. Only 8 cases (25.8%) with PlGF levels between 12–50 pg/mL avoided NICU admission. The Chi-square value of 23.29 and the highly significant p-value of 0.001 confirm a statistically significant association between low PlGF levels and the need for NICU admission.



Graph 23: Association Between PlGF Levels and NICU Admission

Table 28: Association Between PlGF Levels and Maternal Complications

			PIGF Classification				Total	P Value
			<12	12-50	51-100	>100		
Abruption	Present	n	1	5	0	0	6	0.001
		%	100.0%	16.1%	0.0%	0.0%	6.4%	
	Absent	n	0	26	27	35	88	
		%	0.0%	83.9%	100.0%	100.0%	93.6%	
Renal failure	Present	n	0	1	0	0	1	0.56
		%	0.0%	3.2%	0.0%	0.0%	1.1%	
	Absent	n	1	30	27	35	93	
		%	100.0%	96.8%	100.0%	100.0%	98.9%	
Postpartum haemorrhage	Present	n	0	1	0	1	2	0.83
		%	0.0%	3.2%	0.0%	2.9%	2.1%	
	Absent	n	1	30	27	34	92	
		%	100.0%	96.8%	100.0%	97.1%	97.9%	
HELLP	Present	n	0	4	3	4	11	0.98
		%	0.0%	12.9%	11.1%	11.4%	11.7%	
	Absent	n	1	27	24	31	83	
		%	100.0%	87.1%	88.9%	88.6%	88.3%	

Placental abruption was reported in 6 cases (6.4%), with 1 case (100%) occurring in the group with PlGF levels <12 pg/mL and 5 cases (16.1%) in the 12–50 pg/mL group. No cases of abruption were reported in the higher PlGF categories, indicating a significant association between low PlGF levels and placental abruption ($p = 0.001$). Renal failure was reported in only 1 case (1.1%), which occurred in the 12–50 pg/mL group ($p = 0.56$, not statistically significant). Postpartum hemorrhage (PPH) occurred in 2 cases (2.1%), one case in the 12–50 pg/mL group and one case in the >100 pg/mL group, with no significant association ($p = 0.83$). HELLP syndrome was reported in 11 cases (11.7%) and was fairly evenly distributed across all PlGF categories, with 4 cases (12.9%) in the 12–50 pg/mL group, 3 cases (11.1%) in the 51–100 pg/mL group, and 4 cases (11.4%) in the >100 pg/mL group ($p = 0.98$, not statistically significant).

DISCUSSION

The present study was conducted to evaluate the association of serum placental growth factor (PlGF) levels in pregnant women with preeclampsia, eclampsia and or fetal growth restriction (FGR) and to analyze the maternal and neonatal outcomes. A cross-sectional study design was implemented and the study population included in-patient women presenting to the labor room for delivery of KAHER's Dr. Prabhakar Kore Hospital, Belagavi with preeclampsia, eclampsia and or fetal growth restriction. Patients were informed about the study and the women who expressed an interest in participating the study were enrolled after signing a written informed consent.

AGE DISTRIBUTION (Table 1)

In this study, highest percentage of participants were in the 21–25 years age group (44.7%), followed by the 26–30 years group (26.6%). This reflects the fact that pregnancy is most common during the early and mid-20s, which corresponds to the peak reproductive years. Interestingly, there is a considerable representation of women in their 30s (18.1% in the 31–35 years group), highlighting that pregnancies in older age groups are becoming more common, possibly due to delayed childbearing and lifestyle changes.

Benton et al. reported that the mean maternal age was between 28.5 ± 6.1 years to 30.4 ± 5.8 years[71] .Duhig et al. (2019) reported that the mean maternal age was in the range of 31.5 ± 6.0 years and 31.9 ± 5.9.[66] Sibiude et al. reported that the mean maternal age was 32.2 ± 0.8 years in the adverse outcome group and 34.2 ± 1.0 years in the no adverse outcome group, but the difference was not statistically significant (p = 0.14).[19]

Chappell et al. reported that the median maternal age was 31.9 years (IQR: 27.0–35.9) for women enrolled before 35 weeks' gestation, 32.4 years (IQR: 27.5–35.4) for those between 35 and 36+6 weeks, and 32.1 years (IQR: 27.5–36.0) for those beyond 37 weeks..[50] Villalaín et al. (2020) reported that the mean maternal age was 33.7 ± 5.8 years for the total study population. Women with preeclampsia at inclusion had a mean age of 31.6 ± 5.8 years, while those without preeclampsia had a mean age of 32.0 ± 6.1 years. The difference was not statistically significant (p = 0.49).[82] Sung et al. (2017) reported that the median maternal age was 32 years (range: 20–43 years) for the study population[84.] Verma et al. (2022) reported that the mean maternal age was 25.5 ± 3.3 years in the group that developed preeclampsia or fetal growth restriction (FGR), compared to 26.2 ± 3.7 years in the group that did not develop complications. The difference was not statistically significant (p = 0.30).[81]

This aligns with findings from Verma et al. (2022) who reported a similar younger maternal age group, with a mean age of 25.5 ± 3.3 years in those with complications. However, other studies such as Benton et al. , Duhig et al. (2019), Sibiude et al. , Chappell et al., Villalaín et al. (2020), and Sung et al. (2017) reported higher mean maternal ages ranging from approximately 30 to 34 years, suggesting a trend toward delayed childbearing in their populations.

PARITY (Table 3)

In this study primigravida were 57 in number(60.6%) and multigravida were 37 in number(39.4%) Primigravida women are more susceptible to developing pre-eclampsia and hypertensive disorders due to the lack of prior immune tolerance to paternal antigens and the increased physiological stress associated with the first pregnancy. While multigravida women are generally expected to have lower rates of pre-eclampsia compared to primigravida women, those with a history of pregnancy

complications such as pre-eclampsia or fetal growth restriction are at increased risk of recurrence in subsequent pregnancies and warrants more intense observation.

Benton et al. found that 56.4% of women enrolled were nulliparous [71.] Duhig et al. (2019) reported that 47% to 55% of women were nulliparous in both the groups. The percentage of women with two or more previous pregnancies was slightly higher in the concealed group (15%) than in the revealed group (10%).[66] Sibiude et al. found that 61.2% of women with adverse outcomes were nulliparous, compared to 48.3% in the no adverse outcome group. However, the difference was not statistically significant ($p = 0.24$).[19] Chappell et al. (2013) found that 43% of women enrolled before 35 weeks, 44% of those between 35 and 36+6 weeks, and 44% of those beyond 37 weeks were nulliparous (first-time pregnancy).[50] Villalaín et al. (2020) reported that 67.1% of the study population were nulliparous. Among those with preeclampsia at inclusion, 67.6% were nulliparous, compared to 65.4% in the group without preeclampsia. The difference was not statistically significant ($p = 0.77$).[82] Sung et al. (2017) found that 58.9% of the study population were nulliparous and 41.1% were multiparous[84.] Verma et al. (2022) found that nulliparity was more common in the group that developed preeclampsia or FGR (10 cases) compared to the group without complications (51 cases) ($p = 0.05$).[81]

Similar findings were reported by other authors. Verma et al. (2022) reported that nulliparity was more common in the group that developed preeclampsia or FGR, with a statistically significant association ($p = 0.05$). Overall, the proportion of primigravida women in this study is comparable to findings in other studies.

GESTATIONAL AGE (Table 4)

In this study, high rate of preterm deliveries (nearly 70% of cases occurring before 37 weeks) reflects the substantial impact of maternal conditions such as pre-eclampsia, fetal growth restriction, and placental insufficiency, which are known to trigger early delivery to prevent further maternal and fetal complications. The increased occurrence of very and extremely preterm deliveries underscores the need for early diagnosis, careful monitoring, and timely intervention to prolong gestation and improve neonatal outcomes. The findings reinforce the importance of antenatal surveillance and maternal-fetal monitoring in pregnancies at risk of preterm delivery to minimize adverse perinatal outcomes.

According to Benton et al. , the mean gestational age at enrolment was in the range of 33.0 weeks to 34.3 weeks. Preterm delivery (<37 weeks) was reported in 61.7% of pregnancies [71].

Sibiude et al. reported that the mean gestational age at inclusion was 30 ± 0.55 weeks in the adverse outcome group and 32.6 ± 0.96 weeks in the no adverse outcome group. The difference was statistically significant ($p = 0.01$).[19] Chappell et al. (2013) reported that the median gestational age at enrollment was 31.0 weeks (IQR: 27.9–33.4) for women enrolled before 35 weeks, 36.0 weeks (IQR: 35.4–36.4) for those between 35 and 36+6 weeks, and 38.4 weeks (IQR: 37.6–39.6) for those beyond 37 weeks.[50] Villalaín et al. (2020) reported that the median gestational age at inclusion was 29.0 weeks (IQR: 27.1–31.5 weeks). For women with preeclampsia, the median gestational age at inclusion was 29.4 weeks, compared to 27.7 weeks for those without preeclampsia ($p < 0.01$).[82] Sung et al. (2017) reported that the median gestational age at delivery was 38.0 weeks (range: 15–41 weeks)[84.] Comparable results have been documented in earlier studies.

BMI (Table 5)

In this study, mean BMI of the participants was 29.27 with a standard deviation of 10.76. A BMI above 25 kg/m² is classified as overweight, while a BMI above 30 kg/m² is classified as obese according to WHO guidelines. The mean BMI in this study suggests that a significant proportion of the study population was overweight or obese, which is consistent with the increased prevalence of metabolic disorders and lifestyle changes in the general population.

Benton et al. reported that the pre-pregnancy weight was 60.7 ± 14.0 kg to 61.5 ± 11.5 kg.[71]Sibiude et al. reported that the mean BMI was 23.9 ± 0.7 kg/m² in the adverse outcome group and 26.9 ± 1.3 kg/m² in the no adverse outcome group, with a statistically significant difference (p = 0.03).[19] Chappell et al. reported that the median BMI was 28.6 kg/m² (IQR: 24.2–33.6) for women enrolled before 35 weeks, 28.6 kg/m² (IQR: 24.4–32.7) for those between 35 and 36+6 weeks, and 26.9 kg/m² (IQR: 23.1–31.2) for those beyond 37 weeks.[50] Villalaín et al. (2020) reported that the mean pre-pregnancy BMI was 26.7 ± 6.4 kg/m² for the total population. There was no significant difference in BMI between women with and without preeclampsia (p = 0.96).[82] Sung et al. (2017) reported that the median BMI was 21.8 kg/m² (range: 16.2–36.0 kg/m²). PlGF levels were lower in women with a BMI ≥25 kg/m² compared to those with BMI <25 kg/m², but the difference was not statistically significant (p = 0.07).[84] Verma et al. (2022) reported that the mean BMI was 23.8 ± 4.6 kg/m² in the group with complications and 23.3 ± 4.4 kg/m² in the uncomplicated group, but the difference was not statistically significant (p = 0.12).[81] There were a mixed set of findings in the studies discussed above.

PE WITH AND WITHOUT SEVERE FEATURES(Table 7)

In this study, 46.8% had preeclampsia without severe features, while 43.6% had severe preeclampsia, and 9.6% had eclampsia.

