"AN OBSERVATIONAL STUDY OF COAGULATION PROFILE IN SEVERE PRE ECLAMPSIA AND ECLAMPSIA PATIENTS."

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With reference to the above, we wish to inform you that your proposed research project titled **"AN OBSERVATIONAL STUDY OF COAGULATION PROFILE IN SEVERE PRE-ECLAMPSIA AND ECLAMPSIA PATIENTS"**, is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee.

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LIST OF ABBREVIATIONS USED

GLOSSARY	ABBREVIATIONS
ACOG	AMERICAN COLLEGE OF OBSTETRICIANS AND GYNECOLOGIST
AKI	ACUTE KIDNEY INJURY
APTT	ACTIVATED PROTHROMBIN TIME
ART	ASSISSTED REPRODUCTIVE TECHNIQUE
BT	BLEEDING TIME
CT	CLOTTING TIME
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.
ITP	IDIOPATHIC THROMBOCYTIC PURPURA
IVF	IN VITRO FERTILIZATION
PlGF	PLACENTAL GROWTH FACTOR
PT-INR	PROTHROMBIN TIME-INTERNATIONAL NORMALISED RATIO
ROS	REACTIVE OXYGEN SPECIES

sENG	SOLUBLE ENDOGLIN
TNF	TUMOR NECROSIS FACTOR
VEGF	VASCULAR ENDOTHELIAL GROWTH FACTORS
WHO	WORLD HEALTH ORGANISATION

ABSTRACT

Background: Hypertensive disorders in pregnancy complicate 7-15% of pregnancies and are the primary cause of direct maternal death, with hypertension, haemorrhage, and infections contributing to maternal mortality during pregnancy and childbirth. These disorders can be chronic, white coat, or masked, and can be pre-diagnosed before 20 weeks of pregnancy or newly diagnosed after 20 weeks, including gestational hypertension, transient gestational hypertension, and pre-eclampsia. Pre-eclampsia incidence is increasing in developed countries due to predisposing conditions like diabetes and obesity.

Objective: The primary objective of the study is to evaluate coagulation profile (PT-INR, APTT, D-DIMER, Serum Fibrinogen) in Severe Pre-eclampsia and Eclampsia patients.

Methods: In this observational study, a consecutive sampling method was employed to select participants. Over the 12-month study period (January 2024 to February 2025), all antenatal women diagnosed with severe pre-eclampsia or eclampsia who presented to the labor room of KLE's Dr. Prabhakar Kore Hospital, Belagavi, Karnataka, were considered for inclusion. All antenatal women with a gestational age of > 20 weeks, diagnosed with new onset hypertension were screened for the study. Data on demographic, Clinical investigation and Disseminated intravascular coagulation profile details were obtained. Obstetric ultrasound (Doppler) -results were recorded.

Results: The majority of participants (66.2%) were aged between 20 - 30 years, and 50% being primigravida and 50% multigravida. Most participants (73.0%) were in the 28+1 to 37-week range. 93.2% of participants were diagnosed with severe pre-

eclampsia, and 6.8% with eclampsia. Severe preeclampsia has lower platelet counts than eclampsia. D-dimer and fibrinogen do not show significant predictive value for PE or eclampsia. PT-INR is significantly associated with pre-eclampsia and eclampsia. APTT is also associated with both pre-eclampsia and eclampsia, but extreme values and wide odds ratios suggest model inconsistencies.

Conclusion: The study analysed coagulation profiles, maternal complications, and neonatal outcomes in severe pre-eclampsia (PE) and eclampsia, comparing findings with recent literature. Both conditions share common pathophysiological mechanisms but differ in clinical severity, coagulation abnormalities, and perinatal outcomes. Severe PE was more frequent than eclampsia, with PT-INR and APTT significantly associated with both conditions. Maternal complications were more frequent in severe PE, and neonatal mortality was higher in eclampsia.

Keywords: Coagulation profile, severe preeclampsia, eclampsia, pregnancy-induced hypertension (PIH), disseminated intravascular coagulation (DIC), Prothrombin time (PT), fibrinogen, platelet count.

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INTRODUCTION

Hypertensive disorders of pregnancy (HDP) affect maternal and fetal health worldwide. Pre-eclampsia and eclampsia are preeminent causes of maternal and perinatal death [1], [2]. About 5—10% of pregnancies worldwide are affected by pre-eclampsia, which causes new-onset hypertension after 20 weeks and proteinuria or organ failure [3]. Recurrent seizures in a woman with pre-eclampsia without a neurological reason highlight the severity of these illnesses [5]. Pre-eclampsia and eclampsia cause 10—15% of maternal deaths globally, with a higher burden in low-income countries. [7]

These disorders have multiple pathophysiological causes, including aberrant placentation, endothelial dysfunction, and immune system dysregulations [8]. Normal pregnancy causes a physiological shift towards hypercoagulability to avoid excessive haemorrhage after birth [9]. This adaptation may become pathological in pre-eclampsia, causing endothelial activation, procoagulant factor release, and platelet aggregation [10]. A major haematological consequence of severe pre-eclampsia is thrombocytopenia, which is a diagnostic and prognostic marker [11]. Platelet consumption at microvascular damage sites causes low counts, which may indicate disease severity or development to HELLP syndrome [12]. Pre-eclampsia and eclampsia are at risk for disseminated intravascular coagulation (DIC), a life-threatening disease characterised by extensive clotting factor consumption and hemorrhagic consequences [13]. Prolonged prothrombin time (PT) and activated partial thromboplastin time (aPTT), decreased fibrinogen, and higher D-dimer values indicate excessive clot disintegration in pre-eclamptic individuals [14]. In severe coagulopathy, fibrinogen, an acute-phase reactant generally increased in pregnancy, may decrease, indicating DIC [15]. Haematological abnormalities promote maternal

haemorrhage and organ failure and impair placental perfusion, resulting in intrauterine growth restriction, preterm, and stillbirth [16]. Early diagnosis and management in pre-eclamptic patients need coagulation marker monitoring due to these hazards.

The dangers of severe pre-eclampsia and eclampsia emphasize the need for early diagnosis and treatment. Mothers must seek prompt clinical attention to prevent cerebrovascular accidents, abruptio placentae, pulmonary oedema, and hepatic dysfunction [17]. In eclampsia, seizures may worsen haemodynamic instability and increase the risk of placental abruption or cerebral haemorrhage [18]. Hypoxia, growth limitation, and perinatal death result from poor uteroplacental circulation [19]. These issues are caused by poor placental perfusion and coagulation disorders that cause placental vascular thrombus [20]. These early haematological warning indicators allow doctors to adopt preventative interventions including hypertension medicine, magnesium sulphate for seizure prophylaxis, or early delivery in high-risk individuals [21].

Despite the importance of coagulation disorders in pre-eclampsia and eclampsia pathogenesis, clinical coagulation monitoring is inconsistent [22]. Laboratory diagnoses are scarce in resource-limited settings, delaying high-risk patient identification [6]. However, regular platelet count and fibrin degradation product testing may improve risk stratification and guide treatment choices [23]. Identifying patterns like a dramatic reduction in platelet counts or growing D-dimer concentrations might assist doctors prioritise high-risk individuals for immediate treatment, transfer to specialised facilities, or early delivery [24]. Integrating coagulation monitoring into conventional obstetric practices might guide proactive

measures like preventive low-dose aspirin or magnesium sulphate usage in high-risk pregnancies, lowering unfavourable maternal and foetal outcomes [25].

Besides clinical care, coagulation anomalies in pre-eclampsia and eclampsia affect public health policy and maternal mortality reduction. In places with high maternal mortality, population-specific risk factors and coagulation profile may improve preventative strategies and resource allocation [7]. Research into anti-angiogenic factors including soluble fms-like tyrosine kinase-1 (sFlt-1) may lead to new pre-eclampsia treatments for endothelial dysfunction and coagulation disorders. [8]

AIMS AND OBJECTIVES

PRIMARY OBJECTIVE-

The primary objective of the study is to evaluate coagulation profile (PT-INR, APTT, D-DIMER, Serum Fibrinogen) in Severe Pre-eclampsia and Eclampsia patients.

REVIEW OF LITERATURE

BACKGROUND -

Hypertensive disorder complicate 7-15% of the pregnancies. By WHO (World Health Organization's) systematic review hypertensive disorders of pregnancy stand as primary cause of direct maternal death. Hypertension, hemorrhage, infections form lethal trio contributing to maternal mortality during pregnancy and child birth. [33,34]. Hypertensive disorders in pregnancy may be chronic, white coat, or masked hypertension that predates pregnancy (which is diagnosed before 20 weeks of pregnancy) or newly diagnosed after 20 weeks which includes gestational hypertension, transient gestational hypertension, and pre-eclampsia [31].

Occurrence of pre- eclampsia is on rise in developed countries [35].This is due to growing prevalence of predisposing conditions like diabetes, chronic hypertension, obesity .[36]

Hypertensive disorders of pregnancy –

Presenting before20 weeks of pregnancy

1. Essential hypertension or secondary hypertension
2. White coat hypertension
3. Masked hypertension

Presenting beyond 20 weeks of gestation

1. Transient gestational hypertension
2. Gestational hypertension
3. Pre-eclampsia

Definition of preeclampsia and eclampsia

Pre-eclampsia-

As per recent definitions provided by the ISSHP (International Society for the Study of Hypertension in Pregnancy) and ACOG (American College of Obstetricians and Gynaecologists), preeclampsia is characterized as a pregnancy disorder linked with new-onset hypertension appearing after 20 weeks of gestation, often occurring close to term. While proteinuria is commonly present, hypertension and other pre-eclampsia indicators may manifest in some women without proteinuria. They recommend categorizing cases as preeclampsia without severe features or with severe features. [33,37]

Blood pressure

Systolic blood pressure of 140 mmHg or more or diastolic blood pressure of 90 mmHg or more on two occasions at least 4 hours apart after 20 weeks of gestation in a woman with a previous normal blood pressure.

Proteinuria

300 mg or more per 24-hour urine collection, protein /creatinine ratio of 0.3mg/dl or more, or dipstick reading of 1+.

Diagnostic criteria for pre-eclampsia with severe feature

Systolic blood pressure of 160 mmHg or more or diastolic blood pressure of 110 mmHg 15 minutes apart with or without proteinuria as mentioned above along with the new onset of –

- Thrombocytopenia which is platelet count less than $< 100 \times 10^9$ / L.
- Impaired liver function, not explained by another diagnosis, is characterized by significantly elevated levels of liver enzymes in the blood (more than double the upper limit of normal) or by severe and persistent pain in the upper right quadrant or epigastric region that does not respond to medication.
- Renal insufficiency (serum creatinine concentration more than 1.1 mg/dl or a doubling of concentration in the absence of other renal disease.
- Pulmonary oedema
- New onset headache unresponsive to medication and not accounted for by alternative diagnosis.
- Visual disturbances. [33]

Eclampsia-

Eclampsia is a severe complication of pre-eclampsia, characterized by the onset of generalized tonic-clonic seizures in a pregnant woman with pre-eclampsia, in the absence of other neurological disorders. It is a life-threatening condition that can lead to cerebral hemorrhage, stroke, pulmonary edema, and maternal or fetal death. Eclampsia often occurs in the third trimester but can also present during labor or postpartum. Immediate medical intervention, including magnesium sulfate administration and delivery planning, is crucial to prevent further complications and ensure maternal and fetal well-being.

- Renal insufficiency (serum creatinine concentration more than 1.1 mg/dl or a doubling of concentration in the absence of other renal disease.
- Pulmonary oedema

- New onset headache unresponsive to medication and not accounted for by alternative diagnosis.
- Visual disturbances [33]

Studies show that women with a history of pre-eclampsia have a 20—40% likelihood of having it again in future pregnancies. Pre-eclampsia that occurs before 34 weeks increases the likelihood of recurrence.

Improved pre-eclampsia prediction and prevention are ongoing due to its considerable influence on maternal and fetal health. In high-risk women, biomarker screening, Doppler ultrasounds, and low-dose aspirin may reduce occurrence. Pre- eclampsia is a serious public health issue, especially in resource-limited regions where maternal healthcare services must be improved.

Risk factors for pre-eclampsia

Pre-eclampsia is a complex disorder influenced by multiple maternal, fetal, and environmental factors. Several risk factors have been identified that increase a woman's likelihood of developing pre-eclampsia. These risk factors can be classified into maternal characteristics, medical conditions, obstetric history, and genetic/environmental influences.

1. Maternal Characteristics

- First-time pregnancy (Nulliparity): Women experiencing their first pregnancy have a two to three times higher risk of developing pre-eclampsia compared to multiparous women.
- Maternal age factor: Women under 20 years or over 35 years have a significantly higher risk.