Sibiude et al. reported that preeclampsia occurred in 40 cases (41.7).[19] Chappell et al. found that mild preeclampsia was diagnosed in 25 cases before 35 weeks, 24 cases between 35 and 36+6 weeks, and 40 cases after 37 weeks. Severe preeclampsia was more common before 35 weeks (26%) and declined to 23% between 35 and 36+6 weeks and 11% beyond 37 weeks.[50] Villalaín et al. (2020) reported that severe features of preeclampsia were present in 49.2% of cases at inclusion and increased to 77.3% at delivery. Severe maternal complications were more common in cases with preeclampsia at inclusion ($p < 0.01$).[82] Verma et al. (2022) reported that the incidence of preeclampsia was 9.3% (15 out of 161 cases). Of these, 12 cases were non-severe and 3 cases were severe preeclampsia. One case progressed to eclampsia, and one case developed postpartum acute renal failure.[81] These findings are in line with multiple studies

FGR(Table 8)

In this study, total of 94 pregnancies were evaluated for the presence of FGR, out of which 36 cases (38.3%) were identified as having FGR, while 58 cases (61.7%) had normal fetal growth. The relatively high prevalence of FGR (38.3%) in this study indicates a substantial burden of placental dysfunction, which is commonly associated with hypertensive disorders of pregnancy, including preeclampsia. FGR is often linked to increased risks of stillbirth, neonatal morbidity, and long-term developmental issues. However, the high percentage of FGR cases highlights the need for close antenatal monitoring, including Doppler ultrasound assessment of umbilical artery blood flow, to identify early signs of placental insufficiency. Therefore, identifying pregnancies at risk for FGR through early screening of maternal serum

biomarkers such as placental growth factor (PlGF) and Doppler studies can help improve fetal outcomes.

Sibiude et al. (2012) found that 55.2% of cases with adverse outcomes were due to fetal growth restriction (FGR), compared to only 3.4% in the no adverse outcome group ($p < 0.001$).[19] Chappell et al. (2013) reported that suspected fetal growth restriction (FGR) was present in 9% of cases before 35 weeks, 3% of cases between 35 and 36+6 weeks, and 1% of cases beyond 37 weeks. [50] Benton et al. (2016) reported the same findings.[71] Villalaín et al. (2020) reported that fetal growth restriction (FGR) was present in 77.3% of cases with preeclampsia at inclusion and 82.7% of cases without preeclampsia. At delivery, FGR was present in 84.9% of cases with preeclampsia and 90.4% of cases without preeclampsia. [82] Verma et al. (2022) reported that the incidence of FGR was 19.8% (32 out of 161 cases). Among the 15 women who developed preeclampsia, 7 cases (46.6%) were complicated by FGR.[81]

These results are supported by previous literature. Villalaín et al. (2020) reported that FGR was present in over 80% of cases, both with and without preeclampsia, at the time of delivery. Verma et al. (2022) reported that nearly 47% of preeclampsia cases were complicated by FGR.

The above finding are supported by the previous literature.

ABNORMAL DOPPLER (Table 9)

In this study, total of 94 pregnancies were assessed using Doppler studies, out of which 26 cases (27.7%) showed abnormal Umbilical Artery Doppler findings, while 68 cases (72.3%) had normal Doppler results. Abnormal Umbilical Artery Doppler findings typically indicate impaired placental perfusion and fetal compromise. The most common abnormalities include increased umbilical artery resistance (indicating

placental insufficiency), absent or reversed end-diastolic flow (suggesting severe placental dysfunction). The presence of abnormal Doppler findings in 27.7% of cases underscores the significant burden of placental dysfunction in the study population. This aligns with the high incidence of fetal growth restriction (FGR) and preterm delivery reported in the study, which are commonly linked to poor placental blood flow. However, the presence of abnormal Doppler findings in over a quarter of the cases indicates that these pregnancies were at increased risk for adverse outcomes, including fetal distress, preterm delivery, and neonatal intensive care unit (NICU) admission. Abnormal Doppler results are strong predictors of adverse perinatal outcomes, particularly in pregnancies complicated by severe preeclampsia and FGR. The high prevalence of abnormal umbilical artery doppler findings in this study highlights the importance of regular fetal surveillance in high-risk pregnancies. Early detection of Doppler abnormalities allows for timely intervention, including the use of corticosteroids for fetal lung maturity, planned early delivery, and close neonatal care, which can significantly improve neonatal survival and reduce the risk of long-term complications. These findings reinforce the need for routine Doppler assessment in pregnancies complicated by hypertensive disorders and FGR to improve fetal outcomes and reduce perinatal morbidity and mortality.

Sibiude et al. reported that umbilical artery doppler abnormalities were more frequent in these cases and also associated with increased adverse outcomes.[19] Chappell et al. reported that abnormal Doppler findings (elevated resistance in the umbilical artery) in these cases. Doppler abnormalities were identified in 43% of cases before 35 weeks[50.] Benton et al. (2016) reported that the umbilical artery doppler was markedly higher in these cases.[71] Villalaín et al. (2020) reported that Doppler abnormalities were identified in 77.3% of cases with preeclampsia and 82.7% of cases without

preeclampsia at inclusion.[82] This observation is supported by earlier research done by the above authors.

SERUM PLFG LEVELS (Table 10)

In this study, 59 cases (62.8%) were classified as having abnormal PlGF levels, while 35 cases (37.2%) had normal PlGF levels.

Benton et al. (2016) reported that low PlGF was defined as levels below the 5th percentile for gestational age, with an area under the ROC curve of 0.96 for detecting FGR. Very low PlGF (<12 pg/mL) was associated with a shorter sampling-to-delivery interval (13 days vs. 29.5 days for normal PlGF). Sibiude et al. reported that the mean log-transformed PlGF level was significantly lower for women who developed preeclampsia (2.9 vs. 3.7, $p = 0.02$) and even lower for those who experienced adverse outcomes (2.9 vs. 4.3, $p < 0.001$).[71] Villalaín et al. (2020) reported that sFlt-1/PlGF ratio >655 was associated with preeclampsia and rapid progression of placental dysfunction. The median sFlt-1/PlGF ratio was 823 (IQR: 718–10 51) with no significant difference between groups ($p = 0.92$).[82] Sung et al. (2017) reported that the median PlGF MoM level was 0.92 (95% CI: 0.22–2.85) for the total population. Low PlGF levels were significantly associated with FGR but not with preeclampsia.[84] Verma et al. (2022) reported that mean PlGF levels were significantly lower in the preeclampsia and FGR group (1.32 ± 0.57 pg/mL) compared to the group without complications (7.39 ± 17.5 pg/mL) ($p < 0.04$).[81] These results are in agreement with prior studies mentioned in the literature.

MATERNAL COMPLICATIONS(Table 13)

In this study, 11 cases (11.7%) were complicated by HELLP syndrome (Hemolysis, Elevated Liver enzymes, and Low Platelet count), Placental abruption was reported in

6 cases (6.4%), Postpartum hemorrhage (PPH) occurred in 2 cases (2.1%), Renal failure was observed in 1 case (1.1%). Benton et al. (2016) reported that placental abruption occurred in 6.4% of cases. HELLP syndrome was reported in 12.8% of the above cases .[71] Sibiude et al. (2012) reported that severe maternal complications, including HELLP syndrome (5.2%) were diagnosed more frequently. Placental abruption was reported in 6.4% of cases .[19] Chappell et al. (2013) reported that HELLP syndrome occurred in 2 cases before 35 weeks and none after 35 weeks. Placental abruption was reported in 4 cases before 35 weeks.[50] Villalaín et al. (2020) reported that severe maternal morbidity occurred in 41.8% of cases. HELLP syndrome was reported in 13.5% of cases with preeclampsia and 5.8% of cases without preeclampsia. Abruptio placentae occurred in 8.7% of cases with preeclampsia and 21.2% of cases without preeclampsia ($p = 0.01$).[82] Verma et al. (2022) reported that one woman developed acute renal failure postpartum. Severe maternal complications, including HELLP syndrome and placental abruption, were more common in the preeclampsia group.[81] The findings of this study are comparable to existing literature.

FETAL OUTCOMES- BIRTH WEIGHT, NICU ADMISSION (Table 14,16)

In this study, out of 94 newborns, 37 cases (39.4%) had a birth weight of ≥2.5 kg (normal birth weight), while 29 cases (30.9%) were classified as low birth weight (LBW). 17 cases (18.1%) had very low birth weight (VLBW), and 11 cases (11.7%) had extremely low birth weight (ELBW). Out of 94 newborns, 39 cases (41.5%) required NICU admission, while 55 cases (58.5%) did not require intensive care.

Benton et al. reported that mean birth weight was significantly lower in their study participants (1855 ± 721 g) [71]. NICU admission was required in 27.7% of newborns.[71] Sibiude et al. reported that the mean birth weight was 2144 g in their cases with

56.7% requiring NICU admission.[19] Chappell et al. reported that the median birth weight was 2420 g (IQR: 1620–3125 g) for babies delivered before 35 weeks, 2820 g (IQR: 2340–3340 g) for those delivered between 35 and 36+6 weeks, and 3278 g (IQR: 2980–3560 g) for those delivered after 37 weeks. NICU admission was required in 53% of babies born before 35 weeks compared to 36% between 35 and 36+6 weeks and 6.4% after 37 weeks.[50] Villalaín et al. (2020) reported that the mean birth weight was 664 ± 445 g. NICU admission was required in 81.4% of cases, with higher rates in cases with preeclampsia (83.2%) than without preeclampsia (75%) .[82]Verma et al. (2022) reported that the mean birth weight was 2.6 ± 0.69 kg in the complicated group and 2.8 ± 0.52 kg in the uncomplicated group. NICU admission was required in 10% of the complicated group and 8.2% of the uncomplicated group .[81] Similar trends have been reported in previous studies published by other authors.

PLGF LEVELS IT'S ASSOCIATION WITH PREECLAMPSIA WITH AND WITHOUT SEVERE FEATURES AND ECLAMPSIA (Table 19 and 26)

In this study, Out of 94 pregnancies, 59 cases (62.8%) were classified as having abnormal PlGF levels, while 35 cases (37.2%) had normal PlGF levels. Among cases with abnormal PlGF levels, the majority (37 cases; 62.7%) had severe preeclampsia, while 9 cases (15.3%) progressed to eclampsia, highlighting a strong correlation between low PlGF levels and the severity of disease. In contrast, only 13 cases (22.0%) with abnormal PlGF levels had preeclampsia without severe features. On the other hand, among cases with normal PlGF levels, 31 cases (88.6%) had preeclampsia without severe features, while only 4 cases (11.4%) developed severe preeclampsia, and none progressed to eclampsia. The Chi-square value of 39.36 and the highly significant p-value of 0.001 confirm a strong and statistically significant association between low PlGF levels and disease severity.

Also among cases of preeclampsia without severe features, the majority (31 cases; 88.6%) had PlGF levels above 100 pg/mL. 12 cases (44.4%) had PlGF levels between 51–100 pg/mL, while only 1 case (3.2%) had PlGF levels between 12–50 pg/mL. Among cases of severe preeclampsia, the largest proportion of cases (25 cases; 80.6%) had PlGF levels between 12–50 pg/mL. Only 4 cases (11.4%) with severe preeclampsia had PlGF levels above 100 pg/mL. Among cases of eclampsia, 1 case (100%) had PlGF levels below 12 pg/mL. Additionally, 5 cases (16.1%) of eclampsia had PlGF levels between 12–50 pg/mL, and 3 cases (11.1%) had levels between 51–100 pg/mL. The Chi-square value of 57.96 and the highly significant p-value of 0.001 confirm a statistically significant association between low PlGF levels and the severity of preeclampsia.