- Obesity: A body mass index (BMI) ~ 30 kg/m^2 is strongly associated with an increased risk of pre-eclampsia due to its impact on systemic inflammation and endothelial dysfunction.
- Ethnicity: Studies suggest that women of African, Hispanic, and South Asian descent have a higher predisposition to pre-eclampsia compared to Caucasian women, possibly due to genetic and healthcare disparities.
- Family history: A woman with a mother or sister who had pre-eclampsia is at an increased risk, indicating a hereditary component to the condition.

2. Pre-existing Medical Conditions

- Chronic Hypertension: Women with a history of high blood pressure before pregnancy are at greater risk of developing superimposed pre-eclampsia.
- Diabetes Mellitus: Both type 1 and type 2 diabetes increase the likelihood of endothelial dysfunction, predisposing women to pre-eclampsia.
- Kidney Disease: Impaired renal function contributes to hypertension and proteinuria, increasing pre-eclampsia susceptibility.
- Autoimmune Disorders: Conditions such as lupus and antiphospholipid syndrome increase the risk due to systemic inflammation and vascular dysfunction.

3. Obstetric and Pregnancy-Related Factors

- History of Pre-eclampsia: Women who have experienced pre-eclampsia in a previous pregnancy have a 20% chance of recurrence, with early-onset cases posing an even higher risk.
- Multiple Gestation (Twins or More): Carrying more than one fetus increases placental mass, leading to higher vascular demands and increased risk of pre-

eclampsia.

- Short or Long Interpregnancy Interval: An interval of less than 2 years or more than 10 years between pregnancies has been associated with a higher likelihood of developing pre-eclampsia.
- Assisted Reproductive Technology (ART): Pregnancies conceived through in vitro fertilization (IVF) or other assisted reproductive technologies have a higher risk, possibly due to altered placental development.
- Genetic and Environmental Factors
- Paternal Contribution: Studies suggest that paternal genetic factors may influence pre-eclampsia risk, as a history of pre-eclampsia in previous partners increases susceptibility.
- High-altitude Pregnancy: Women living at high altitudes have an increased risk due to lower oxygen availability, which may impact placental function.
- Dietary and Lifestyle Factors: Poor nutrition, excessive salt intake, and lack of physical activity may contribute to vascular dysfunction and increased pre-eclampsia risk.
- Socioeconomic Status: Limited access to prenatal care in low-income populations contributes to delayed diagnosis and higher incidence of pre-eclampsia.

Pre-eclampsia is a multifactorial condition influenced by genetic, medical, obstetric, and environmental factors. Early identification of high-risk women through comprehensive screening and risk stratification is crucial for implementing preventive measures, such as low-dose aspirin, lifestyle modifications, and close antenatal monitoring, to reduce the incidence and severity of pre-eclampsia.

Pathogenesis of preeclampsia

Pre-eclampsia is a complex, multisystem disorder arising from abnormal placentation, endothelial dysfunction, systemic inflammation, and coagulation disturbances. The pathogenesis begins with defective trophoblast invasion, which prevents proper remodeling of the maternal spiral arteries. This results in high- resistance, poorly perfused vessels, leading to placental hypoxia and oxidative stress. In response, the placenta releases anti-angiogenic factors such as soluble fms-like tyrosine kinase-1 (sFlt-1) and soluble endoglin (sEng), which disrupt endothelial function by reducing the availability of vascular endothelial growth factor (VEGF) and placental growth factor (PIGF). The imbalance between pro-angiogenic and anti-angiogenic factors leads to widespread maternal endothelial dysfunction, increased vascular permeability, and vasoconstriction, ultimately causing hypertension and multi-organ involvement.

Maternal vascular dysfunction plays a crucial role in the progression of pre-eclampsia. Women with this condition exhibit heightened sensitivity to vasoconstrictors like endothelin-1, thromboxane A2, and angiotensin II, while vasodilators such as nitric oxide and prostacyclin are significantly reduced. This vascular imbalance contributes to sustained hypertension, renal impairment, and cerebral edema, increasing the risk of seizures in cases of eclampsia. Additionally, pre-eclampsia is associated with systemic inflammation due to an exaggerated immune response. Elevated levels of pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-a}, interleukin-6 (IL-6), and interleukin-1 (IL-1) exacerbate endothelial damage, while activated monocytes and neutrophils further contribute to oxidative stress and vascular dysfunction.

Another key feature of pre-eclampsia is the disruption of normal coagulation pathways, leading to a hypercoagulable state. Increased platelet aggregation at sites of endothelial injury results in thrombocytopenia, which serves as a clinical marker of disease severity. Additionally, the coagulation cascade becomes dysregulated, with increased thrombin generation and fibrin deposition, predisposing patients to Disseminated Intravascular Coagulation (DIC). In severe cases, pre-eclampsia can progress to HELLP syndrome (Hemolysis, Elevated Liver Enzymes, Low Platelet Count), further worsening coagulation abnormalities and increasing the risk of hemorrhage and maternal mortality.

Oxidative stress also plays a significant role in the pathogenesis of pre-eclampsia. The hypoxic placenta generates excessive Reactive Oxygen Species (ROS), leading to endothelial damage, lipid peroxidation, and reduced Nitric Oxide bioavailability. These factors collectively impair vascular relaxation, promote systemic inflammation, and exacerbate hypertension. Given the multifactorial nature of pre- eclampsia, early identification of at-risk patients through monitoring biomarkers such as sFlt-1, PlGF, platelet count, and D-dimer is essential for timely intervention.

Understanding the interplay between placental insufficiency, endothelial dysfunction, immune dysregulation, and coagulation abnormalities can help in developing targeted therapies such as low-dose aspirin during antenatal period, antioxidant supplementation, and angiogenic factor modulation to prevent disease progression and improve maternal and fetal outcomes.

Vascular changes in preeclampsia

Pre-eclampsia is primarily a vascular disorder characterized by widespread endothelial dysfunction, impaired vasodilation, increased vascular resistance, and

abnormal placental circulation. These vascular changes contribute to the hypertension, proteinuria, and multi-organ complications observed in affected women. The main vascular alterations in pre-eclampsia include defective placental remodeling, endothelial dysfunction, altered vascular reactivity, and increased capillary permeability.

One of the hallmark vascular abnormalities in pre-eclampsia is defective placental vascular remodeling. In a normal pregnancy, the maternal spiral arteries undergo extensive remodeling through trophoblast invasion, transforming from high-resistance, narrow vessels to large, low-resistance channels that ensure adequate blood flow to the placenta. However, in pre-eclampsia, this remodeling is incomplete, resulting in narrow, high-resistance vessels that lead to placental ischemia and hypoxia. The ischemic placenta releases anti-angiogenic factors such as soluble FMS-like tyrosine kinase-1 (sFIt-1) and soluble endoglin (sEng), which inhibit the effects of vascular endothelial growth factor (VEGF) and placental growth factor (PIGF). This disruption impairs blood vessel growth and function, exacerbating maternal endothelial dysfunction.

Endothelial dysfunction is a key feature of pre-eclampsia, leading to increased vascular permeability, impaired nitric oxide production, and systemic inflammation. The normal balance between vasodilators (such as nitric oxide and prostacyclin) and vasoconstrictors (such as thromboxane A2 and endothelin-1) is disrupted, resulting in a state of heightened vasoconstriction. This contributes to the development of hypertension and reduced organ perfusion, affecting the kidneys, liver, brain, and placenta. The increased sensitivity to vasoconstrictors like angiotensin II further worsens maternal hypertension, reducing blood flow to vital organs.

Another significant vascular change in pre-eclampsia is increased capillary permeability, which contributes to edema and proteinuria. The dysfunctional endothelium allows plasma proteins and fluids to leak into the interstitial spaces, leading to swelling, particularly in the face, hands, and lower extremities. In the kidneys, this vascular leakage results in proteinuria, a defining feature of pre-eclampsia.

The cerebrovascular system is also affected in pre-eclampsia, increasing the risk of complications such as eclampsia and stroke. The combination of hypertension, endothelial dysfunction, and vascular permeability can lead to cerebral edema, vasospasm, and hemorrhage, predisposing patients to seizures. This vascular instability is a major contributor to eclampsia, the most severe complication of pre-eclampsia.

Given these profound vascular changes, early identification and management of pre-eclampsia are essential. Monitoring biomarkers such as sFIt-1, PIGF, and endotheIin-1, along with Doppler ultrasound assessments of uteroplacental blood flow, can help predict the severity of vascular dysfunction. Preventive measures such as low-dose aspirin therapy, antihypertensive treatment, and magnesium sulfate prophylaxis can help mitigate the vascular effects of pre-eclampsia and reduce the risk of maternal and fetal complications.

Coagulation profile — derangements

Due to endothelial dysfunction, platelet activation, and increased clotting factor intake, pre-eclampsia causes hypercoagulability. Early and significant haematological abnormalities in pre-eclampsia include thrombocytopenia, when platelet counts decrease below 150,000/mcL. Increased platelet activation and aggregation at

endothelial injury sites causes platelet consumption. Progressive platelet count fall indicates illness severity, with values below 100,000/mcL indicating greater risk of consequences such HELLP syndrome, worsening coagulation dysfunction. Hepatic dysfunction and consumptive coagulopathy may be indicated by extended prothrombin time (PT) and activated partial thromboplastin time (aPTT) in severe instances of thrombocytopenia. Since most coagulation factors are synthesized in the liver, pre-eclampsia 's hepatic impairment reduces clotting factor production, exacerbating hemorrhagic consequences. In extreme situations, hypofibrinogenemia—low fibrinogen levels—occurs. Fibrinogen levels generally rise during pregnancy as an acute-phase reactant. However, a considerable decrease (<200 mg/dL) suggests development towards disseminated intravascular coagulation (DIC), a life-threatening disease characterized by clot formation and haemorrhage.

Elevated D-dimer levels suggest excessive clot formation and disintegration in pre-eclampsia, another coagulation disruption sign. Despite normal D-dimer levels rising throughout pregnancy, severe pre-eclampsia suggests continuous intravascular coagulation, increasing the risk of placental abruption, multi-organ failure, and postpartum haemorrhage. In severe situations, DIC causes microthrombi throughout the circulation, which inhibits blood flow to important organs and causes extensive bleeding. Due of these severe coagulation abnormalities, pre-eclampsia patients must have their platelet count, PT-INR/ aPTT, fibrinogen, and D-dimer levels monitored regularly to identify coagulation failure. Early detection of anomalies permits platelet transfusion, plasma replacement, and early birth, decreasing maternal and fetal problems. Pre-eclampsia-induced coagulation problems increase morbidity and mortality, therefore thorough observation and timely therapy are essential for safe pregnancy.

Management of preeclampsia

The management of pre-eclampsia focuses on early diagnosis, monitoring, and timely interventions to prevent complications for both the mother and fetus. The primary goal is to control hypertension, prevent seizures, and manage coagulation abnormalities to avoid severe outcomes like eclampsia, organ failure, or fetal death. Blood pressure management is essential in pre-eclampsia. Antihypertensive medications such as labetalol, nifedipine, or methyldopa are commonly used to lower blood pressure to target levels (usually <140/90 mmHg), which helps reduce maternal risks, including stroke or organ damage. However, treatment should be carefully balanced to avoid causing placental hypoperfusion. Magnesium sulfate is administered as seizure prophylaxis, particularly in severe cases or when the risk of progression to eclampsia is high. The management of coagulation abnormalities is also critical. Routine monitoring of platelet count, fibrinogen levels, and D-dimer helps assess the risk of Disseminated Intravascular Coagulation (DIC). In cases of severe thrombocytopenia or coagulation abnormalities, transfusions of platelets or fresh frozen plasma (FFP) may be required.

Delivery remains the definitive treatment for pre-eclampsia. The timing of delivery depends on the severity of the condition, gestational age, and fetal well-being. If the mother's condition is stable and the pregnancy is less than 34 weeks, corticosteroids are given to accelerate fetal lung maturity, and delivery is usually planned between 34-37 weeks. In severe cases, such as eclampsia or HELLP syndrome, immediate delivery is often necessary, regardless of gestational age, to protect the mother's health. In addition to medical management, antenatal care plays a crucial role in early detection of pre-eclampsia. Regular prenatal visits, blood pressure monitoring, and urine protein screening can help identify high-risk women early, allowing for timely

interventions and improving outcomes. Lastly, postpartum care is equally important as pre-eclampsia can persist or even worsen after delivery. Blood pressure should continue to be monitored closely for at least 72 hours postpartum, and appropriate antihypertensive therapy should be continued until blood pressure normalizes. Women with a history of pre-eclampsia should be advised about the higher risk of developing the condition in future pregnancies.