Benton et al. reported that low PlGF levels were significantly associated with severe preeclampsia and eclampsia. Severe preeclampsia was diagnosed in 8.5% of low PlGF cases, while no cases were reported in the normal PlGF group ($p < 0.001$).[71] Sibiude et al. reported that the odds of developing severe preeclampsia were significantly higher in women with low PlGF levels (<12 pg/mL) (OR = 216, 95% CI: 18–2571). The predictive value of PlGF for severe preeclampsia was high ($p < 0.001$) .[19] Chappell et al. reported that low PlGF levels were significantly associated with the development of severe preeclampsia. Sensitivity for predicting preeclampsia within 14 days of testing was 96% for PlGF <5th centile and 63% for PlGF <12 pg/mL.[50] Villalaín et al. (2020) reported that an sFlt-1/PlGF ratio >655 was almost always associated with preeclampsia or fetal growth restriction. Severe features of preeclampsia were present in 77.3% of cases with preeclampsia at delivery.[82] These results are strongly supported by existing literature.

PLGF WITH FGR (Table 21)

Out of 94 pregnancies, 36 cases (38.3%) were complicated by FGR, while 58 cases (61.7%) had normal fetal growth. Among the pregnancies with abnormal PlGF levels, 34 cases (57.6%) were associated with FGR. Only 2 cases (5.7%) with normal PlGF levels were complicated by FGR. Among cases without FGR, 33 cases (94.3%) had normal PlGF levels. The Chi-square value of 25.05 and the highly significant p-value of 0.001.

Benton et al. showed that low PlGF levels had a sensitivity of 98.2% and specificity of 75.1% for identifying FGR, confirming that low PlGF is a strong marker for placental insufficiency leading to FGR.[71] Sibiude et al. showed that low PlGF levels were significantly associated with FGR, with a sensitivity of 91% for detecting FGR ($p < 0.001$).[19] Chappell et al. showed that low PlGF levels were significantly associated with FGR, with a sensitivity of 93% for predicting small for gestational age (SGA) infants born below the 1st centile.[50] Villalaín et al. (2020) found that FGR was present in 84.9% of cases with preeclampsia and 90.4% of cases without preeclampsia at delivery. Severe FGR (stage III or IV) was more frequent in the preeclampsia group .[82] Sung et al. (2017) showed that low PlGF levels were strongly associated with FGR (OR = 0.143; 95% CI: 0.025–0.806; p = 0.027).[84] Verma et al. (2022) found that low PlGF levels were significantly associated with FGR.[81] This finding is consistent with previous studies found in the literature.

PLGF LEVEL AND ITS ASSOCIATION WITH MATERNAL COMPLICATIONS (Table 23)

In this study, Renal failure was observed in 1 case (1.1%) and was linked to abnormal PlGF levels, though the association was not statistically significant (p = 0.62).

Postpartum hemorrhage occurred in 2 cases (2.1%), one in the abnormal PlGF group and one in the normal PlGF group, with no significant difference between the two groups ($p = 0.60$). HELLP syndrome was reported in 11 cases (11.7%), with 7 cases (11.9%) in the abnormal PlGF group and 4 cases (11.4%) in the normal PlGF group, indicating no significant difference between groups ($p = 0.61$). Notably, there were no reported cases of disseminated intravascular coagulation (DIC), cerebrovascular accident, or respiratory distress in either group.

Benton et al. reported that placental abruption was significantly higher in the low PlGF group (16.1%) compared to the normal PlGF group (0%) ($p < 0.001$). HELLP syndrome was also more common in the low PlGF group.[71] Sibiude et al. found that low PlGF levels were associated with a higher incidence of maternal complications such as HELLP syndrome and placental abruption ($p < 0.001$).[19] Chappell et al. found that low PlGF levels were associated with a higher incidence of maternal complications, including placental abruption and HELLP syndrome.[50] Villalaín et al. (2020) found that HELLP syndrome, abruptio placentae, and severe hypertension were more common in cases with sFlt-1/PlGF >655. Severe hypertension was present in 19.5% of cases with preeclampsia and 5.8% without preeclampsia ($p = 0.02$).[82] Verma et al. (2022) reported that low PlGF levels were associated with increased maternal complications, including postpartum acute renal failure and HELLP syndrome.[84]

In contrast, several previous studies found a stronger link between low PlGF levels and maternal complications. Benton et al. reported significantly higher rates of placental abruption and HELLP syndrome in women with low PlGF ($p < 0.001$) [71]. Sibiude et al. also found a significant association between low PlGF and HELLP syndrome/abruption ($p < 0.001$) [19]. Chappell et al. and Villalaín et al. (2020) similarly

noted increased rates of placental abruption, severe hypertension, and HELLP syndrome in pregnancies with low PlGF or high sFlt-1/PlGF ratios. Verma et al. (2022) observed maternal complications such as postpartum renal failure and HELLP syndrome associated with low PlGF. This finding is consistent with previous studies found in the literature.

PLGF LEVEL AND ITS ASSOCIATION FETAL OUTCOMES(NICU ADMISSIONS, BIRTH WEIGHT) (Table 24 and 27)

In this study, Among cases with abnormal PlGF levels, 11 cases (18.6%) had extremely low birth weight (ELBW) (<1.0 kg), while none of the cases with normal PlGF levels were in this category. Similarly, 13 cases (22.0%) with abnormal PlGF levels had very low birth weight (VLBW) (<1.5 kg) compared to 4 cases (11.4%) with normal PlGF levels. Low birth weight (LBW) (<2.5 kg) was reported in 22 cases (37.3%) with abnormal PlGF levels, compared to 7 cases (20.0%) with normal PlGF levels. In contrast, normal birth weight (≥2.5 kg) was more common among cases with normal PlGF levels (24 cases; 68.6%) compared to those with abnormal PlGF levels (13 cases; 22.0%). The Chi-square value of 22.10 and the highly significant p-value of 0.001 confirm a statistically significant association between low PlGF levels and poor fetal growth. Among cases with abnormal PlGF levels, 32 cases (54.2%) required NICU admission, compared to only 7 cases (20.0%) with normal PlGF levels. In contrast, most newborns with normal PlGF levels (28 cases; 80.0%) did not require NICU admission, compared to 27 cases (45.8%) with abnormal PlGF levels. The Chi-square value of 10.60 and the highly significant p-value of 0.001 confirm a statistically significant association between low PlGF levels and increased NICU admissions. Also among newborns requiring NICU admission (39 cases; 41.5%), the majority (23 cases; 74.2%) had PlGF levels between 12–50 pg/mL, and 8 cases

(29.6%) had levels between 51–100 pg/mL. Notably, the only case with PlGF levels below 12 pg/mL resulted in NICU admission. Only 7 cases (20.0%) with PlGF levels above 100 pg/mL required NICU admission. Among cases not requiring NICU admission (55 cases; 58.5%), the majority (28 cases; 80.0%) had PlGF levels above 100 pg/mL, and 19 cases (70.4%) had levels between 51–100 pg/mL. Only 8 cases (25.8%) with PlGF levels between 12–50 pg/mL avoided NICU admission. The Chi-square value of 23.29 and the highly significant p-value of 0.001 confirm a statistically significant association between low PlGF levels and the need for NICU admission.

Also among newborns requiring NICU admission (39 cases; 41.5%), the majority (23 cases; 74.2%) had PlGF levels between 12–50 pg/mL, and 8 cases (29.6%) had levels between 51–100 pg/mL. Notably, the only case with PlGF levels below 12 pg/mL resulted in NICU admission. Only 7 cases (20.0%) with PlGF levels above 100 pg/mL required NICU admission. Among cases not requiring NICU admission (55 cases; 58.5%), the majority (28 cases; 80.0%) had PlGF levels above 100 pg/mL, and 19 cases (70.4%) had levels between 51–100 pg/mL. Only 8 cases (25.8%) with PlGF levels between 12–50 pg/mL avoided NICU admission. The Chi-square value of 23.29 and the highly significant p-value of 0.001 confirm a statistically significant association between low PlGF levels and the need for NICU admission.

Benton et al. (2016) reported that NICU admission was required in 27.7% of newborns from the low PlGF group compared to 10.1% in the normal PlGF group. Low birth weight was significantly more common in the low PlGF group ($p < 0.001$) .[71] Sibiude et al. reported that the mean birth weight was significantly lower in the low PlGF group. NICU admissions were more common among newborns from the low PlGF group (56.7%) compared to the normal PlGF group ($p < 0.001$).[19] Chappell et al.

found that low PlGF levels were associated with increased rates of NICU admissions and low birth weights. Sensitivity for predicting adverse fetal outcomes was 96% for PlGF <5th percentile.[50] Villalaín et al. (2020) reported that NICU admission was required in 83.2% of cases with preeclampsia and 75% of cases without preeclampsia. Birth weight was significantly lower in cases with high sFlt-1/PlGF ratios ($p < 0.01$) .[82] Verma et al. (2022) found that low PlGF levels were associated with lower birth weight and increased NICU admissions.[81] These findings are in alignment with studies conducted earlier.

PE WITH AND WITHOUT SEVERE FEATURES OF ECLAMPSIA AND MATERNAL COMPLICATIONS (Table 28)

In this study placental abruption was reported in 6 cases (6.4%), with 1 case (100%) occurring in the group with PlGF levels <12 pg/mL and 5 cases (16.1%) in the 12–50 pg/mL group. No cases of abruption were reported in the higher PlGF categories, indicating a significant association between low PlGF levels and placental abruption ($p = 0.001$). Renal failure was reported in only 1 case (1.1%), which occurred in the 12–50 pg/mL group ($p = 0.56$, not statistically significant). Postpartum hemorrhage (PPH) occurred in 2 cases (2.1%), one case in the 12–50 pg/mL group and one case in the >100 pg/mL group, with no significant association ($p = 0.83$). HELLP syndrome was reported in 11 cases (11.7%) and was fairly evenly distributed across all PlGF categories, with 4 cases (12.9%) in the 12–50 pg/mL group, 3 cases (11.1%) in the 51–100 pg/mL group, and 4 cases (11.4%) in the >100 pg/mL group ($p = 0.98$, not statistically significant).

Benton et al. (2016) showed that severe preeclampsia was associated with higher rates of placental abruption (16.1% in the low PlGF group), HELLP syndrome, and low birth weight compared to mild preeclampsia.[71] Sibiude et al. (2012) found that severe

maternal complications such as HELLP syndrome and placental abruption were more frequent among cases of severe preeclampsia with low PlGF levels ($p < 0.001$).[19] Villalaín et al. (2020) found that maternal complications such as abruptio placentae and HELLP syndrome were more common in cases with severe preeclampsia and high sFlt-1/PlGF ratios.[82] Chappell et al. (2013) reported that severe maternal complications such as HELLP syndrome and placental abruption were more common in cases of severe preeclampsia with low PlGF levels.[50] These results are comparable to findings in previous literature.