Past Studies

Research done by Sneha Tadu (2023) highlighted that pre-eclampsia complicates 5—10% of pregnancies, with a higher risk of complications in developing countries. The study compared the coagulation profile among normotensive, mild, and severe pre-eclampsia patients, revealing significantly lower platelet counts, prolonged bleeding times, and elevated D-dimer levels in pre-eclampsia cases. It emphasized the importance of early detection to prevent severe outcomes and recommended using additional parameters such as thrombin time, fibrinogen levels, and fibrinopeptide A for more accurate diagnosis and management of coagulation failure in pre-eclampsia and eclampsia patients.

A study conducted by Namita Bhutani et al. (2022) focused on pregnancy-induced hypertension (PIH), which is a leading cause of maternal and perinatal morbidity and mortality worldwide. The study highlighted the association of hypercoagulability with hypertensive disorders, particularly pre-eclampsia. The aims of the study were to compare platelet parameters and coagulation profiles among normotensive pregnant women, those with gestational hypertension, and pre- eclampsia patients. The findings emphasized the distinct differences in coagulation markers between these groups, underscoring the importance of coagulation profile monitoring in managing hypertensive disorders during pregnancy to prevent complications.

The study by Jayashree et al. (2021) investigated coagulopathy complications in severe preeclampsia and eclampsia, focusing on platelet count and its predictive value for coagulation disorders. The research demonstrated that thrombocytopenia and biochemical coagulopathy were commonly observed in eclampsia patients, especially those with platelet counts below 80,000 cells/mcI. This platelet count was found to be critical in predicting coagulopathy, which contributed to increased maternal and perinatal morbidity. The study emphasized the importance of monitoring platelet count in severe preeclampsia and eclampsia to identify patients at risk of severe coagulation failure, thus potentially reducing complications.

A study conducted by Asha (2015) analyzed maternal and perinatal outcomes in cases of eclampsia, reporting that the condition was associated with high maternal and perinatal morbidity and mortality. The study emphasized the critical need for improved prenatal care and more aggressive management strategies for eclampsia, highlighting the importance of early detection and intervention.

Research done by Redman and Staff (2015) examined the significance of biomarkers such as soluble fms-like tyrosine kinase-1 (sFlt-1) in understanding and predicting pre-eclampsia. Their findings demonstrated that elevated levels of these biomarkers could predict the onset of pre-eclampsia weeks before clinical symptoms appear, suggesting a crucial window for preventive strategies to mitigate the disease's severity.

Research done by Varunashree (2015) investigated thrombocytopenia in pregnancy and its impact on maternal and fetal outcomes. The study found that thrombocytopenia was prevalent among a significant number of women with hypertensive disorders and was associated with an increased risk of hemorrhage during delivery and adverse neonatal outcomes. These findings underscored the need

for comprehensive platelet monitoring in pregnant women, particularly those with hypertension.

Research done by Magee and colleagues (2014) provided a comprehensive review of the diagnosis, evaluation, and management of hypertensive disorders in pregnancy. The study detailed evidence-based approaches for effectively managing these conditions, emphasizing the importance of integrated care that includes timely diagnosis, appropriate use of antihypertensive medications, and careful consideration of delivery timing to optimize maternal and fetal outcomes.

A study conducted by Lowe et al. (2014) introduced the SOMANZ guidelines, providing detailed protocols for the management of hypertensive disorders in pregnancy. The guidelines emphasized a balanced approach that prioritizes both maternal and fetal health, advocating for the use of magnesium sulfate for seizure prophylaxis in severe pre-eclampsia and underscoring the importance of continuous fetal monitoring in high-risk pregnancies.

The study by Nirmala et al. (2015) examined the coagulation profile in pregnancy-induced hypertension (PIH), which includes gestational hypertension (GH), preeclampsia, and eclampsia. The study evaluated various coagulation parameters such as prothrombin time (PT), activated partial thromboplastin time (aPTT), and D-dimer levels in 100 cases of PIH. The findings showed that patients with severe preeclampsia had significantly higher levels of aPTT and D-dimer, suggesting a higher risk of coagulopathy. The study concluded that raised aPTT and D-dimer levels are significant indicators of severe preeclampsia, and their monitoring is crucial for initiating aggressive treatment to reduce maternal and perinatal morbidity and mortality.

The study conducted by Chaware et al. (2015) explored the coagulation profiles in different stages of pregnancy-induced hypertension (PIH), focusing on mild preeclampsia, severe preeclampsia, and eclampsia. The research involved 120 patients and compared their coagulation parameters, including platelet count, bleeding time (BT), clotting time (CT), prothrombin time (PT), and activated partial thromboplastin time (aPTT) with a control group of 45 healthy pregnant women. The results revealed significant alterations in the coagulation profile, with severe preeclampsia and eclampsia showing a marked decrease in platelet count and significant prolongation in coagulation times, indicating the presence of intravascular coagulation. This study emphasizes the importance of coagulation monitoring in hypertensive disorders of pregnancy to detect and manage potential complications like disseminated intravascular coagulation (DIC)

A comparative study conducted by Mushtaque (2013) examined the coagulation profile across normal pregnancy, pre-eclampsia, and eclampsia, revealing marked differences in coagulation markers, particularly in patients with eclampsia who exhibited significantly elevated levels of prothrombin time and partial thromboplastin time. This thesis reinforced the concept of pre-eclampsia and eclampsia as prothrombotic states and underscored the need for vigilant hematological monitoring in managing these conditions.

Research done by Prakash (2013) assessed the hematological profiles of newborns from mothers with hypertensive disorders, finding that newborns of eclamptic mothers had lower hemoglobin levels and higher rates of neutropenia. This study provided critical data on the hematological impact of gestational hypertension on neonates, suggesting that these children might require additional monitoring and care post-birth to mitigate potential health complications.

Research done by Savadi (2013) investigated the implications of elevated serum lipoprotein (a), acute-phase proteins, and serum electrolytes in pre-eclampsia. The findings indicated that elevated lipoprotein (a) levels were strongly correlated with the severity of pre-eclampsia, suggesting that these biomarkers could serve as early indicators of adverse outcomes and potential targets for therapeutic interventions to reduce complications.

Research done by Luci M. Dusse (2011) examined pre-eclampsia as a multisystem disorder characterized by hypertension and proteinuria, with endothelial dysfunction playing a key role in its pathogenesis. The study highlighted the association between pre-eclampsia, abnormal haemostatic activation, and inflammation, suggesting that these factors contribute to disease progression. Despite extensive research, no reliable screening test or definitive treatment exists to prevent complications. The study emphasized that understanding the interaction between haemostasis and inflammation could help in developing primary preventive measures and targeted therapies at an early stage, potentially improving maternal and fetal outcomes in high-risk pregnancies. A study conducted by Leduc et al. (1992) investigated the relationship between platelet count and coagulation abnormalities in women with severe preeclampsia and chronic hypertension with superimposed preeclampsia. The study found that a normal platelet count reliably predicts the absence of clinically significant clotting abnormalities. However, when platelet counts were below 150,000/mcL, abnormalities such as low fibrinogen levels and prolonged prothrombin time (PT) or partial thromboplastin time (PTT) were observed. The study concluded that monitoring platelet count at admission is an excellent predictor of subsequent thrombocytopenia, while PT, PTT, and fibrinogen levels should be reserved for cases with platelet counts below 100,000/mcL.

Research done by Bewley (1990) utilized Doppler ultrasound to investigate placental blood flow in the second trimester as a predictive measure for pre-eclampsia and fetal growth retardation. The study found that abnormal placental blood flow was significantly associated with the development of pre-eclampsia and related outcomes. This early use of Doppler technology in pregnancy highlighted its potential as a non-invasive tool for early detection of high-risk pregnancies, paving the way for its routine use in prenatal screenings today.

A study conducted by McKillop (1977) explored the relationship between soluble fibrinogen-fibrin complexes and pre-eclampsia, providing early insights into the coagulopathies associated with hypertensive disorders of pregnancy. The research highlighted that patients with pre-eclampsia showed significantly higher levels of these complexes compared to normal pregnancies, suggesting an activated coagulation system. This study was foundational in illustrating the hypercoagulable state induced by pre-eclampsia and laid the groundwork for future investigations into therapeutic interventions to manage coagulation disorders in pregnancy.

MATERIALS AND METHODS

4.1 Study design

The present study was a hospital based observational study- to evaluate coagulation profile in pregnancy with severe pre-eclampsia and eclampsia. This study was conducted at KLE'S Dr. Prabhakar Kore Hospital, Belagavi for a period of 12 months. Data and samples were obtained from pregnant mothers presented to labour room who had been informed about the study's purpose. Patients who expressed an interest in participating in the trial were enrolled after signing a written informed consent.

4.2 Study setting

The study was conducted at the Department of Obstetrics and Gynaecology of KLE'S Dr. Prabhakar Kore Hospital, Belagavi, Karnataka. The hospital is a clinical training facility that provides health care to the underprivileged in basic specialties.

4.3 Study period

The study was conducted for a period of 12 months (January 2024 – February 2025).

4.4 Study Population

The study population consisted of antenatal women diagnosed with severe pre-eclampsia and eclampsia presented to the labour room at the Department of Obstetrics and Gynaecology of KLE'S Dr. Prabhakar Kore Hospital, Belagavi, Karnataka during the study period fulfilling the inclusion criteria and consenting to participate in the study.

4.5 Sample Size

Sample size at 95% confidence interval and 95 %power

$$n = \frac{(z\,1-\frac{\alpha}{2}+Z1-\beta)^2\ (SD1^2+SD2^2)}{(\overline{X1}-\overline{X2})^2}$$

$n \approx 74.36$

Therefore, the required sample size is approximately 74.

$z\,1-\frac{\alpha}{2}$	=1.96
$Z1-\beta$	=0.85
$\overline{X1}$ ($PT\ \ value\ in\ severe\ pre\ eclampsia$)	=19.21
$\overline{X2}$ (PT value in eclampsia)	=20.04
SD1	=1.74
SD2	=1.86

4.6 Sampling methods

In this observational study, a consecutive sampling method was employed to select participants. This non-probability technique involves enrolling every eligible case that meets the inclusion criteria during the study period, ensuring that all relevant cases are included for comprehensive analysis. Over the 12-month study period (January 2024 to February 2025), all antenatal women diagnosed with severe pre-eclampsia or eclampsia who presented to the labor room of KLE's Dr. Prabhakar Kore Hospital, Belagavi, Karnataka, were considered for inclusion. Participants were selected consecutively until the required sample size was achieved.

4.7 Selection Criteria

4.7.1 – Inclusion criteria

Gestational age >20 weeks with

- **Severe pre-eclampsia -**BP >160/110 mmHg on 2 occasions 15 min apart or signs of maternal end organ dysfunction.

BP of >140/90 mmHg with or without proteinuria with maternal end organ dysfunction. (placental insufficiency- fetal growth restriction)

- **Eclampsia** - pregnant females with BP >140/90 mm Hg with convulsions or coma.

4.7.2- Exclusion criteria

- Chronic hypertension.
- Gestational Hypertension.
- Pre- Existing renal disease.
- Diabetes Mellitus
- ITP, APLA, SLE
- Drugs affecting platelet count

4.8 Data Collection and sampling techniques

All antenatal women with a gestational age of > 20 weeks, diagnosed with new onset hypertension were screened for the study. Severe Pre-eclampsia was defined as a SBP>= 140 mmHg and/or DBP >= 90 mmHg with or without proteinuria with evidence of end organ damage or SBP>= 160 mmHg and/or DBP >=110 mmHg in the absence of proteinuria . Participants were also classified as severe pre -eclampsia

if any of the imminent signs such as headache, visual disturbances, epigastric pain was present along with hypertension. Patient which were presenting with generalised - tonic clonic convulsion in the absence of the other cause were diagnosed as eclampsia. After identifying study participant, written informed consent was obtained for enrollment in the study. Details of methods employed for sampling and investigation are as follows:

Data on demographic details were obtained such as age, obstetric history, last menstrual period, expected date of delivery, period of amenorrhea. Imminent signs such headache, epigastric pain, blurring of vision were asked, history of any seizure was asked in detail. Past history and previous surgical history were elicited. Height, weight, body mass index (BMI), pulse rate, blood pressure, pallor, icterus, pedal edema was noted for each of the participant. Systemic examination, per abdomen, per vaginal examination was recorded.

Obstetric ultrasound (Doppler) -results were recorded

Clinical investigation includes haemoglobin estimation, platelet count, peripheral smear, PIH profile including urea, serum creatinine, urine albumin, uric acid, LDH levels, liver function test including liver enzymes (alanine aminotransferase *[SGPT]* aspartate aminotransferase *[SGOT]* and alkaline phosphatase *[ALP]*.

Disseminated intravascular coagulation (DIC) profile including tests for D-Dimer, fibrinogen activated partial thromboplastin time (aPTT), prothrombin time/international normalized ratio (PT/INR) were sent.

Based on the clinical examination and the investigation reports decision for mode of delivery was decided, induction of labour or caesarean was done. Maternal and fetal outcomes and complications were observed and noted.

4.9 Method of estimation of Platelet count and coagulation profile

Platelet count and PT-INR, APTT, Fibrinogen, D-DIMER was estimated by standard technique using an automated analyzer. 1 ml of venous blood was collected in EDTA bulb and 2.7 ml blood was collected in citrate bulb from patient. Citrate Blood is centrifuged and after plasma is separated, respective reagents are added to the plasma samples and then analyzed in ACLTOP 550 instrument. Sample processing time is 45 mins.

Figure no.1

4.10 Statistical analysis

Analysis of the collected data was done using descriptive statics since the study was an observational study. The data obtained was coded and entered into Microsoft excel worksheet

Data is analysed using statistical software R version 4.4.0. and Microsoft Excel. Categorical variables given in the form of frequency tables. SPSS26 Software has been used.

4.11 Ethical issue and ethical clearance

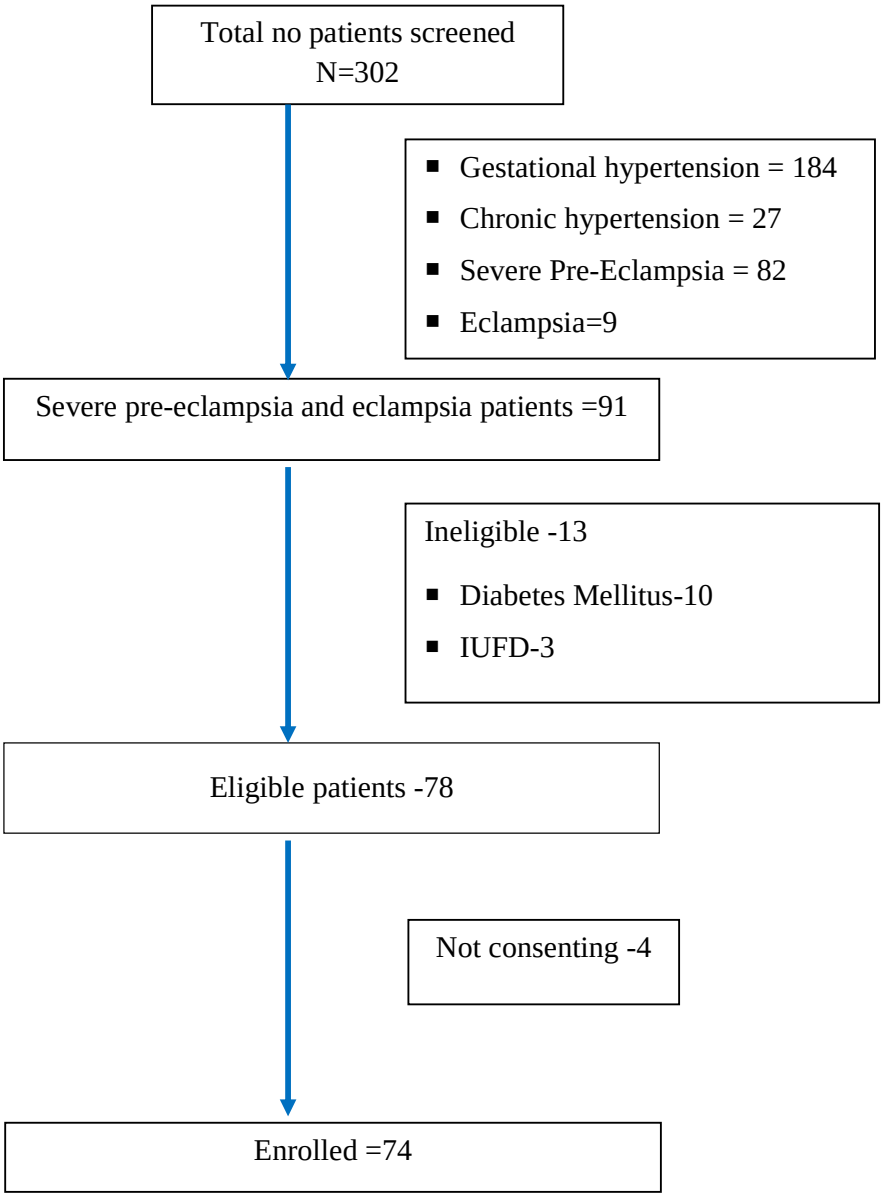
An informed choice was given by each participant based on the participant's full understanding of the method or procedure, including its characteristics, actions and possible risks and benefits. The participant's consent was sought and obtained after adequate information about all aspects covered by the study, during the process of obtaining consent, the rights to decline participation or to withdraw participation at any time of the study if they wish to do so, were emphasized. Information regarding privacy and confidentiality of the patient were provided. It was also ensured that the participants were educated about the warning signs and need for the follow- ups.

Ethical clearance for this study was obtained from the institutional ethics and research committee, KLE's Dr. Prabhakar Kore Hospital, Belagavi, Karnataka in the prescribed format.

RESULTS

RESULTS AND ANALYSIS -

Recruitment of participants -



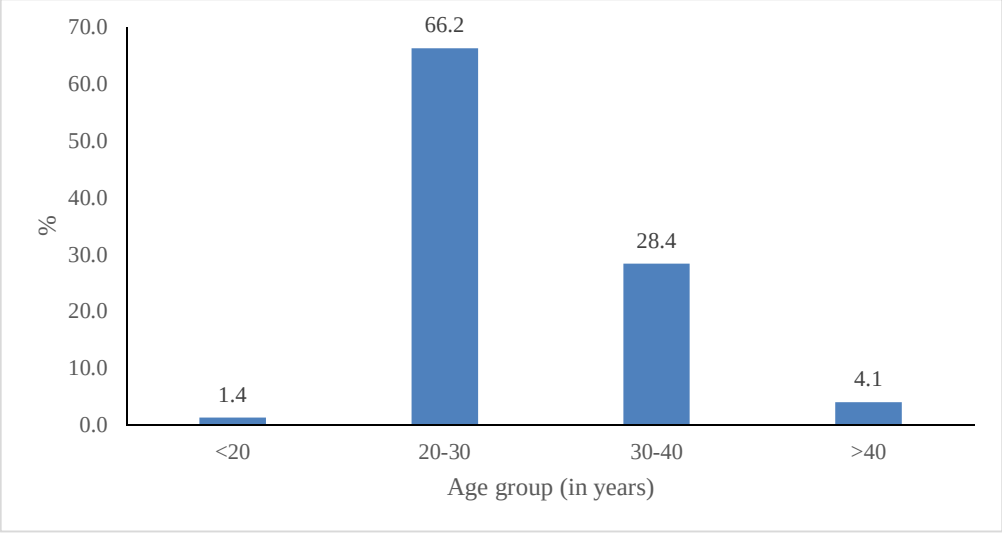
DEMOGRAPHIC DISTRIBUTION OF PARTICIPANTS

The study population comprised women of varying age groups, obstetric scores, gestational periods, and body mass index (BMI) classifications.

Table no.1 Distribution of participant according to age

Age (in years)	n	%
<20	**1**	**1.4**
20-30	**49**	**66.2**
30-40	**21**	**28.4**
>40	**3**	**4.1**

The majority of participants (66.2%) were aged between 20 and 30 years, followed by 28.4% in the 30–40 age group, while a smaller proportion was either below 20 years (1.4%) or above 40 years (4.1%).

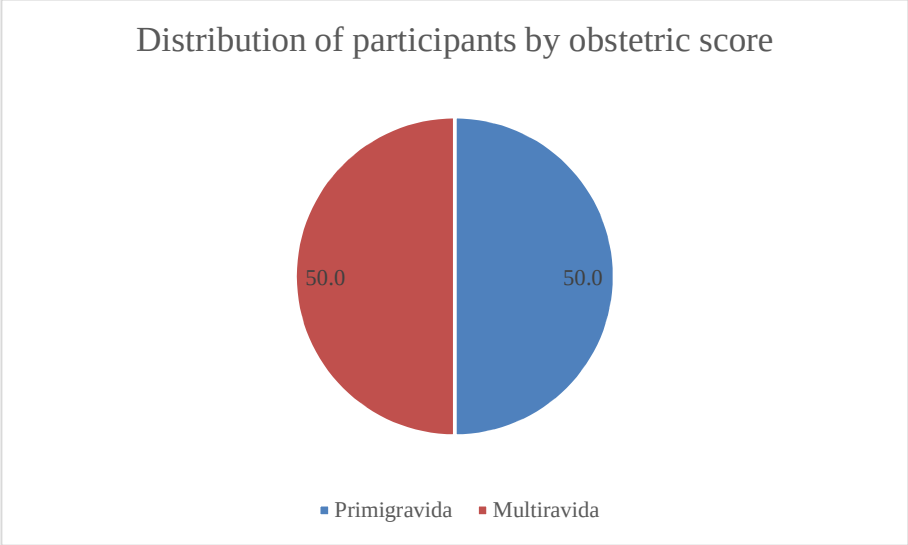


Graph 1 : Age wise distribution of participants

Table 2 Distribution of participants according to obstetric score.

Obstetric Score	n	%
Primigravida	**37**	**50.0**
Multigravida	**37**	**50.0**

Obstetric history was evenly distributed, with 50% being primigravida (first pregnancy) and 50% multigravida (having previous pregnancies).

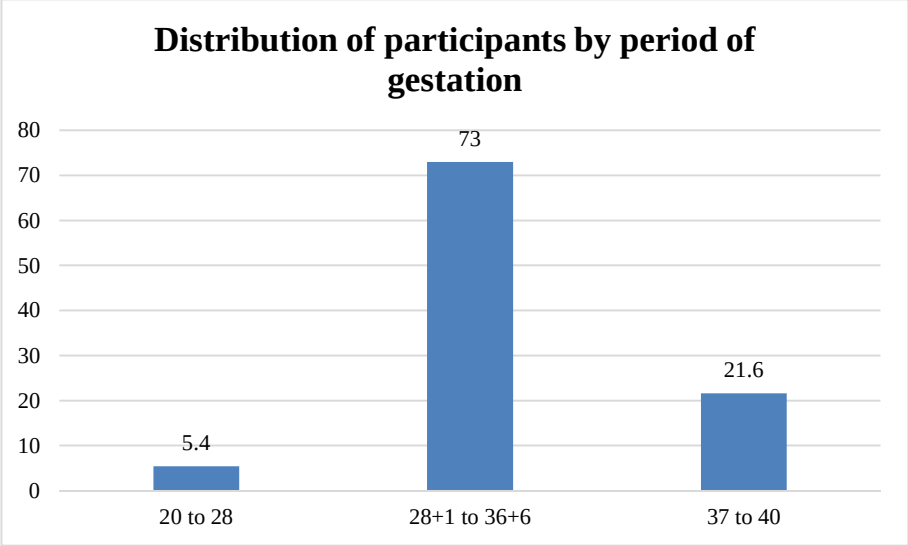


Graph 2 Distribution of participants by obstetric score

Table 3: Distribution of participants according to period of gestation.

Period Of Gestation (in weeks)	n	%
20 to 28	**4**	**5.4**
28+1 to 36+6	**54**	**73.0**
37 to 40	**16**	**21.6**

Regarding the period of gestation, most participants (73.0%) were in the 28+1 to 36+6week range, followed by 21.6% in the 37 to 40-week period, and a minority (5.4%) in the early gestational phase of 20–28 weeks. This indicates that a significant proportion of the study population was in the late second or third trimester of pregnancy.

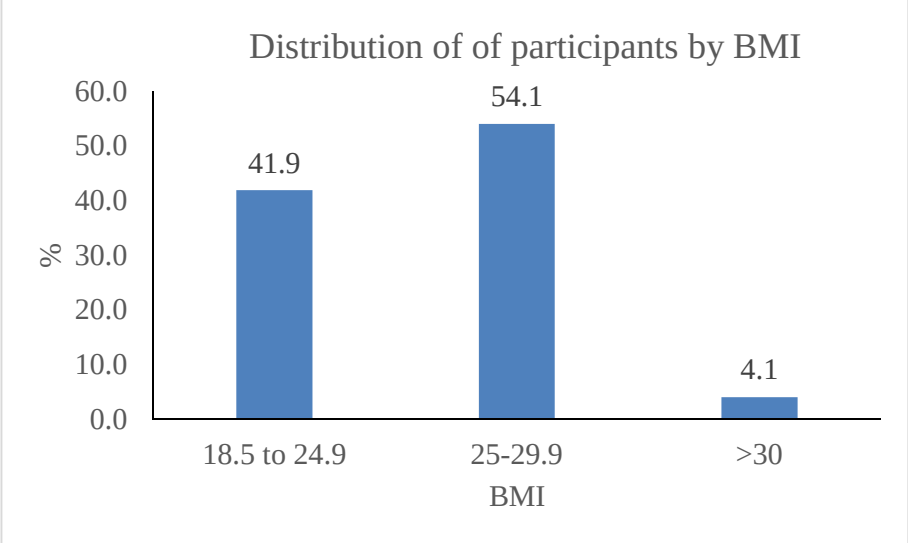


Graph 3 Distribution of participants by period of gestation

Table 4: Distribution of participants according to BMI (kg/m^2)

BMI group in kg/m^2	n	%
18.5 to 24.9	**31**	**41.9**
25-29.9	**40**	**54.1**
>30	**3**	**4.1**

In terms of BMI classification, 54.1% of participants were overweight (BMI 25–29.9 kg/m²), while 41.9% had a normal BMI (18.5–24.9 kg/m²), and only 4.1% were categorized as obese (BMI >30 kg/m²).