CONCLUSION

The study demonstrated a significant association between low serum placental growth factor (PlGF) levels and adverse maternal and fetal outcomes in pregnancies complicated by preeclampsia and fetal growth restriction (FGR). Low PlGF levels were strongly linked with the severity of preeclampsia, with a higher incidence of severe preeclampsia and progression to eclampsia in cases with abnormal PlGF levels. Abnormal PlGF levels were also significantly associated with fetal growth restriction, low birth weight, and increased rates of NICU admissions. Furthermore, maternal complications such as placental abruption and HELLP syndrome were more frequent in cases with low PlGF levels. These findings suggest that measuring PlGF levels can serve as a valuable early marker for identifying high-risk pregnancies and guiding timely intervention to improve maternal and neonatal outcomes. Our study has a limited sample size could have added more strength to the study.

LIMITATIONS

Limitations of the present study is its relatively small sample size, a larger sample size could have added to the strength of the study.

Secondly the PlGF estimation was done at one point only that is at the time of admission to the labor ward.

PlGF estimation done in the first trimester around 11- 13wks helps in the early prediction of preeclampsia and its adverse outcomes. A positive test and early prediction of the condition and helps the clinician to intervene at the appropriate time thereby reducing both maternal and fetal adverse outcomes. Our study was a cross sectional one and we have studied only one bio marker.

SUMMARY

The study included 94 pregnant women and examined the association between serum placental growth factor (PlGF) levels and maternal and fetal outcomes in cases of preeclampsia and fetal growth restriction (FGR). The majority of participants (44.7%) were aged 21–25 years, while 60.6% were primigravida. Most deliveries occurred in the late preterm period (34 to <37 weeks), accounting for 37.2% of cases. The PlGF were abnormal in 59 cases (62.8%) and normal in 35 cases (37.2%). Fetal growth restriction was identified in 38.3% of cases, and 27.7% of cases had abnormal Doppler findings.

In terms of preeclampsia severity, 46.8% of cases were classified as preeclampsia without severe features, 43.6% had severe preeclampsia, and 9.6% progressed to eclampsia. PlGF levels were classified as abnormal in 62.8% of cases, which showed a statistically significant association with preeclampsia severity ($p = 0.001$). Among cases with abnormal PlGF levels, 62.7% had severe preeclampsia, and 15.3% progressed to eclampsia. Abnormal PlGF levels were also significantly associated with FGR (57.6% of cases with FGR had abnormal PlGF levels, $p = 0.001$). According to the NICE PlGF classification there was one case of PlGF level <12 pg/mL which was a case of eclampsia and 31 cases were in the range of 12-50 pg/mL and 27 cases were in the range of 51-100 pg/mL and 35 cases were with >100 pg/mL.

The study reported significant differences in birth weight based on PlGF levels. Extremely low birth weight (ELBW) was observed in 18.6% of cases with abnormal PlGF levels, compared to none in the normal PlGF group ($p = 0.001$). Low APGAR scores at 1 minute and 5 minutes were more common in cases with abnormal PlGF levels ($p = 0.02$). NICU admission was required in 54.2% of cases with abnormal

PlGF levels compared to 20.0% of cases with normal PlGF levels (p = 0.001). Out of the 90 cases (95.7%) were live births with high rates of low birth weight newborns.

Maternal complications were also linked to abnormal PlGF levels. Placental abruption occurred in 10.2% of cases with abnormal PlGF levels, compared to none in the normal PlGF group (p = 0.001). HELLP syndrome was reported in 11.9% of cases with abnormal PlGF levels but was not significantly different from cases with normal PlGF levels (p = 0.98). Postpartum hemorrhage occurred in 1.7% of cases with abnormal PlGF levels and 2.9% of cases with normal PlGF levels (p = 0.83). The association between low PlGF levels and severe maternal complications suggests that PlGF could serve as an early marker for pregnancy-related complications.

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ANNEXURE – I - INFORMED CONSENT FORM

"ASSOCIATION OF SERUM PLACENTAL GROWTH FACTOR IN PRE-ECLAMPSIA AND FGR WITH MATERNAL AND PERINATAL OUTCOMES-A ONE YEAR CROSS SECTIONAL STUDY"

Introduction: Preeclampsia is one of the most dangerous disorder related to pregnancy which affects 2%-8% of all pregnancies and is also associated with adverse maternal and new born outcomes. Even in the present times our capacity to predict maternal and perinatal outcomes remains poor. Many of the diagnostic test have shown various degrees of predictive accuracy. In this study the role of angiogenic biochemical markers placental growth factor(PLGF)

Explanation of procedure: In this study 2ml of venous blood sample will be taken for the testing a parameter of blood (PlGF) which may help us in predicting the maternal and perinatal outcomes subsequently. You will be followed till delivery and discharge to know the possible outcomes of you and the baby.

Withdrawal from participation in the study: Participation in this study in voluntary. You will be free to decide whether to participate in this study or continue participation once enrolled. In case you decide to withdraw your participation, you are free to do so. However, please convey the decision to the principal investigator.

Possible benefits from participating in the study: You will be benefitted by participating in this study. The data gathered will also help population at large.

Possible risks from participating in the study: There are no risks involved in participating in this study.

Privacy and confidentiality: The information collected from you will be coded, to prevent any person to identify you. Your identity will never be revealed. The data

collected from you will be kept confidential and only processed or aggregated data will be used for publication.

Financial incentives: You will not receive any payment for participating in this study.

Cost of investigations done during the course of study will be paid by the **principal investigator.**

Authorization for publication of aggregated data: Results obtained after processing of the aggregated data will be published for scientific purpose and or presented to scientific groups. However, your identity will never be revealed.

Questions: In case of any questions with regard to this study, you are free to contact: Dr Harsha Hegde, Chairperson, Ethical committee of JNMC, 0831-2473777 Extension 4052.

Legal rights: By signing this consent form, we are not waving any of your legal rights

CONSENT STATEMENT

I am making a voluntary decision to participate in the study "Association of serum placental growth factor in pre-eclampsia and FGR with maternal and perinatal outcomes-A one-year cross sectional study". My signature below indicates that I have decided to participate and I have read the information provided above or the information provided above has been read to me in the language that I understand best. I was given the opportunity to ask questions and that they have been answered to my satisfaction.

Name of the participant:

Signature or left thumb impression of the participant:

Name of the witness:

Signature or left thumb impression of the witness:

Name of the investigator:

Signature of the investigator:

ANNEXURE – II - SCREENING FORM

Screening number: ☐☐☐☐ IP number: ☐☐☐☐☐☐

Date of screening (dd-mm-yyyy): ☐☐ ☐☐ ☐☐☐☐

First name: ☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐

Middle name: ☐☐☐☐☐☐☐

Last name: ☐☐☐☐☐☐☐☐☐

Age (years): ☐☐

Address: H.no. - ☐☐☐

Street - ☐☐☐☐☐☐☐☐☐☐☐☐

Taluka - ☐☐☐☐☐☐☐☐☐☐

District - ☐☐☐☐☐☐☐

Phone number ☐☐☐☐☐☐☐☐☐☐

Registered	
Unregistered	

Eligibility: YES –1 , NO – 2

Singleton pregnancy ☐

> 20 weeks gestation ☐

Patient with USG (<20weeks) ☐

Preeclampsia(BP and proteinuria) ☐

Is she eligible? If eligible, consent to be taken. ☐

Consent:

Does the woman agree to participate? ☐

Enrollment done:

Investigator's name :

Signature :

ANNEXURE: III PROFORMA

"Association of serum placental growth factor in pre-eclampsia and FGR with maternal and perinatal outcomes-A one-year cross sectional study"

Screening Id: ☐☐☐☐ Enrollment number: ☐☐☐

First name: ☐☐☐☐☐☐☐☐

Middle name: ☐☐☐☐☐☐☐

Last name: ☐☐☐☐☐☐☐☐☐

Age (years): ☐☐

Date of 1ST visit (dd-mm-yyyy): ☐☐ ☐☐ ☐☐☐☐

COMPLAINTS AND HISTORY OF PRESENTING COMPLAINT: (Yes-1, No-2)

Period of amenorrhea (in months) ☐

Duration of pain abdomen(days/hours) ☐

Duration of leak per vagina(hours) ☐

Duration of bleeding per vagina(hours) ☐

Perception of fetal movements (Yes/No) ☐

Any history of the following at the time of admission-

(YES – 1, NO – 2)

Headache ☐

Blurring of vision ☐

Persistent severe Epigastric pain ☐

Seizures ☐

Antenatal history:

a. Regular Antenatal visits ☐

Obstetric history:

Married Life (years) : ☐☐

Consanguinity: ☐ (YES - 1, NO - 2)

If yes,

Degree of consanguinity: ☐☐☐☐☐☐

Obstetric score: _____

Contraceptive use: ☐ (YES - 1, NO - 2)

Menstrual history:

Menarche (age in years): ☐☐

Last menstrual period (dd-mm-yyyy): ☐☐ ☐☐ ☐☐☐☐

Expected date of delivery (dd-mm-yyyy): ☐☐ ☐☐ ☐☐☐☐

Period of gestation (weeks/ days): ☐☐ ☐☐

Past History :

YES – 1 , NO – 2

a. Known case of Diabetes mellitus : ☐

   If yes, Duration (in years) : ☐☐

   Treatment received : ☐☐☐☐☐☐☐

b. Known case of Hypertension : ☐

   If yes, Duration (in years) : ☐☐

   Treatment received : ☐☐☐☐☐☐☐

c. Known case of Cardiac disorder : ☐

   If yes, Duration (in years) : ☐☐

   Treatment received : ☐☐☐☐☐☐☐

d. Known case of Hypothyroidism. : ☐

If yes, Duration (in years) :

Treatment received : ☐☐☐☐☐☐☐☐☐

e. H/O any surgery in past : ☐

Personal History :

YES – 1 , NO – 2

Bowel and bladder normal ☐

General physical examination-

Height (in centimeters) ☐☐☐

Weight (in kilogram) ☐☐☐

BMI ☐☐☐

YES – 1, NO – 2

Pallor ☐

Icterus ☐

Pedal Oedema ☐

Blood pressure (mmHg) ☐☐☐ / ☐☐

Pulse rate (beats per minute) ☐☐☐

Systemic examination :

Per Abdomen:

Size of uterus(weeks) ☐ ☐

Presentation_____

Fetal heart sound(bpm) ☐ ☐☐

Cardiovascular: _____

Respiratory : _____

CNS : _____

Investigations-

Date (dd-mm-yyyy):

Blood Group:

Haemoglobin (g/dl): ..

Platelets

HIV: (Non- reactive – 1, Reactive – 2)

HbsAg: (Non- reactive – 1, Reactive – 2)

VDRL: (Non- reactive – 1, Reactive – 2)

Urine routine and microscopy-

Proteinuria

<u>Liver function Test-</u>

Total Bilirubin

SGOT

SGPT

Serum LDH

<u>Renal function Test-</u>

Uric acid

Urea

Serum Creatinine

Placental growth factor (Yes-1,No-2)

Result

Obstetric Scan report-

NORMAL(1) ☐

ABNORMAL(2)

Doppler –

NORMAL(1)- ☐

ABNORMAL(2)

DELIVERY OUTCOME -

Mode of delivery -

Vaginal delivery ☐

C section ☐

Indication of C- section ☐☐☐☐☐

DIAGNOSIS

Maternal outcomes-

Peripartum Complications:

1) PPH
2) DIC
3) HELLP syndrome
4) Abruption ☐
5) Sepsis
6) Cerebrovascular accident
7) Respiratory Distress/Pulmonary complication.
8) Acute kidney injury
9) Maternal death
10) Any other.