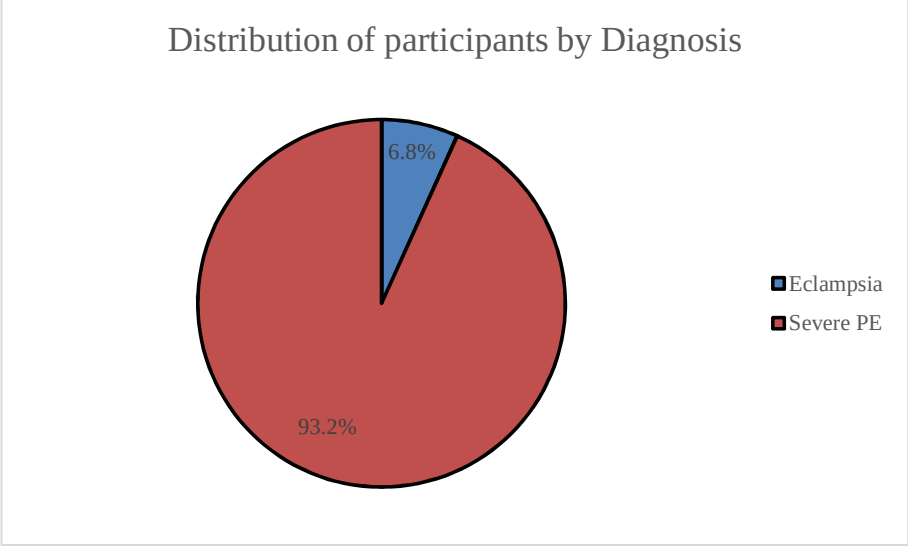


Graph 4 Distribution of participants by BMI

Table 5 Distribution of participants by Diagnosis

Diagnosis	n	%
Eclampsia	**5**	**6.8%**
Severe PE	**69**	**93.2%**
Total	**74**	**100%**

In the study, 93.2% of participants were diagnosed with severe pre-eclampsia, and 6.8% with eclampsia.



Graph 5 Distribution of participants by Diagnosis

Association of Demographic and Clinical Parameters with Diagnosis

In terms of age distribution, the majority of patients diagnosed with eclampsia (80%) and severe PE (65.2%) were in the 20–30 years age group. However, the association between age and diagnosis was not statistically significant ($p = 1.000$).

Regarding obstetric history, 60% of eclampsia cases and 49.3% of severe PE cases were among primigravida women, whereas 40% of eclampsia cases and 50.7% of severe PE cases were seen in multigravida women.

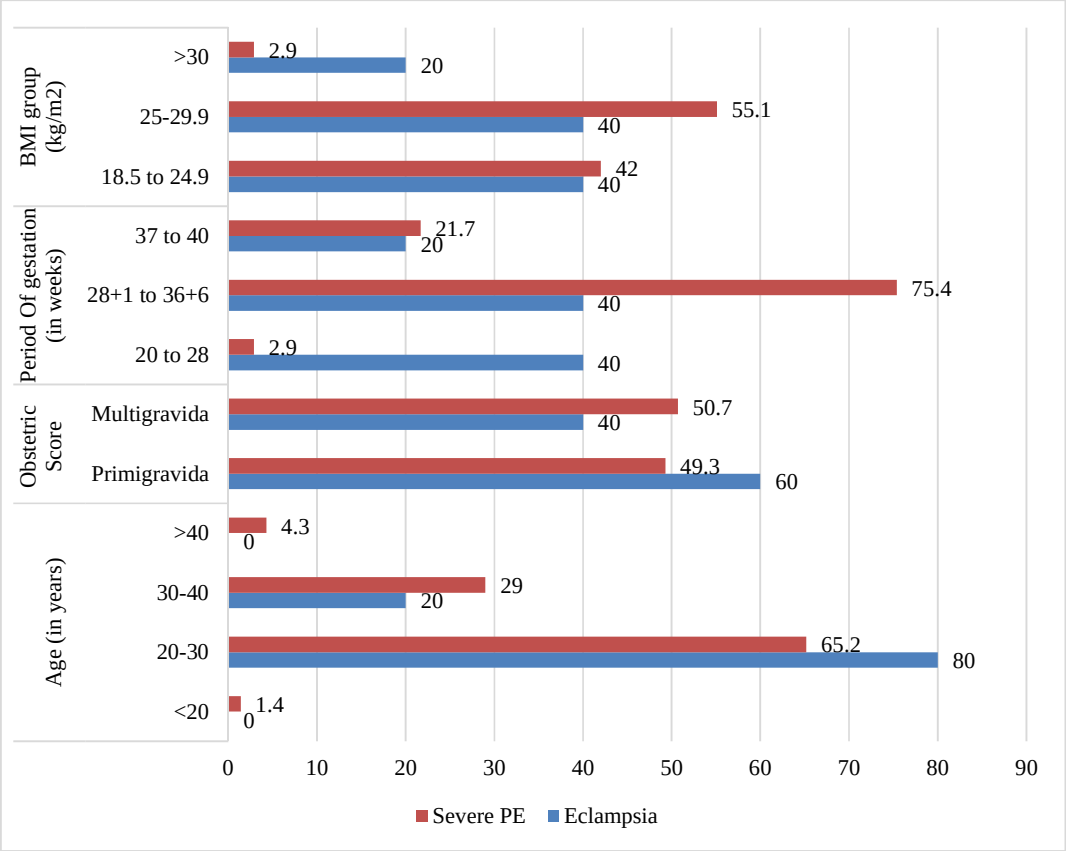
The period of gestation showed a statistically significant association with diagnosis ($p = 0.026$). Notably, 40% of eclampsia cases occurred between 20–28 weeks, compared to only 2.9% of severe PE cases in this gestational period. Similarly, 40% of eclampsia cases were between 28+1 and 36+6 weeks, compared to 75.4% of severe PE cases. Only 20% of eclampsia cases were observed beyond 37 weeks, compared to 21.7% of severe PE cases.

Analysis of BMI groups revealed no statistically significant association with diagnosis ($p = 0.243$). Among eclampsia cases, 40% had a BMI of 18.5–24.9 kg/m² (normal weight), another 40% were overweight (BMI 25–29.9 kg/m²), and 20% were obese (BMI >30 kg/m²). In severe PE cases, 42% had a normal BMI, 55.1% were overweight, and only 2.9% were obese.

Table 6 Association of clinical parameters by diagnosis (Severe pre-eclampsia and eclampsia).

		Diagnosis				Chi-square (p value)
		Eclampsia		Severe PE		
		n	%	n	%	
Age (in years)	<20	0	0.0	1	1.4	1.622 (1.000)
	20-30	4	80.0	45	65.2	
	30-40	1	20.0	20	29.0	
	>40	0	0.0	3	4.3	
Obstetric Score	Primigravida	3	60.0	34	49.3	-
	Multigravida	2	40.0	35	50.7	
Period Of gestation (in weeks)	20 to 28	2	40.0	2	2.9	7.538 (0.026) *
	28+1 to 36+6	2	40.0	52	75.4	
	37 to 40	1	20.0	15	21.7	
BMI group ($kg/m^{2)}$	18.5 to 24.9	2	40.0	29	42.0	3.326 (0.243)
	25-29.9	2	40.0	38	55.1	
	>30	1	20.0	2	2.9	
	Total	5	100	69	100	
	Mean$\pm SD$	26.32$\pm$3.13		25.93$\pm$2.27		
	Mean difference	0.387				
	t	0.359				
	p value	0.721				

***<0.05 significance is obtained by fisher exact test *<0.05 p-value obtained by independent sample t test**



Graph 6 : Association of clinical parameters with Severe Pre-eclampsia and Eclampsia.

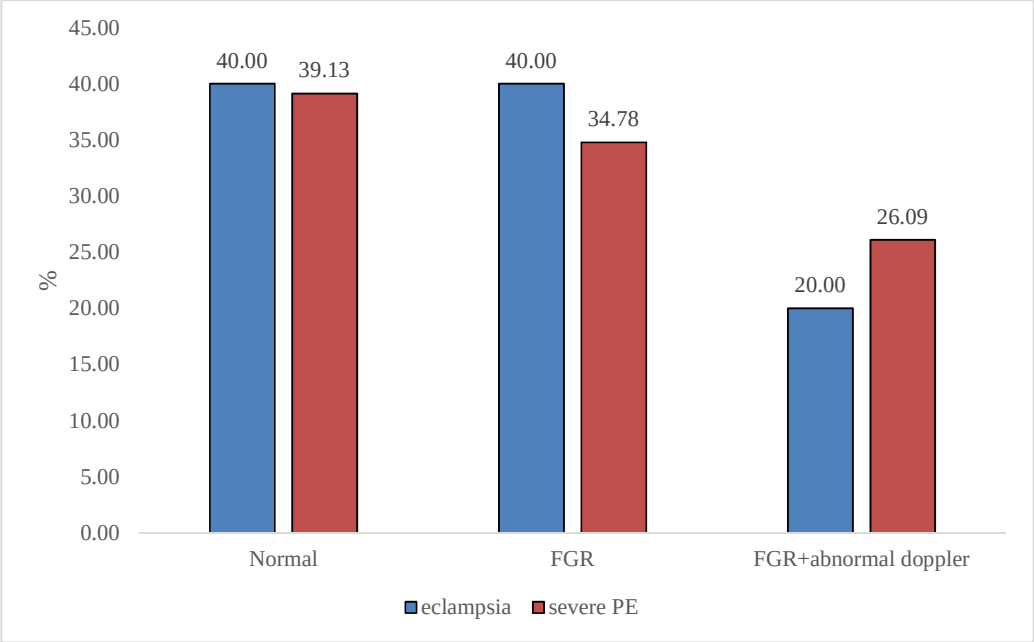
Table 7 -Distribution of patients by USG finding

Among eclampsia cases, 40 percent had normal foetal growth, while an equal proportion (40 percent) experienced foetal growth restriction (FGR). Additionally, 20 percent of eclampsia cases had FGR with abnormal Doppler findings.

In the severe PE group, 39.13 percent of cases had normal foetal growth, 34.78 percent had FGR, and 26.08 percent had FGR with abnormal Doppler. The total distribution across both groups showed that 39.19 percent of all cases had normal foetal growth, 35.14 percent had FGR, and 25.68 percent had FGR with abnormal Doppler findings

Table 7 Distribution of participants by USG finding

USG finding	Eclampsia		severe PE		Total	
	n	%	N	%	n	%
Normal growth and doppler	2	40	27	39.13	29	39.19
FGR	2	40	24	34.78	26	35.14
FGR+ abnormal doppler	1	20	18	26.08	19	25.68
Total	5	100	69	100	74	100.00



Graph 7 Distribution of participants by USG findings

DATA Analysis of laboratory parameters

Table 8 Comparison of platelet count by diagnosis

The mean platelet count in eclampsia patients was 235,600.00 ± 46,209.30 per microliter, whereas in severe PE patients, it was 204,579.71 ± 92,032.02 per microliter. Although the mean platelet count was higher in the eclampsia group compared to the severe PE group, the difference was not statistically significant, with a t-value of 0.743 and a p-value of 0.460. The mean difference between the two groups was 31,020.28, with a standard error difference of 41,728.24.

Similarly, the standard error of the mean values were 20,665.43 for eclampsia and 11,079.35 for severe PE.

Table 8 Comparison of platelet count by diagnosis

Diagnosis		n	Mean	Std. Deviation	Std. Error Mean	t	p value	Mean difference	Std. Error Difference
Platelet count	Eclampsia	5	235600.00	46209.30	20665.43	0.743	0.460	31020.28	41728.24
	Severe PE	69	204579.71	92032.02	11079.35	1.323	0.230	31020.28	23448.07
	Total	74	206675.68	89823.28					

p value is obtained by independent t test

Table no 9-Comparison of Hemoglobin by diagnosis

The mean hemoglobin level in eclampsia patients was 11.26 g/dL (±1.09), while in severe preeclampsia (PE) patients, it was 11.01 g/dL (±1.83). These findings suggest that hemoglobin concentrations are similar in patients with eclampsia and severe PE.