ICU admission (Yes-1/No-2) ☐

Duration of stay ☐☐

Condition at discharge_____

FETAL OUTCOME-

(Live Birth-1,Still Birth-2,IUFD-3, ☐

Perinatal Death-4,)

Birth weight- ☐

APGAR

At 1 mins ☐/10

At 5 mins ☐/10

NICU admission(yes-1/no-2) ☐

ANY OTHER FETAL/MATERNAL COMPLICATIONS

Please Specify _____

ANNEXURE – IV-

MASTER CHART

NAME	AGE	DATE OF FIRST VISIT	IP NUMBER	REGISTERED(1) /UNREGIST(2)	EDD	LMP	POG	FGR(Y/N)	ABN DOPPLER(Y/N)	HEADACHE	BLURRING OF VISION	EPIGASTRIC PAIN	SEIZURE	OLIGOURIA	ML(YRS)	OBS SCORE	CONSANGUINITY	HD	HYPOTHYROIDISM	HTN	DM	HT	WT	BMI	PALLOR	ICTERUS	OEDEMA	BP	NON SEVERE(1)/SEVERE(2)	PR	UT SIZE	PRESENT	FHR	TENDER	BLD GRP	HB	PLT	URINE ALB	TB	SGOT	SGPT	LDH	CREAT	U.A	PE WITHOUT SEVERE FEATURES(1)/PE WITH SEVERE(2)/ECLAMPSIA(3)	PIGF	VD	LSCS	INDICATION	DIC	ABRUPTION	CVA	RD	RF	MM	PPH	HELLP	ICU	LB	SB	BW	APGAR(1)	APGAR(5)	NICU
NSP	30	30-09-2024	10096101	2	UNKNOWN	UNKNOWN	35+0	1	1	1	2	2	2	2	12	PRIMI	2	2	2	2	2	165	68	25.08542388	1	2	1	162/110	2	132	36	C	158	2	O+	12.4	78K	2	1	33	26	352	0.9	6.2	2	52	Y	N	SEVERE PE	2	2	2	2	2	2	2	2	2	1	2	2.1	7	8	2
JQM	33	05-10-2024	10095635	1	07-11-2024	25-01-2024	35+0	2	2	2	2	2	2	2	16	G4P3L3	2	2	2	2	1	165	62	30.17559834	1	2	1	150/96	1	74	34	C	130	2	O+	11.1	98K	1	0.6	20	22	180	0.7	4.2	1	133	N	Y	PE WITH FOETAL	2	2	2	2	2	2	2	2	2	1	2	2.7	8	9	2
NM	23	07-10-2024	10096718	1	26-10-2024	24-01-2024	37+2	1	2	2	2	2	2	2	1	PRIMI	2	2	2	2	1	160	64	27.4609375	1	2	1	166/100	2	88	32	C	130	2	O+	12.4	2.2L	2	0.8	20	22	360	0.8	4	2	47.5	Y	N	SEVERE PE	2	2	2	2	2	2	2	2	2	1	2	1.4	6	7	1
MAM	24	23-09-2024	10093265	1	29-02-2024	23-02-2024	30+3	1	1	2	2	2	2	2	4	G3P2L2	2	2	2	2	2	156	92	12.95699433	2	2	1	168/110	2	102	28	UNSTABLE	142	2	O+	10.1	80K	2	1.3	34	34	280	0.8	6	2	33.8	N	Y	SEVERE PE WITH FGR	2	2	2	2	2	2	2	2	2	1	2	1.2	6	8	1
SJK	27	04-10-2024	10095945	2	16-11-2024	23-02-2024	33+6	1	1	1	1	2	2	2	3	PRIMI	2	2	2	2	2	150	64	25.74462891	2	2	1	176/102	2	91	30	C	130	2	O+	11.5	1.2L	NIL	0.76	16	16	220	0.8	5.4	2	92.6	N	Y	SEVERE PE WITH FGR	2	2	2	2	2	2	2	2	2	1	2	1.4	8	9	1
SVD	23	04-04-2024	10051683	2	31-05-2024	25-08-2023	37+6	1	2	2	2	2	2	2	2.5	PRIMI	2	2	1	2	2	154	66	24.85353535	1	2	1	150/94	1	86	36	C	166	2	B +	9.3	2L	2	0.64	20	24	300	0.8	26	1	79.3	N	Y	PE with FGR	2	2	2	2	2	2	2	2	2	1	2	2.8	7	9	2
LAK	24	20-04-2024	10055206	1	08-06-2024	02-09-2023	33+2	1	1	1	2	1	2	2	1	PRIMI	2	2	2	2	2	148	52	38.47781065	1	2	1	164/100	2	100	30	C	168	2	B +	12.2	48K	3	0.92	30	32	320	1	8.2	2	56.1	N	Y	SEVERE PE WITH HELLP	2	2	2	2	2	2	2	1	2	1	2	1.2	6	7	1
MGM	30	22-04-2024	10055444	1	NIL	NIL	NIL	1	1	1	1	1	1	2	1.5	PRIMI	2	2	2	2	2	174	88	15.79571281	1	2	1	164/106	2	110	36	C	158	2	A +	11.6	1.86L	2	0.8	28	26	380	0.8	7.8	3	75	N	Y	ECLAMPSIA	2	2	2	2	2	2	2	2	2	1	2	2.3	8	9	2
PSK	25	24-04-2024	10056023	1	13-04-2024	06-07-2023	37+0	2	2	2	2	2	2	2	2	PRIMI	2	2	2	2	2	150	60	29.29166667	1	2	1	154/100	1	102	36	C	148	2	A +	11.8	1.4L	3	0.9	30	24	250	1.1	7	1	121	N	Y	PE WITH FOETAL DISTRESS	2	2	2	2	2	2	2	2	2	1	2	3.1	8	9	2
SAP	34	10-04-2024	10052932	1	24-03-2024	18-08-2023	33+5	1	1	1	1	2	1	2	6	G3P1L1A1	2	2	2	2	2	148	50	41.6176	1	2	1	160/120	2	120	32	C	134	2	AB -	14	1.7L	4	0.9	24	32	320	0.9	8.2	3	23.3	Y	N	ECLAMPSIA	2	2	2	2	2	2	2	2	2	1	2	1.48	6	7	1
GAP	26	10-04-2024	10052887	2	07-05-2024	01-08-2023	36+1	2	2	2	2	2	2	2	4	G2A1	2	2	2	2	2	154	56	34.52232143	1	2	2	140/90	1	90	36	C	152	2	A +	12.5	1.6L	1	0.6	22	20	260	0.6	4.2	1	182	N	Y	PE WITH FOETAL DISTRESS	2	2	2	2	2	2	2	2	2	1	2	2.7	7	9	2
ADH	31	22-04-2024	10056673	1	22-05-2024	15-08-2023	34+2	1	1	2	2	2	2	2	7	G3P2L2	2	2	2	2	2	144	48	43.9375	1	2	1	170/104	2	92	34	C	152	2	B+	10.5	1.5L	4	0.9	32	20	302	0.9	6.4	2	24	N	Y	SEVERE PE WITH ABNORMAL DOPPLER	2	2	2	2	2	2	2	2	2	1	2	1.8	8	9	1
PSG	31	03-05-2024	10058206	2	19-05-2024	12-08-2023	37+6	1	1	2	2	2	2	2	12	G5P4L4	2	2	2	2	2	150	52	38.99778107	1	2	1	162/100	2	88	38	C	142	2	A+	12.5	60K	1	0.3	32	34	300	0.8	4	2	18.4	N	Y	SEVERE PE WITH HELLP	2	2	2	2	2	2	2	1	2	1	2	2.3	7	8	2
PSS	22	09-05-2024	10059465	2	25-05-2024	19-08-2023	37+5	1	1	1	2	2	2	2	2	PRIMI	2	2	2	2	2	152	54	36.64471879	1	2	2	166/102	2	86	36	C	158	2	AB+	11.8	60K	2	0.8	24	28	420	0.8	5.2	2	42	N	Y	SEVERE PE WITH HELLP	2	2	2	2	2	2	2	1	2	1	2	1.8	7	9	2
PSG	21	16-05-2024	10060934	2	29-06-2024	15-09-2023	33+4	1	2	2	2	2	2	2	1.5	PRIMI	2	2	1	2	2	146	50	41.0552	1	2	1	160/110	2	110	34	C	168	2	B+	10	98K	2	1	24	20	360	0.8	4.2	2	34	N	Y	ABRUPTIO PLACENTA	2	1	2	2	2	2	2	2	2	1	2	1.5	8	9	1
SPV	23	26-06-2024	10079019	2	03-09-2024	26-11-2023	34+3	1	1	1	1	2	1	2	1	PRIMI	2	2	2	2	2	148	58	30.92865636	1	2	1	162/100	2	92	32	C	136	2	O+	12	1.2L	2	0.8	34	36	426	1.1	6	3	36	N	Y	APH WITH ECLAMPSIA	2	1	2	2	2	2	2	2	1	1	2	2.1	6	7	1
PNN	25	24-06-2024	10070944	1	03-08-2024	10-11-2023	32+2	1	1	1	2	2	2	2	7	G2P1L1	2	2	1	2	2	142	51	38.3798539	1	2	1	160/112	2	92	30	C	158	2	A+	13.3	1.2L	2	0.6	36	34	200	1.1	4.2	2	30.3	N	Y	SEVERE PE WITH ABNORMAL DOPPLER	2	2	2	2	2	2	2	2	2	1	2	1.3	5	7	1
AAP	30	21-06-2024	10070315	1	23-07-2024	18-10-2023	35+3	1	1	1	1	2	1	2	2	G2P1L1	2	2	2	2	2	164	72	22.23996914	1	2	1	164/110	2	110	34	C	142	2	B+	12.1	1.2L	3	1	36	38	320	0.8	5.8	3	24	N	Y	ECLAMPSIA	2	2	2	2	2	2	2	2	1	1	2	1.8	2	7	1
PJM	38	05-07-2024	10073977	2	31-08-2024	25-11-2023	31+6	2	2	1	2	2	2	2	5	G2P1L1	2	2	1	2	2	154	72	20.88387346	1	2	2	142/96	1	96	30	C	160	2	A+	11.6	1.6L	3	0.9	32	34	356	1	4.2	1	152.5	N	Y	PE WITH FGR WITH ABNORMAL DOPPLER	2	2	2	2	2	2	2	2	2	1	2	2.6	5	7	1
RDK	26	27-07-2024	10079399	2	12-10-2024	06-01-2024	27+0	1	1	1	1	2	2	2	1	PRIMI	1	2	2	2	2	142	50	39.9304	1	2	1	164/112	2	50	24	UNSTABLE	164	2	AB+	12.2	1.8L	2	1.2	38	34	280	1	4.2	2	37.4	Y	N	SEVERE PE WITH AEDF	2	2	2	2	2	2	2	2	2	2	1	0.615	6	7	2
MNO	30	07-08-2024	10082117	1	09-08-2024	06-11-2023	39+5	1	2	2	2	2	2	2	2	PRIMI	2	2	1	2	2	158	60	30.85388889	1	2	1	150/96	1	60	36	B	142	2	O+	11.3	1.6L	1	0.9	32	28	120	0.8	3.2	1	50	N	Y	PE WITH BREECH	2	2	2	2	2	2	2	2	2	1	2	2.2	8	10	2
CPM	24	20-08-2024	10081985	1	20-08-2024	22-11-2023	37+0	2	2	2	2	2	2	2	2	PRIMI	2	2	2	2	2	154	57	33.32163743	1	2	1	154/96	1	88	36	C	150	2	A+	12.6	2.3L	1	0.8	30	32	350	0.9	4	1	119	N	Y	PE WITH FOETAL DISTRESS	2	2	2	2	2	2	2	2	2	1	2	2.5	7	8	2