Table 9 Comparison of Hemoglobin by diagnosis

Diagnosis		n	Mean	Std. Deviation	Std. Error Mean	t	p value	Mean difference	Std. Error Difference
Hemoglobin	Eclampsia	5	11.26	1.09	.488	.299	.766	.250	.836
	Severe PE	69	11.01	1.83	.221				
	Total	74	11.03	1.79					

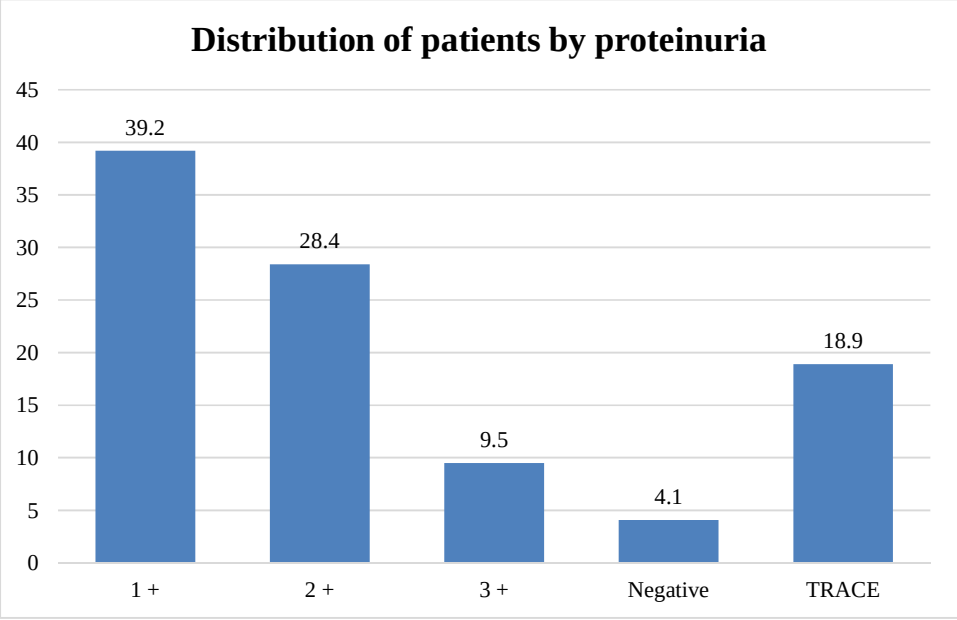
p value is obtained by independent t test

Table 10 -Distribution of participants by protenuria

The majority of cases had 1+ proteinuria (39.2%), followed by 2+ proteinuria (28.4%). Severe proteinuria (3+) was observed in 9.5% of cases, while trace proteinuria was present in 18.9% of patients. A small proportion (4.1%) had no detectable proteinuria.

Table 10 Distribution of patients by proteinuria

Proteinuria	n	%
1 +	29	39.2
2 +	21	28.4
3 +	7	9.5
Negative	3	4.1
TRACE	14	18.9



Graph 8 - Distribution of patients by proteinuria

Table 11 Comparison of coagulation profile parameter by type of diagnosis

For D-dimer levels, the mean value in eclampsia patients was 917.25 ± 723.55 ng/mL, compared to 859.16 ± 1093.39 ng/mL in severe PE cases. The mean difference was 58.086, but this was not statistically significant (p = 0.917). Fibrinogen levels were slightly higher in eclampsia patients (434.20 ± 57.75 mg/dL) compared to severe PE cases (407.62 ± 132.68 mg/dL). However, the mean difference of 26.577 mg/dL was not statistically significant (p = 0.659). For prothrombin time-international normalized ratio (PT-INR), the mean value in eclampsia patients was 0.98 ± 0.09, while in severe PE cases, it was 1.09 ± 1.11. The mean difference was -0.111, but this was not statistically significant (p = 0.825). Activated partial thromboplastin time (APTT) was also assessed, with a mean value of 0.83 ± 0.09 seconds in eclampsia patients and 3.52 ± 21.55 seconds in severe PE patients. The mean difference was -2.695 seconds, but the results were not statistically significant (p = 0.782).

Table 11 Comparison of coagulation profile parameter by type of diagnosis

Diagnosis		n	Mean	SD	Std. Error Mean	t	P value	Mean difference	Std. Error Difference
DDIMER	Eclampsia	4	917.25	723.55	361.778	.104	.917	58.086	555.862
	Severe PE	67	859.16	1093.39	133.579				
Fibrinogen	Eclampsia	5	434.20	57.75	25.829	.443	.659	26.577	60.052
	Severe PE	69	407.62	132.68	15.973				
PT-INR	Eclampsia	5	0.98	0.09	.044	-.222	.825	-.111	.501
	Severe PE	69	1.09	1.11	.134				
APTT	Eclampsia	5	.83	0.09	.041	-.278	.782	-2.695	9.703
	Severe PE	69	3.52	21.55	2.595				

Table 12 Association of Laboratory Parameters with Pre- Eclampsia: Adjusted Odds Ratios and 95% Confidence Intervals

Table 12 highlights D-dimer, Fibrinogen, PT-INR, and APTT in relation to pre-eclampsia. D-dimer has an odds ratio of 0.999, suggesting a minimal effect, with a p-value of 0.279 confirming no significant association. Similarly, Fibrinogen has an odds ratio of 1.000 and a p-value of 0.940, indicating no significant impact on pre-eclampsia risk. PT-INR, however, shows a p-value of 0.046, suggesting a statistically significant protective effect against pre-eclampsia, despite the odds ratio of 0.000 potentially indicating model issues or small sample size. Lastly, APTT has an extraordinarily high odds ratio of 79812058.160 and a p-value of 0.028, indicating a significant association with pre-eclampsia, but the extreme odds ratio and wide confidence interval suggest potential data inaccuracies or model problems.

Table 12 Association of Laboratory Parameters with Pre- Eclampsia: Adjusted Odds Ratios and 95% Confidence Intervals

Parameter	Exp(B)	95% C.I. for EXP(B)		Sig.
		Lower	Upper	
DDIMER	.999	.998	1.001	.279
Fibrinogen	1.000	.989	1.012	.940
PT-INR	.000	.000	.821	.046*
APTT	79812058.160	7.510	848151186976389.000	.028*

Table 13 Association of Laboratory Parameters with Eclampsia: Adjusted Odds Ratios and 95% Confidence Intervals

Table 13 examines D-dimer, Fibrinogen, PT-INR, and APTT in the context of eclampsia. Similar to Table 12, D-dimer and Fibrinogen show no significant impact on eclampsia risk, with p-values of 0.279 and 0.940, respectively. PT-INR shows an odds ratio of 38448.712 and a p-value of 0.046, suggesting a significant association, but the extremely high odds ratio indicates potential issues with data fit or model accuracy. APTT shows a p-value of 0.028 and an odds ratio of 0.000, suggesting a significant association with eclampsia, but, like PT-INR, the extreme value raises concerns about model validity. Overall, while PT-INR and APTT show significant associations, the extreme odds ratios and large confidence intervals suggest potential data quality issues.

Table 13 Association of Laboratory Parameters with Eclampsia: Adjusted Odds Ratios and 95% Confidence Intervals

Parameter	Exp(B)	95% C.I.for EXP(B)		Sig.
		Lower	Upper	
DDIMER	1.001	.999	1.002	.279
Fibrinogen	1.000	.988	1.011	.940
PT-INR	38448.712	1.219	1213135498.14537	.046*
APTT	.000	.000	.133	.028*

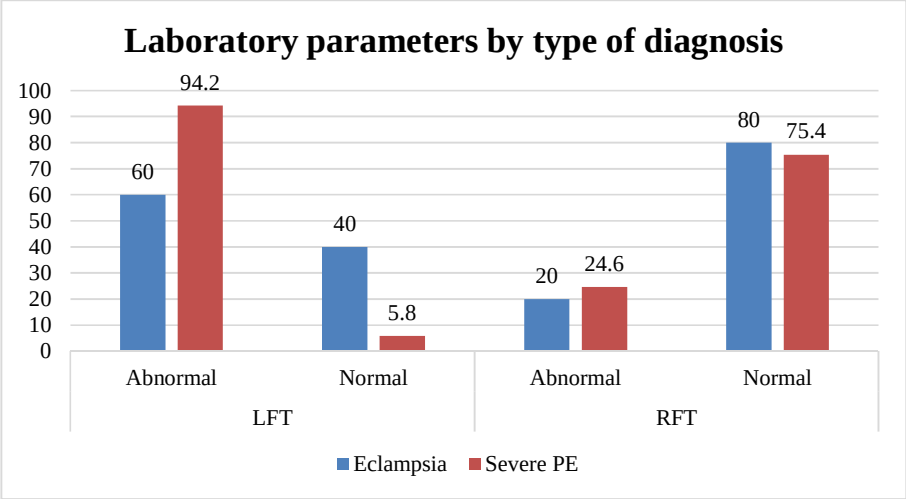
Table 14 Comparison of LFT, RFT by type of diagnosis

For LFTs (Liver Function Test), abnormal values were observed in 60.0% of eclampsia cases, compared to 94.2% of severe PE cases. The Chi-square test showed a statistically significant association (p = 0.037). Regarding RFTs (Renal Function Test), abnormal values were found in 20.0% of eclampsia cases and 24.6% of severe PE cases. The p-value was 1.000, suggesting no significant association between renal dysfunction and diagnosis.

Table 14 Comparison of laboratory parameters by type of diagnosis (Severe Pre-eclampsia and Eclampsia)

		Eclampsia		Severe PE		Total	Chi-square (P value)
		n	%	N	%	n	
LFT	Abnormal	3	60.0	65	94.2	68	4.371 (0.037) *
	Normal	2	40.0	4	5.8	6	
RFT	Abnormal	1	20.0	17	24.6	18	0.54 (1.000)
	Normal	4	80.0	52	75.4	56	

*<0.05 Significance is obtained by independent t test



GRAPH 9 Laboratory parameters by type of diagnosis

Table 15 Comparison of Lactate Dehydrogenase (LDH) Levels by Diagnosis

The mean LDH level in eclampsia patients was 417.00 ± 165.09 U/L, while in severe PE cases, it was 434.82 ± 320.05 U/L. The mean difference was -17.826 U/L, indicating slightly lower LDH levels in eclampsia patients compared to severe PE patients. However, this difference was not statistically significant (p = 0.903, t = -0.123).

Table 15 Comparison of Lactate Dehydrogenase (LDH) Levels by Diagnosis

Diagnosis		n	Mean± *SD*	Std. Error Mean	t	P value	Mean difference	Std. Error Difference
LDH	Eclampsia	5	417.00±**165.09**	73.833	-.123	.903	-17.826	145.173
	Severe PE	69	434.82±**320.05**	38.529				

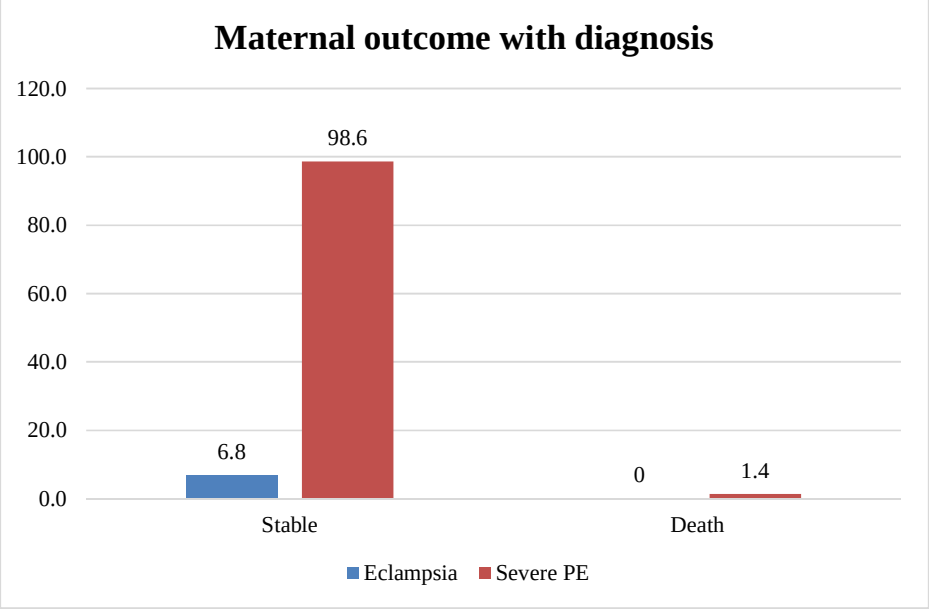
*<0.05 Significance is obtained by independent t test

Maternal and Foetal outcomes

In this study, all eclampsia patients (100%) had stable maternal outcomes, while 91.9% of severe preeclampsia (PE) patients were stable, with one death (1.4%). The chi-square statistic was 0.073 with a p-value of 1.000, indicating no significant association between diagnosis and maternal outcome. This suggests that, within this sample, both eclampsia and severe PE patients had predominantly stable outcomes, with a minimal difference in mortality rates.

Table 16 Association of Maternal outcome with diagnosis

Maternal Outcome	Diagnosis				Chi-square (P value)
	Eclampsia		Severe PE		
	n	%	n	%	
Stable	5	6.8	68	91.9	0.073 (1.000)
Death	0	0.0	1	1.4	



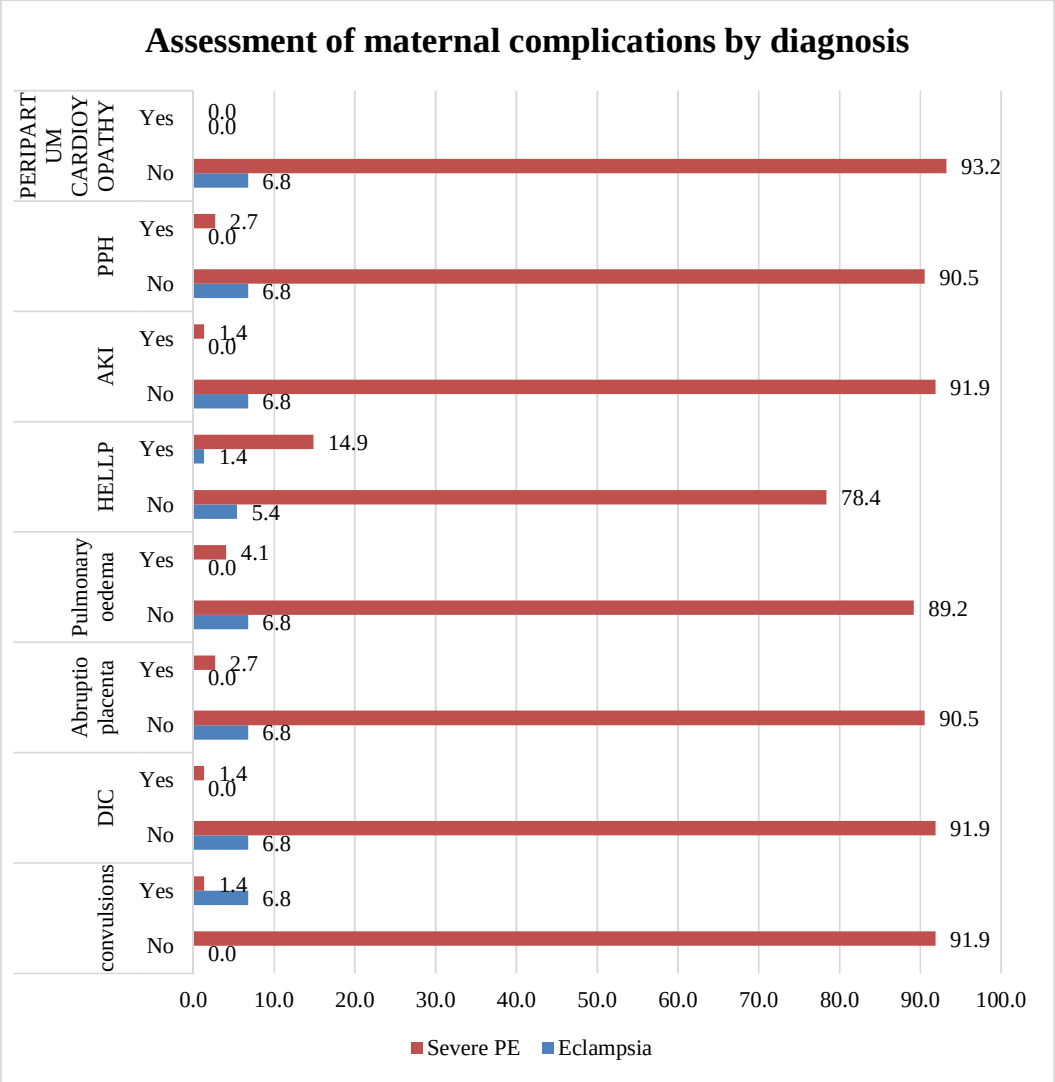
Graph 10 Maternal outcome with diagnosis

Assessment of maternal complications by diagnosis

In this study, maternal complications were assessed in patients diagnosed with eclampsia and severe preeclampsia (PE). Among eclampsia patients, 100% experienced convulsions. Disseminated Intravascular Coagulation (DIC) was occurred in 1.4% of severe PE patients. Abruptio placenta was noted in 2.7% of severe PE group. Pulmonary edema affected 4.1% of severe PE patients. HELLP syndrome was present in 14.9% of severe PE patients and 1.4% of eclampsia cases. Acute Kidney Injury (AKI) occurred in 1.4% of severe PE patients. Postpartum hemorrhage (PPH) was observed in 2.7% of severe PE patient. Peripartum cardiomyopathy was not seen in both the groups. (0%).

Table 17 Assessment of maternal complications by diagnosis

Maternal complication		Diagnosis			
		Eclampsia		Severe PE	
		n	%	n	%
Convulsions	No	0	0.0	68	91.9
	Yes	5	6.8	1	1.4
DIC	No	5	6.8	68	91.9
	Yes	0	0.0	1	1.4
Abruptio placenta	No	5	6.8	67	90.5
	Yes	0	0.0	2	2.7
Pulmonary oedema	No	5	6.8	66	89.2
	Yes	0	0.0	3	4.1
HELLP	No	4	5.4	58	78.4
	Yes	1	1.4	11	14.9
AKI	No	5	6.8	68	91.9
	Yes	0	0.0	1	1.4
PPH	No	5	6.8	67	90.5
	Yes	0	0.0	2	2.7
PERIPARTUM CARDIOYOPATHY	No	5	6.8	69	93.2
	Yes	0	0.0	0	0.0



Graph 11: Assessment of maternal complications by diagnosis

Association of Neonatal outcome by diagnosis

Among neonates born to mothers with eclampsia, 60.0% were born alive, while 40.0% were fresh still births (FSB). In contrast, in the severe PE group, 94.2% of neonates survived, with 5.8% resulting in FSB. The Chi-square test ($p = 0.037$) indicated a statistically significant association between maternal diagnosis and neonatal survival.

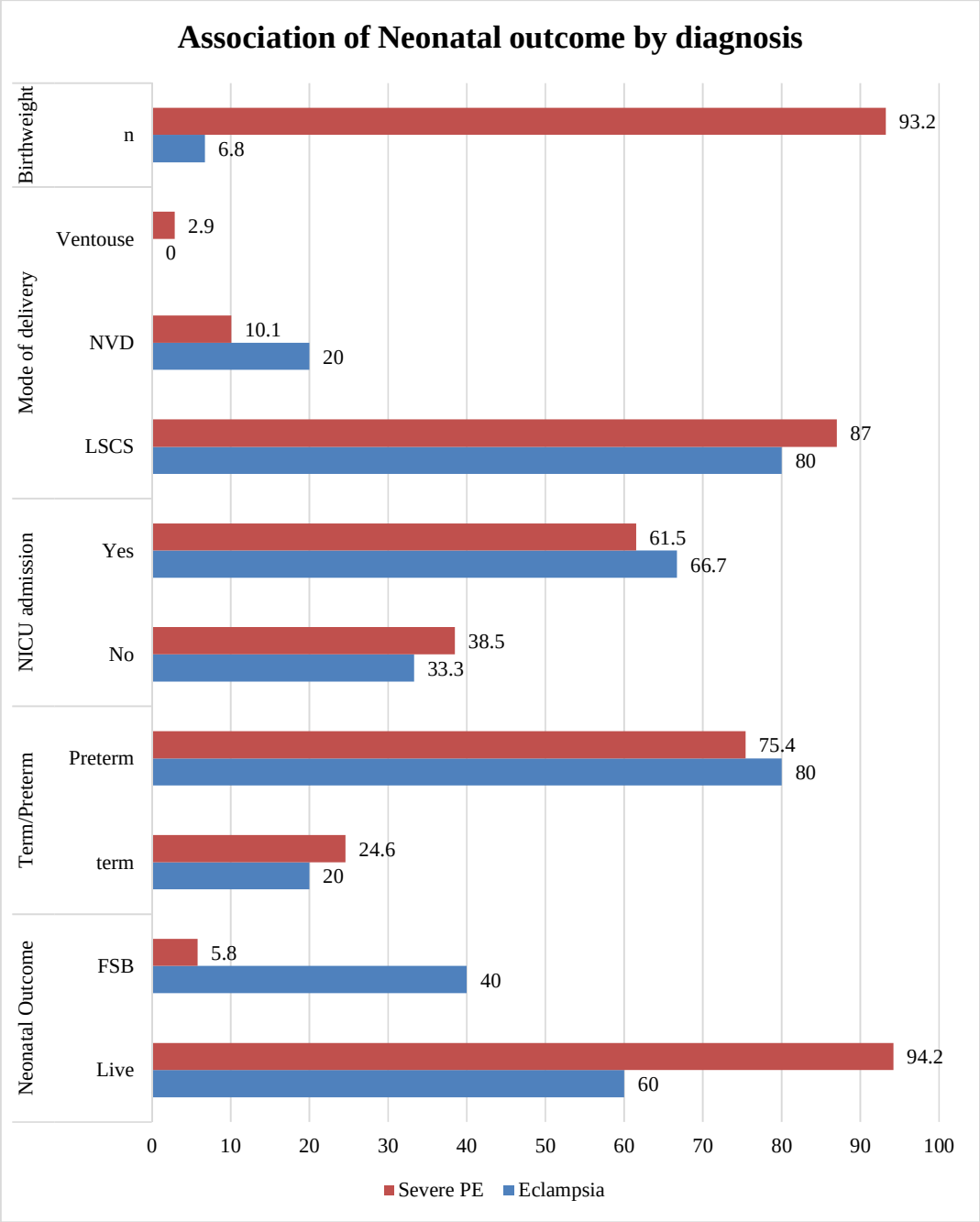
Regarding gestational age at birth, 80.0% of neonates from eclampsia cases were preterm, compared to 75.4% in severe PE cases. Term deliveries were observed in 20.0% of eclampsia cases and 24.6% of severe PE cases, with no significant association found ($p = 1.000$). In terms of mode of delivery, 80.0% of eclampsia cases and 87.0% of severe PE cases underwent lower segment caesarean section (LSCS), while 10.1% of severe PE cases and 20.0% of eclampsia cases had normal vaginal deliveries (NVD). Ventouse-assisted delivery was performed in 2.9% of severe PE cases, whereas no eclampsia cases required this intervention. The association between mode of delivery and diagnosis was not statistically significant ($p = 0.527$). The mean birth weight of neonates born to eclampsia mothers was 1.275 ± 0.75 kg, significantly lower than 1.972 ± 0.70 kg in severe PE cases. The mean difference of -0.696 kg was statistically significant ($p = 0.036$).

Table 18 Association of Neonatal outcome by diagnosis

		Eclampsia		Severe PE		Total	Chi-square (p value)
		n	%	n	%	n	
Neonatal Outcome	Live	3	60.0	65	94.2	68	4.371 (0.037) *
	FSB	2	40.0	4	5.8	6	
Term/Preterm	Term	1	20.0	17	24.6	18	0.54 (1.000)
	Preterm	4	80.0	52	75.4	56	
NICU admission	No	1	33.3	25	38.5	26	0.032 (1.000)
	Yes	2	66.7	40	61.5	42	
Mode of delivery	LSCS	4	80.0	60	87.0	64	1.533 (0.527)
	NVD	1	20.0	7	10.1	8	
	Ventouse	0	.0	2	2.9	2	
Birthweight	N	5		69			
	Mean	1.275 ± 0.75		1.972 ± 0.70			
	Mean difference	-0.696					
	T	-2.133					
	p value	0.036*					

<0.05 Significance is obtained by chi-square test

*<0.05 Significance is obtained by independent t test



Graph 12 Association of Neonatal outcome by diagnosis

DISCUSSION

In this study, as per the table no 1 the majority of participants (66.2%) were between 20 and 30 years of age, followed by 28.4% in the 30–40 age group. A smaller proportion of participants were either below 20 years (1.4%) or above 40 years (4.1%). Regarding obstetric history, the table no.2 showed the distribution was equal, with 50% being primigravida and 50% multigravida.