MVP	32	03-08-2024	10081111	1	17-10-2024	12-01-2024	31+5	1	2	1	2	2	2	2	3	PRIMI	2	2	2	2	1	144	48	43.9375	1	2	1	170/110	2	106	28	C	130	2	0+	12.5	2.1L	2	0.8	28	35	400	1	4	2	50	N	Y	SEVERE PE WITH FGR	2	2	2	2	2	2	2	2	2	1	2	1.1	6	7	1
AMT	24	16-09-2024	10091405	1	19-11-2024	13-02-2024	30+6	1	1	2	2	2	2	2	1	PRIMI	2	2	2	2	2	148	50	41.6176	1	2	1	160/100	2	88	26	C	152	2	A+	12.1	1.6L	2	0.7	28	32	320	0.9	4.1	2	76	N	Y	SEVERE PE WITH FGR WITH ABNORMAL DOPPLER	2	2	2	2	2	2	2	2	2	1	2	1.4	6	7	1
SAP	25	26-09-2024	10091640	1	08-10-2024	18-12-2023	38+5	2	2	2	2	2	2	2	2	G3P1L1A1	2	2	2	2	2	146	50	41.0552	1	2	1	150/96	1	92	26	C	146	2	A+	10.2	1.8L	2	0.9	30	32	320	0.5	4.2	1	103	N	Y	PREV LSCS WITH PE	2	2	2	2	2	2	2	2	2	1	2	2.3	7	9	2
SMG	24	27-09-2024	10093615	1	12-10-2024	06-01-2024	37+5	2	2	2	2	2	2	2	2	PRIMI	2	2	2	2	2	156	52	40.55769231	2	2	1	148/98	1	90	36	C	140	2	0+	12.8	2.18L	1	0.6	20	18	220	0.8	6.8	1	69.7	N	Y	PE WITH FOETAL DISTRESS	2	2	2	2	2	2	2	2	2	1	2	2.6	8	9	2
AAG	30	24-09-2024	10093562	1	05-11-2024	30-01-2024	34+2	1	2	2	2	2	2	2	7	G4P1L1A2	2	2	2	2	2	151	58	31.55558859	2	2	1	168/100	2	102	34	C	146	2	O+	11.4	1.8L	2	0.9	18	20	300	0.8	3	2	74.5	N	Y	SEVERE PE WITH PPROM	2	2	2	2	2	2	2	2	2	1	2	2.2	6	8	1
DRT	22	27-09-2024	10094282	1	07-12-2024	02-03-2024	29+6	1	1	2	2	2	2	2	1	G2A1	2	2	2	2	2	146	48	44.54774306	1	2	1	170/110	2	102	28	B	122	2	A+	12.7	1.6L	1	0.8	28	20	300	1	6	2	29.3	N	Y	SEVERE PE WITH ABNORMAL DOPPLER	2	2	2	2	2	2	2	2	2	1	2	1	6	8	1
JBM	26	27-09-2024	10094301	2	02-10-2024	27-12-2023	36+1	2	2	1	2	2	2	2	2	PRIMI	2	2	2	2	2	150	52	38.99778107	2	2	1	158/102	1	96	34	C	140	2	A+	10.7	2L	2	0.8	18	21	280	1	7.2	1	405	N	Y	PE WITH FOETAL DISTRESS	2	2	2	2	2	2	2	2	2	1	2	2.4	8	9	2
GSB	27	28-09-2024	10094595	2	05-11-2024	30-01-2024	34+6	1	2	1	1	2	1	2	2	PRIMI	2	2	1	2	2	140	44	50.83677686	1	2	1	194/116	2	104	30	C	132	2	O+	10.9	1.8L	1	1.1	16	18	280	1	6.4	3	57.6	N	Y	ECLAMPSIA	2	2	2	2	2	2	2	2	2	1	2	1.8	7	9	2
AJM	21	03-10-2024	10095422	2	26-10-2024	08-01-2024	36+5	2	2	2	1	2	2	2	1	G3A2	2	2	1	2	2	151	78	17.44789612	1	2	1	147/98	1	98	34	C	126	2	B+	11.5	3.4L	2	1	20	22	312	0.8	7.4	1	107	N	Y	PE WITH FOETAL DISTRESS	2	2	2	2	2	2	2	2	2	1	2	2.7	7	8	2
AMK	20	04-10-2024	10050400	2	04-07-2024	29-09-2024	26+6	1	1	1	2	2	2	2	1	PRIMI	2	2	2	2	2	146	50	41.0552	1	2	1	170/110	2	100	24	B	120	2	O+	11.8	1.7L	3	0.9	28	30	310	0.9	7.4	2	12	Y	N	SEVERE PE WITH BREECH WITH ABNORMAL DOPPLER	2	2	2	2	2	2	2	2	2	1	2	0.6	3	5	1
RPT	27	04-10-2024	10095747	1	26-10-2024	20-01-2024	37+2	2	2	2	2	2	2	2	7	G3P2L2	2	2	2	2	2	162	58	33.85434007	1	2	1	154/98	1	92	36	C	144	2	A+	13.7	1.5L	2	0.7	16	18	300	0.8	4.4	1	364	N	Y	PE WITH PREVIOUS LSCS	2	2	2	2	2	2	2	2	2	1	2	2.6	7	8	2
MUH	35	18-12-2024	10117348	1	19-01-2025	14-04-2024	37+2	2	2	2	2	2	2	2	7	G2A1	2	2	1	2	2	150	94	11.93413309	2	2	2	150/94	1	96	36	C	122	2	B-	10.2	2.1L	1	0.6	6	14	267	0.5	6.5	1	259	N	Y	PE WITH FOETAL DISTRESS	2	2	2	2	2	2	2	2	2	1	2	3.1	8	9	2
MIA	24	03-01-2025	10118198	1	26-01-2025	20-04-2024	36+0	YES	2	2	2	2	2	2	2	G2P1L1	1	2	1	1	1	158	58	33.01843044	1	2	1	160/104	2	88	36	C	134	2	0+	5.6	43K	2	1.9	36	44	296	0.9	8	2	128	N	Y	SEVERE PE WITH HELLP	2	2	2	2	2	2	2	1	2	1	2	2.1	5	8	1
PAK	34	04-01-2025	10118697	2	16-01-2025	11-04-2025	38+0	2	2	1	2	2	2	2	6	G2A1	2	2	2	1	2	152	66	24.53076217	1	2	1	156/100	1	98	36	C	156	2	O+	11.5	2.08L	2	0.52	14	26	368	0.6	7	1	134	N	Y	PE WITH FOETAL DISTRESS	2	2	2	2	2	2	2	2	2	1	2	2.5	7	9	2
BSK	24	24-12-2024	10115607	1	13-01-2025	08-04-2024	36+4	2	2	2	2	2	2	2	1	PRIMI	2	2	2	1	2	162	64	27.80419922	1	2	2	154/90	1	92	36	C	148	2	B+	11.9	2.4L	1	0.36	12	21	240	0.5	8	1	140	N	Y	PE WITH FOETAL DISTRESS	2	2	2	2	2	2	2	2	2	1	2	3.1	8	9	2
SCB	27	16-12-2024	10113704	1	16-02-2025	10-05-2024	31+5	1	2	2	2	2	2	2	2.5	PRIMI	2	2	2	2	2	156	48	47.59895833	1	2	1	150/90	1	88	32	B	144	2	A+	10.4	1.9L	1	0.24	16	25	252	0.7	6.1	1	135	N	Y	PE WITH BREECH	2	2	2	2	2	2	2	2	2	1	2	1.06	6	8	1
ALA	26	18-12-2024	10114105	2	28-01-2025	23-04-2024	34+1	1	2	1	2	2	2	2	1.5	PRIMI	2	2	2	2	2	155	62	28.34677419	1	2	1	164/110	2	102	32	C	158	2	B+	11	2.4L	2	0.48	24	14	282	0.9	7.1	2	49.9	N	Y	SEVERE PE	2	2	2	2	2	2	2	2	2	1	2	1.6	6	8	1
RAK	32	19-12-2024	10114664	1	26-01-2025	19-04-2024	35+3	2	2	1	2	2	2	2	2	G2P1L1	2	1-MS	1	2	1	150	72	20.34143519	2	2	2	146/96	1	84	34	C	128	2	AB+	11.9	1.7L	1	0.64	12	8	357	0.6	3	1	202	N	Y	PE WITH PREVIOUS LSCS	2	2	2	2	2	2	1	2	2	1	2	2.7	7	9	2
SMI	21	21-12-2024	10115157	2	09-03-2025	02-06-2024	26+5	1	1	1	1	2	2	2	2	PRIMI	1	2	2	2	2	159	50	44.7108	1	2	1	158/116	2	98	26	UNSTABLE	156	2	A+	11.4	3.4L	2	0.14	2	9	529	0.5	5	2	96	Y	N	SEVERE PE	2	2	2	2	2	2	2	2	2	2	1	900GM			
VLS	22	17-12-2024	10114091	2	26-01-2025	21-04-2024	34+2	1	1	1	1	2	1	2	2	PRIMI	2	2	2	2	2	152	50	42.7424	1	2	1	152/116	2	96	32	C	148	2	AB -	13.1	4.6L	2	0.9	23	8	545	0.66	4	3	78	N	Y	ECLAMPSIA	2	2	2	2	2	2	2	2	1	1	2	1.7	6	9	1
DAD	30	17-10-2024	1009880	1	10-11-2024	04-12-2023	36+5	1	2	1	2	2	2	2	11	G3P2L2	2	2	2	2	2	152	54	36.64471879	1	2	1	152/102	1	88	36	C	160	2	O-	11.3	2.2L	2	0.4	32	7	290	0.74	1	1	114	N	Y	PE WITH PREVIOUS LSCS	2	2	2	2	2	2	2	2	2	1	2	2.1	7	8	1
NVB	26	16-12-2024	10113608	1	17-05-2025	10-08-2024	24+5	1	1	1	1	2	2	2	3	PRIMI	2	2	2	2	2	148	62	27.06659729	1	2	1	170/120	2	102	22	UNSTABLE	ABSENT	2	AB+	9.3	2.3L	3	0.88	37	10	381	1.3	5.8	2	50	Y	N	SEVERE PE WITH AEDF	2	2	2	2	2	2	2	2	2	2	1	600GM			
SUM	30	15-12-2024	10112020	2	06-01-2025	01-04-2024	37+0	2	2	1	2	2	2	2	5	G2P1L1	2	2	2	2	1	154	110	8.947272727	1	2	1	154/100	1	96	36	C	132	2	A+	12.7	4.1L	1	0.4	14	25	256	0.88	6.6	1	160	N	Y	PE WITH PREVIOUS LSCS	2	2	2	2	2	2	2	2	2	1	2	3.2	6	9	2
DSE	23	26-12-2024	10115994	1	09-0-2025	04-04-2024	38+0	2	2	1	2	2	2	2	5	G2P1L1	1	2	2	2	2	156	51	42.16378316	1	2	1	152/96	1	68	38	C	154	2	A-	12.9	2.1L	1	0.7	15	15	283	0.65	2.3	1	136	N	Y	PE WITH FOETAL DISTRESS	2	2	2	2	2	2	2	2	2	1	2	2.5	8	9	2
RSK	33	26-12-2024	10116255	1	10-01-2025	03-04-2024	38+0	2	2	2	2	2	2	2	10	G4P1L1A2	2	2	2	2	2	150	86	14.25770687	1	2	2	150/100	1	88	38	C	132	2	A+	13.7	1.9L	1	0.54	17	14	212	0.7	5.1	1	224	N	Y	PE WITH PREVIOUS LSCS	2	2	2	2	2	2	2	2	2	1	2	3.3	8	9	2
PBY	33	02-12-2024	10116388	1	15-02-2024	21-Nov	40+0	2	2	2	2	2	2	2	2	PRIMI	2	2	2	2	2	152	56	34.07397959	1	2	1	146/90	1	98	36	C	144	2	O+	11.2	1.9L	1	0.23	8	19	364	0.63	2.5	1	94.2	Y	N	PE	2	2	2	2	2	2	2	2	2	1	2	3	7	8	2
SBK	36	03-12-2024	10114684	2	UNKNOWN	UNKNOWN	34+5	1	2	1	1	2	2	2	10.5	G4P3L3	2	2	2	1	1	148	52	38.47781065	1	2	1	160/124	2	110	32	C	140	2	B+	13.2	1.31L	3	0.37	55	30	269	0.5	2.4	2	18.5	N	Y	SEVERE PE WITH FGR	2	2	2	2	2	2	2	2	2	1	2	1.5	5	7	1
MLK	26	26-11-2024	10108924	2	16-02-2025	12-05-2024	26+0	YES	1	YES	YES	1	2	2	1	PRIMI	1	2	1	2	2	156	48	47.59895833	1	2	1	160/110	2	86	24	B	158	2	B+	10.2	2.3L	3	0.64	9	25	202	0.67	4.9	2	30	N	Y	SEVERE PE WITH BREECH WITH ABNORMAL DOPPLER	2	2	2	2	2	2	2	2	2	1	2	680GM	4	5	1
SGB	40	22-11-2024	10107883	1	16-01-2025	11-04-2024	38+3	2	2	2	2	2	2	2	3	PRIMI	2	2	2	2	2	150	56	33.62563776	1	2	2	144/92	1	84	38	C	136	2	B+	8.8	2.1L	1	0.84	8	21	218	0.74	5.2	1	120	N	Y	PE WITH FOETAL DISTRESS	2	2	2	2	2	2	2	2	2	1	2	2.8	7	9	2
SKL	28	27-11-2024	10109006	1	UNKNOWN	UNKNOWN	26+0	1	1	1	1	1	1	2	2	PRIMI	2	2	2	2	2	142	48	43.32725694	1	2	1	162/114	2	88	26	B	122	2	A+	13.2	2.2L	2	0.37	6	24	184	0.85	2.4	3	39.6	Y	N	ECLAMPSIA WITH ABRUPTION	2	1	2	2	1	2	2	2	1	2	1	554GM	0	0	2
SAG	22	29-10-2024	10109884	1	29-11-2024	22-02-2024	36+6	2	2	1	2	2	2	2	1.5	PRIMI	2	2	2	2	2	156	52	40.55769231	1	2	1	154/92	1	90	36	B	154	2	A+	11.7	1L	1	0.64	11	22	292	0.88	6	1	102	N	Y	PE WITH BREECH	2	2	2	2	2	2	2	2	2	1	2	2.8	7	8	2
PMH	24	16-11-2024	10106779	1	19-12-2024	12-Mar	36+0	2	2	1	2	2	2	2	2.5	PRIMI	2	2	2	2	2	141	53	35.28764685	1	2	1	150/92	1	96	36	B	128	2	A+	8.2	1.92L	1	0.36	12	28	208	0.67	6.7	1	58.8	N	Y	PE WITH BREECH	2	2	2	2	2	2	2	2	2	1	2	2.7	8	9	2
NSG	31	05-12-2024	10111164	1	22-12-2024	15-03-2024	37+2	1	2	2	2	2	2	2	7	PRIMI	1	2	1	1	1	160	88	14.52479339	1	2	1	164/102	2	88	36	C	148	2	O+	11.2	3.02L	2	0.84	14	8	264	0.63	7.4	2	64	N	Y	SEVERE PE WITH FGR	2	2	2	2	2	2	2	2	2	1	2	2.5	6	9	2
YAS	28	15-11-2024	10106120	1	22-12-2024	15-03-2024	35+2	2	2	1	2	2	2	2	7.5	G3P2D2	1	2	1	2	1	155	50	43.586	1	2	1	160/100	2	96	34	C	142	2	A+	13.8	78K	1	0.65	21	9	368	0.39	6.4	2	120	N	Y	SEVERE PE WITH HELLP	2	2	2	2	2	2	2	1	2	1	2	2.6	7	9	2
AAP	38	19-11-2024	10107253	1	17-01-2025	10-04-2024	31+0	1	1	1	2	2	2	2	15	G3P1L1D1	2	2	2	1	1	149	60	29.09638889	1	2	1	162/114	2	106	32	B	144	2	A+	9	60K	2	0.23	45	18	428	0.68	8.8	2	54.5	Y	N	SEVERE PE WITH HELLP	2	2	2	2	2	2	2	1	2	1	2	1	4	6	1
SSM	19	19-11-2024	10107120	1	01-01-2025	25-03-2024	36+0	1	2	1	1	2	2	2	1.5	PRIMI	2	2	2	2	2	152	49	44.50478967	1	2	1	164/112	2	124	32	B	132	2	A+	8.2	90K	3	0.52	20	22	300	0.77	7.2	2	21.2	N	Y	SEVERE PE WITH HELLP WITH ABRUPTIO	2	1	2	2	2	2	2	1	2	1	2	2.1	5	7	1
USM	34	30-11-2024	10110989	1	25-11-2024	19-03-2024	37+0	2	2	2	2	2	2	2	7	G3P2L1D1	2	2	2	2	2	161	82	16.83268888	1	2	1	136/90	1	106	36	C	144	2	B+	12.4	1.8L	1	0.47	19	5	202	0.8	3.4	1	120	N	Y	PE WITH PREVIOUS LSCS	2	2	2	2	2	2	2	2	2	1	2	2.9	8	9	2
AAM	31	18-12-2024	10114386	1	29-12-2024	22-03-2024	38+5	2	2	1	2	2	2	2	12	G3P1L1A1	2	2	1	2	1	168	72	22.78240741	1	2	1	150/94	1	90	38	C	144	2	B+	11.6	92K	1	0.52	25	2	400	0.6	6.2	1	71.9	N	Y	PE WITH FOETAL DISTRESS	2	2	2	2	2	2	2	2	2	1	2	3	6	8	2
LGD	21	19-12-2024	10114536	1	22-12-2024	15-03-2024	36+4	2	2	2	2	2	2	2	2	PRIMI	2	2	1	2	2	163	70	23.3855102	1	2	1	164/108	2	72	36	C	152	2	AB+	11.4	2.3L	1	0.34	24	24	345	0.54	4.2	2	59.6	Y	N	SEVERE PE	2	2	2	2	2	2	2	2	2	1	2	2.7	8	9	2
LMP	22	02-12-2024	101110289	2	17-12-2024	10-03-2024	38+5	2	2	2	2	2	2	2	1	PRIMI	2	2	1	2	2	145	51	39.19069589	1	2	1	142/90	1	96	38	C	156	2	O+	12.2	2.1L	1	0.36	23	26	282	0.69	2.7	1	86.9	Y	N	PE	2	2	2	2	2	2	2	2	2	1	2	2.9	8	9	2
AAN	21	27-11-2024	10109264	1	22-12-2024	17-03-2024	36+3	2	2	2	2	2	2	2	2	PRIMI	2	2	2	2	2	146	49	42.74802166	1	2	2	144/90	1	80	36	C	146	2	A+	12.2	1.5L	1	0.21	21	21	420	0.87	7.8	1	82.9	Y	N	PE	2	2	2	2	2	2	2	2	2	1	2	2.6	8	9	2
AAH	29	09-11-2024	10112640	2	07-12-2024	01-03-2024	34+1	1	1	2	2	2	2	2	2	G2P1L1	2	2	2	2	2	151	52	39.25776627	1	2	1	168/114	2	98	34	B	128	2	O+	10.8	96K	1	0.62	28	30	220	0.42	2.5	2	108	N	Y	SEVERE PE WITH HELLP	2	2	2	2	2	2	2	1	2	1	2	1.3	8	9	1
TSL	35	08-11-2024	10102801	2	29-12-2024	22-03-2024	28+2	1	1	1	1	2	1	2	1	G3P2L2	1	2	2	1	2	152	60	29.68222222	1	2	1	162/116	2	84	26	B	142	2	AB+	10.6	1.2L	3	0.8	25	25	510	0.63	2.6	3	10	N	Y	ECLAMPSIA AND ABRUPTIO	2	1	2	2	2	2	2	2	2	1	2	1	5	6	1
SSP	21	28-10-2024	10101640	1	12-12-2024	05-03-2024	30+1	1	1	1	1	1	2	2	3	G2A1	1	2	1	2	2	156	54	37.6090535	2	2	2	170/120	2	100	26	B	112	2	B+	10.4	1.2L	2	0.4	21	15	452	0.45	2.1	2	16.2	N	Y	SEVERE PE WITH BREECH WITH ABNORMAL DOPPLER	2	2	2	2	2	2	1	2	2	1	2	1.06	6	7	2
JMD	21	26-10-2024	10105100	1	01-01-2025	27-03-2024	30+1	1	1	1	1	1	2	2	7.5	G4A3	2	2	2	1	1	168	79	18.9238904	1	2	1	170/116	2	110	30	B	148	2	A+	12.4	1.5L	2	0.5	14	13	492	0.85	3.5	2	12.7	N	Y	SEVERE PE WITH BREECH WITH ABNORMAL DOPPLER	2	2	2	2	2	2	2	2	2	1	2	780GM	4	4	1
ASP	33	22-11-2024	10101164	1	UNKNOWN	UNKNOWN	36+6	1	2	2	2	2	2	2	6	PRIMI	2	2	2	1	1	164	72	22.23996914	1	2	1	150/100	1	88	32	C	144	2	AB+	10.4	2.1L	1	0.12	16	14	208	0.81	6.3	1	82.3	N	Y	PE WITH FGR	2	2	2	2	2	2	2	2	2	1	2	2.3	8	9	2
MSM	25	22-11-2024	10107617	1	03-01-2025	27-03-2024	35+1	2	2	2	2	2	2	2	1.5	PRIMI	2	2	1	2	2	144	54	34.71604938	1	2	1	144/90	1	96	34	C	144	2	O+	12	2.1L	2	0.36	18	17	356	0.9	4.3	1	207	N	Y	PE WITH FOETAL DISTRESS	2	2	2	2	2	2	2	2	2	1	2	2	8	9	1
KAL	20	06-12-2024	10114861	2	31-01-2025	26-04-2024	35+1	1	1	1	2	2	2	2	2	PRIMI	2	2	2	2	2	148	62	27.06659729	2	2	1	172/106	2	98	28	C	152	2	O+	9.9	1.2L	2	0.95	6	9	394	0.83	4.7	2	42.6	N	Y	SEVERE PE WITH ABNORMAL DOPPLER	2	2	2	2	2	2	2	2	2	1	2	1.6	7	9	1
MAM	22	03-12-2024	10166426	1	10-03-2025	03-06-2024	26+1	1	1	1	2	2	2	2	5	PRIMI	2	2	2	1	2	156	82	16.30993456	1	2	2	186/112	2	102	18	UNSTABLE	148	2	O+	10	2.1L	2	0.4	9	10	468	0.72	4.5	2	23.6	N	Y	SEVERE PE WITH FGR WITH ABNORMAL DOPPLER	2	2	2	2	2	2	2	2	2	1	2	630GM	4	5	1
YSN	26	22-11-2024	10612438	2	04-01-2025	28-03-2024	35+1	2	2	2	2	2	2	2	4	PRIMI	2	2	1	2	2	160	80	17.575	2	2	1	146/98	1	84	32	C	144	2	AB+	10.2	1.8L	1	0.7	5	20	168	0.6	4.8	1	120	N	Y	PE WITH FOETAL DISTRESS	2	2	2	2	2	2	2	2	2	1	2	2.4	7	8	2

SRT	40	05-11-2024	10105163	2	15-01-2025	20-04-2024	30+5	2	2	1	2	2	2	2	12	PRIMI	1	2	2	2	2	151	81	16.17939339	1	2	1	152/98	1	102	30	C	144	2	O+	12	1.4L	1	0.2	24	15	108	0.4	5.1	1	191	Y	N	PE	2	2	2	2	2	2	2	2	2	1	2	2.6	7	8	2
MSS	24	13-11-2024	10105277	1	06-12-2024	01-03-2024	30+4	1	2	1	1	2	2	2	2	G2A1	2	2	1	2	2	150	68	22.8049308	1	2	1	182/110	2	94	34	C	162	2	B+	9.1	82K	2	0.338	26	21	922	0.6	6	2	52.3	N	Y	SEVERE PE WITH FGR	2	2	2	2	2	2	2	2	2	1	2	2.1	7	8	2
PRK	29	01-02-2025	10125011	1	06-05-2024	27-05-2024	35+0	2	2	2	2	2	2	2	7	PRIMI	2	2	2	2	1	159	94	12.65018108	1	2	1	140/92	1	98	34	C	142	2	O+	12.8	2.1L	1	0.14	15	13	272	0.74	2.7	1	316	N	Y	PE WITH FOETAL DISTRESS	2	2	2	2	2	2	2	2	2	1	2	2.4	7	8	2
SPH	46	03-11-2024	10110547	1	29-01-2025	22-04-2024	31+6	1	2	1	1	2	2	2	16	PRIMI	1	2	2	1	1	158	69	23.32997269	1	2	1	150/104	1	102	32	C	148	2	B+	11.4	1.6L	1	0.52	14	16	302	0.72	2.1	1	103	N	Y	PE WITH FGR	2	2	2	2	2	2	2	2	2	1	2	1.2	5	7	1
ASK	25	30-01-2025	10124993	1	29-02-2025	22-05-2024	36+1	1	2	1	1	2	2	2	2.5	G3A2	2	2	2	2	2	153	50	43.0236	1	2	1	160/102	2	84	36	C	152	2	A+	12.7	60K	2	0.64	12	14	2293	0.64	3.6	2	49.4	N	Y	SEVERE PE WITH HELLP	2	2	2	2	2	2	2	1	2	1	2	2.4	7	8	2
BSK	24	24-12-2024	10115607	1	03-01-2025	28-03-2024	37+3	2	2	2	2	2	2	2	2	PRIMI	2	2	2	2	2	158	70	22.66816327	1	2	1	144/92	1	96	36	C	144	2	B+	11.8	1.8L	1	0.48	15	11	310	0.54	6.1	1	140	N	Y	PE WITH FOETAL DISTRESS	2	2	2	2	2	2	2	2	2	1	2	3	8	9	2
SNH	24	22-01-2025	10122908	1	05-02-2025	07-05-2024	36+0	2	2	1	2	2	2	2	2	PRIMI	2	2	2	2	2	146	64	25.05810547	1	2	1	150/100	1	96	36	C	152	2	B+	7.5	1.86L	2	2.6	129	9	1796	0.7	9.6	1	51.8	N	Y	PE WITH HELLP	2	2	2	2	2	2	2	1	2	1	2	2.6	6	8	2
ALP	24	20-01-2025	10121973	1	20-01-2025	15-04-2024	39+6	2	2	2	2	2	2	2	2	PRIMI	2	2	2	2	2	152	81	16.28654169	2	2	1	170/104	2	94	36	C	128	2	O+	11.2	1.46L	1	0.4	15	9	241	0.7	4.3	2	103	Y	N	SEVERE PE WITH HELLP	2	2	2	2	2	2	2	1	2	1	2	2.6	6	7	2
JBP	19	17-01-2025	10121661	2	27-03-2025	20-06-2024	30+1	2	2	1	1	2	2	2	1	PRIMI	2	2	2	2	2	152	71	21.19738147	2	2	1	168/106	2	96	24	B	132	2	B+	10.9	3L	2	0.7	9	7	315	0.6	5.2	2	22	Y	N	SEVERE PE WITH ABRUPTION	2	1	2	2	2	2	2	2	2	1	2	730GM	4	5	1
DPK	29	08-01-2025	11011026	2	15-03-2025	08-06-2024	30+4	1	1	1	2	2	2	2	5	PRIMI	2	2	2	1	2	146	80	16.0371875	2	2	1	162/110	2	106	28	C	154	2	B-	12.4	2.5L	1	0.84	6	3	816	0.52	2.1	2	13.7	N	Y	SEVERE PE WITH FGR WITH ABNORMAL DOPPLER	2	2	2	2	2	2	2	2	2	1	2	1.3	7	8	1
SMR	24	26-12-2025	1001547	1	30-03-2024	04-01-2025	38+5	2	2	2	2	2	2	2	2	PRIMI	2	2	2	2	2	154	74	19.77027027	2	2	1	146/92	1	86	36	C	132	2	A+	11.8	2.7L	1	0.25	5	21	124	0.48	2.8	1	124	Y	N	PE	2	2	2	2	2	2	2	2	2	1	2	2.7	6	9	2
KPS	26	13-12-2024	10112346	2	15-01-2025	10-04-2024	35+2	2	2	2	2	2	2	2	3	G2P1L1	2	2	2	2	2	142	78	16.40795529	1	2	1	142/94	1	89	34	C	124	2	O+	12.1	1.9L	1	0.31	12	16	206	0.85	6	1	106.3	N	Y	PE WITH PREVIOUS LSCS	2	2	2	2	2	2	2	2	2	1	2	2.2	7	8	2
MRP .	24	24-12-2024	10111234	2	11-01-2025	06-04-2024	37+3	1	1	1	2	2	2	2	2	PRIMI	2	2	2	2	2	154	82	16.10083284	1	2	1	164/110	2	78	34	C	132	2	AB+	9.8	1.6L	2	0.47	14	14	307	0.6	7.6	2	57	N	Y	SEVERE PE WITH ABNORMAL DOPPLER	2	2	2	2	2	2	2	2	2	1	2	2.08	7	8	2
PSR.	26	14-11-2024	10115210	2	30-12-2024	25-03-2024	33+3	1	2	1	2	2	2	2	5	G2P1L1	2	2	2	2	2	148	96	11.28949653	2	2	1	152/92	1	94	28	B	143	2	B+	10.3	1.7L	2	0.9	5	18	286	0.7	6.8	1	77.1	N	Y	PE WITH BREECH	2	2	2	2	2	2	2	2	2	1	2	1.6	6	7	1
LEK	23	16-12-2024	10121581	1	19-01-2025	14-04-2024	35+1	2	2	2	2	2	2	2	3	PRIMI	2	2	2	2	2	148	64	25.40136719	2	2	1	156/90	1	98	34	C	125	2	A+	11.3	1.6L	1	0.51	12	19	203	0.9	2.1	1	93.2	N	Y	PE WITH FOETAL DISTRESS	2	2	2	2	2	2	2	2	2	1	2	2.5	8	9	2
NMD.	35	10-02-2025	1012752	1	01-03-2025	25-05-2024	37+2	1	2	1	1	2	2	2	7	G2P1L1	2	2	2	2	2	154	79	17.34689954	2	2	1	160/94	2	98	36	C	135	2	B=	11.9	4.2L	2	0.72	14	15	334	0.5	1.8	2	87.6	N	Y	SEVERE PE WITH FGR	2	2	2	2	2	2	2	2	2	1	2	2.3	8	9	2
KCW	24	07-12-2024	10111945	2	10-01-2025	05-04-2024	35+1	2	2	2	2	2	2	2	3	PRIMI	2	2	2	2	2	147	76	17.89144737	1	2	1	154/92	1	88	34	C	156	2	B+	12.6	3.4L	1	0.36	19	1	302	0.6	8.1	1	98	N	Y	PE WITH FOETAL DISTRESS	2	2	2	2	2	2	2	2	2	1	2	2.8	7	9	2
ASK.	21	10-02-2025	10127475	1	UNKNOWN	23-04-2025	29+5	2	2	1	1	2	1	2	2	PRIMI	2	2	2	2	2	154	68	23.41306228	2	2	1	172/106	2	108	26	B	154	2	A+	10	1.9L	3	0.47	17	16	453	0.8	2.6	3	34.6	Y	N	ECLAMPSIA	2	2	2	2	2	2	2	2	2	1	2	1.06	4	7	1
DK.	24	14-11-2024	10021547	2	30-12-2024	25-03-2024	33+3	1	1	1	1	2	2	2	2	PRIMI	2	2	1	2	2	149	75	18.62168889	1	2	1	164/108	2	88	28	C	158	2	O+	10.6	1.6L	2	0.4	1	13	534	0.74	3.7	2	13.7	N	Y	SEVERE PE WITH FGR WITH ABNORMAL DOPPLER	2	2	2	2	2	2	2	2	2	1	2	1.5	6	7	1
JP.	23	07-12-2024	10231104	1	10-01-2025	05-04-2024	35+1	1	2	1	2	2	2	2	1	PRIMI	2	2	2	2	2	154	82	16.10083284	2	2	1	160/104	2	86	32	C	135	2	B+	13.4	2.3L	2	0.74	15	9	422	0.63	7.5	2	22	N	Y	SEVERE PE WITH FGR	2	2	2	2	2	2	2	2	2	1	2	2.06	7	8	2
AL.	32	02-12-2024	10165472	1	14-12-2024	09-03-2024	38+2	2	2	2	2	2	2	2	7	G3P2L2	2	2	2	2	2	152	75	18.99662222	2	2	1	144/94	1	98	36	C	122	2	A+	12.6	2.4L	1	0.65	12	8	132	0.54	4.6	1	103	Y	N	PE	2	2	2	2	2	2	2	2	2	1	2	2.6	7	8	2
RG.	23	23-11-2024	10215431	2	08-12-2024	03-03-2024	37+6	2	2	2	2	2	2	2	2	PRIMI	2	2	2	2	2	155	74	19.89864865	2	2	1	144/92	1	64	36	C	132	2	B+	11.9	1.8L	1	0.4	11	12	108	0.7	4.4	1	131	Y	N	PE	2	2	2	2	2	2	2	2	2	1	2	2.5	7	8	2