A study by Bhutani et al. (2022) reported that the highest prevalence of preeclampsia cases was in the 20–30 age group, aligning with the present study's findings. However, their study observed a slightly lower percentage (around 60%) in this age range, with a higher proportion (35%) in the 30–40 age group. [1]

Alemu (2017) reported a higher prevalence of severe pre-eclampsia among primigravida women (approximately 65%), which contrasts with the equal distribution observed in this study. [2]

In table no 3 of gestational age, the majority of participants (73.0%) were between 28+1 and 36+6 weeks, followed by 21.6% in the 37 to 40 weeks range. A smaller proportion (5.4%) were in the early gestational phase of 20–28 weeks. This distribution suggests that most cases occurred during the late second and third trimesters of pregnancy.

Bhutani et al. (2022) found that the majority of severe pre-eclampsia cases were diagnosed between 28 and 37 weeks of gestation, similar to the findings of this study. They reported that over 70% of cases presented in the third trimester, reinforcing the trend of late-onset preeclampsia.[1]

Tadu et al. (2023) reported a similar gestational distribution, with severe preeclampsia cases frequently occurring between 28 and 36 weeks, while early-onset cases (before 28 weeks) were relatively rare, consistent with this study's findings.[4]

Based on BMI classification seen in table no 4, the majority of participants (54.1%) were overweight (BMI 25–29.9 kg/m²), followed by 41.9% with a normal BMI (18.5–24.9 kg/m²). A smaller proportion (4.1%) fell into the obese category (BMI >30 kg/m²). The high prevalence of overweight individuals highlights the importance of monitoring maternal weight gain, as excessive weight is linked to increased pregnancy-related complications.

Bhutani et al. (2022) reported that overweight and obese women had a higher risk of developing preeclampsia, with approximately 50% of their study population classified as overweight, a finding that closely aligns with the present study. [1]

Roberge et al. (2018) emphasized that maternal obesity (BMI >30 kg/m²) is a well-established risk factor for preeclampsia, though their study observed a higher obesity rate (~10%) compared to the 4.1% found in our study. This variation may be due to differences in population demographics or lifestyle factors. [5]

While the obesity rate in this study is lower than in some reports, it remains an important factor requiring careful maternal weight management to reduce adverse pregnancy outcomes.

Among the study population, severe preeclampsia (PE) was the predominant diagnosis, accounting for 93.2% of cases, while eclampsia was observed in 6.8% of participants. Out of 74 total cases, 69 were classified as severe PE, indicating that the majority of patients experienced significant hypertension and related complications during pregnancy. Only five cases were diagnosed as eclampsia, a condition marked

by seizures in preeclamptic women, underscoring its relatively lower prevalence in this cohort.

Study by Tadu et al. (2023) highlighted that while severe PE is the most common hypertensive disorder of pregnancy, the occurrence of eclampsia remains low in tertiary care settings where monitoring and timely treatment are available, a finding that mirrors this study's results. [4] The high prevalence of severe PE in this study aligns with recent literature, indicating that it is the most frequently observed hypertensive disorder in pregnancy. The relatively lower incidence of eclampsia may reflect improved antenatal care and timely medical intervention. However, continued vigilance is necessary to prevent progression to more severe complications.

As per table 8 the mean platelet count in eclampsia patients was 235,600.00 ± 46,209.30 per microliter, whereas in severe preeclampsia (PE) patients, it was 204,579.71 ± 92,032.02 per microliter.

Jayashree & Renuka (2021) observed a progressive decline in platelet count with increasing severity of hypertensive disorders in pregnancy, noting that thrombocytopenia was more prevalent in severe PE than in eclampsia cases. [6]

Bhutani et al. (2022) reported a significantly lower mean platelet count in severe PE cases compared to normotensive pregnancies, with values declining as disease severity increased.[1]

The results from Table no 11 showed none of the values of D-Dimer, fibrinogen, aPTT, PT-INR were significant statistically.

Tadu et al. (2023) observed mild alterations in PT-INR in hypertensive pregnancies, with eclampsia cases sometimes showing lower values than severe PE cases. This

matches the present study's observation that PT-INR was slightly lower in eclampsia (0.98) than in severe PE (1.09), though the difference was not statistically significant ($p = 0.825$).[4]

Bhutani et al. (2022) reported that APTT values vary widely in severe PE due to coagulation disturbances, a trend also reflected in this study, where APTT was longer in severe PE than in eclampsia but was not statistically significant ($p = 0.782$)[1].

This study confirms that D-dimer, fibrinogen, PT-INR, and APTT levels are altered in both severe PE and eclampsia, but the differences between the two conditions are not statistically significant. The findings align with recent studies, suggesting that while coagulation abnormalities are present in hypertensive disorders of pregnancy, they may not always differ significantly between severe PE and eclampsia. This reinforces the need for individualized coagulation monitoring rather than relying solely on mean differences.

D-Dimer: The odds ratio values of D-Dimer in both severe Pre-eclampsia and eclampsia indicate no significant association between D-Dimer levels. The confidence interval, and the p-value suggests that changes in D-Dimer levels do not strongly predict severe pre-eclampsia or Eclampsia.

Bhutani et al. (2022) and Leppert & Kalliala (2021) found that D-Dimer levels increase significantly in both severe pre-eclampsia and eclampsia, indicating enhanced coagulation and fibrinolysis. Our study, however, finds no significant association ($p = 0.279$), possibly due to variation in gestational age at testing or cut-off values used.[1]

Fibrinogen: The odds ratio of fibrinogen in both severe Pre-eclampsia and eclampsia shows no significance. The wide confidence interval and high p-value reinforce the lack of a strong relationship.

Tadu et al. (2023) and Dusse et al. (2011) reported elevated fibrinogen levels in severe pre-eclampsia and eclampsia is due to increased clot formation and intravascular inflammation. Our study does not support this association ($p = 0.940$), which may be due to differences in severity of pre-eclampsia cases or sample characteristics or fibrinogen measurement methods.[4,11]

PT-INR (Prothrombin Time - International Normalized Ratio): The odds ratio, suggests a statistically significant association with severe pre-eclampsia ($p < 0.05$) and with eclampsia ($p < 0.05$). A prolonged PT-INR might indicate increased coagulation activity, supporting the hypercoagulable state seen in severe pre-eclampsia. The extremely high odds ratio suggests a potential link between prolonged PT-INR and coagulopathy in eclampsia, but the wide confidence interval suggests considerable variability in the data or outliers.

Jayashree & Renuka (2021) and Mohapatra et al. (2007) reported significant coagulation abnormalities in severe Pre-eclampsia and eclampsia, particularly prolonged PT-INR due to clotting factor consumption. [6] Our study aligns with this finding ($p = 0.046$), reinforcing the hypercoagulable state and increased risk of disseminated intravascular coagulation (DIC).

APTT (Activated Partial Thromboplastin Time): The odds ratio is extremely high (79,812,058.160) with a very broad confidence interval ($7.510 – 8.48 \times 10^{14}$), and $p = 0.028$, indicating a significant association in severe Pre-eclampsia but, suggest high variability or an outlier effect, which could limit the reliability of this result. The odds

ratio suggests a significant association with eclampsia ($p < 0.05$). The odds ratio of zero and the narrow confidence interval indicate a strong correlation, likely reflecting clotting abnormalities in eclamptic patients. Studies done by Mushtaque (2013) and Gohil & Herrmann (2019) report prolonged APTT in severe pre-eclampsia, and eclampsia is linked with defective clot formation and risk of hemorrhagic complications, HELLP syndrome.

While D-Dimer and fibrinogen do not show a significant association with pre-eclampsia, PT-INR and APTT demonstrate statistically significant relationships, supporting the coagulopathy hypothesis in pre-eclampsia and eclampsia. However, the extreme values in APTT and PT-INR suggest potential data inconsistencies or outliers. requiring further analysis with larger datasets for more reliable conclusions.

Abnormal liver function test (LFT) values were observed in 60.0% of eclampsia cases and 94.2% of severe preeclampsia (PE) cases. Regarding renal function tests (RFTs), abnormal values were found in 20.0% of eclampsia cases and 24.6% of severe PE cases.

Similar to our study, Bhutani et al. (2022) found that LFT abnormalities were significantly more common in severe PE than in eclampsia. [1]

Dusse et al. (2011) noted that renal impairment is often present in hypertensive disorders of pregnancy, but the degree of dysfunction varies and reported that renal dysfunction markers (elevated creatinine, urea) were significantly associated with worsening severe pre-eclampsia. [11]

Tadu et al. (2023) found that severe PE cases had a higher incidence of liver dysfunction than renal impairment, supporting this study's findings that LFT

abnormalities were significantly more common than RFT abnormalities in both groups. [4] .

The mean lactate dehydrogenase (LDH) level difference indicated slightly lower LDH levels in eclampsia patients compared to severe PE patients.

Bhutani et al. (2022) reported that LDH levels were elevated in both severe PE and eclampsia, with no significant difference between the two groups, which aligns with the present study's findings ($p = 0.903$). [1]

Tadu et al. (2023) reported that LDH levels above 600 U/L were associated with increased maternal and fetal complications. In this study, the mean LDH levels in both groups were below 600 U/L, suggesting a lower risk of severe complications. [4] The mean LDH levels remaining below 600 U/L suggest that most cases were not associated with severe organ dysfunction.

Across both groups, 39.19% of all cases had normal fetal growth, 35.14% had FGR, and 25.68% had FGR with abnormal Doppler findings.

Bewley (1990) demonstrated that Doppler studies could predict placental insufficiency in hypertensive pregnancies, with abnormal Doppler findings correlating strongly with FGR. The present study's finding that 20% of eclampsia cases and 26.08% of severe PE cases had FGR with abnormal Doppler supports Bewley's conclusions regarding the role of Doppler in detecting compromised placental circulation. [7]

Redman & Staff (2015) found that placental dysfunction is a hallmark of severe PE, with a significant proportion of cases exhibiting FGR. Their study reported FGR in

around 30–40% of severe PE cases, which is comparable to the 34.78% FGR rate observed in this study. [8]

Among the five eclampsia patients, all (100%) had a stable outcome, with no reported maternal deaths. Similarly, in the severe preeclampsia (PE) group, 98.6% of patients remained stable, while 1.4% (one patient) experienced maternal mortality.

Say et al. (2014) (WHO Study) estimated that hypertensive disorders contribute to 14% of maternal deaths globally, though mortality rates are significantly lower in well-equipped healthcare settings, supporting this study's low mortality rate. [9]

Magee et al. (2016) (FIGO Guidelines) emphasized that early diagnosis and treatment of severe PE and eclampsia have significantly reduced maternal mortality worldwide. This aligns with the high survival rate in this study, where 98.6% of severe PE patients had a stable outcome. [10]

The lack of a statistically significant association between diagnosis and maternal outcome (p = 1.000) suggests that both conditions, when managed properly, have comparable maternal survival rates. Based on the deranged values of the coagulation profile and PIH profile obstetrician should anticipate the complication and should manage the case aggressively.

In this study, maternal complications were assessed in patients diagnosed with eclampsia and severe preeclampsia (PE). Among eclampsia patients, 100% experienced convulsions, while 1.4% of severe PE patients also had convulsions. Disseminated Intravascular Coagulation (DIC) was absent in eclampsia cases but occurred in 1.4% of severe PE patients. Abruptio placenta was noted in 2.7% of severe PE patients, whereas none in the eclampsia group. Pulmonary edema affected 4.1% of severe PE patients; none in the eclampsia group. HELLP syndrome was

present in 14.9% of severe PE patients and 1.4% of eclampsia cases. Acute Kidney Injury (AKI) occurred in 1.4% of severe PE patients; absent in eclampsia. Postpartum hemorrhage (PPH) was observed in 2.7% of severe PE patients; none in eclampsia. These findings highlight that, severe PE patients face a broader range of complications.

Dusse et al. (2011) highlighted that DIC is a rare but serious complication of severe PE, often triggered by placental abruption or HELLP syndrome. [11] Tadu et al. (2023) reported that AKI and pulmonary edema are more prevalent in severe PE than in eclampsia, consistent with this study where 100% of AKI and pulmonary edema cases were found in severe PE patients. [4] Bhutani et al. (2022) similarly reported that convulsions were exclusive to eclampsia cases, with a small percentage (~15–20%) of severe PE patients exhibiting seizure-like activity due to cerebral edema or hypertensive crisis, aligning with this study's 16.7%. [1]

Say et al. (2014) (WHO Study) identified postpartum hemorrhage (PPH) as a significant complication in hypertensive pregnancies, particularly in cases of severe PE. This aligns with the two cases of PPH in severe PE found in this study [Say et al., 2014].

Severe PE is associated with a broader range of complications such as HELLP syndrome, AKI, pulmonary edema, and PPH. The higher prevalence of complications in severe PE compared to eclampsia reinforces the need for close monitoring and timely intervention to prevent life-threatening maternal outcomes.

Among neonates born to mothers with eclampsia, 60.0% were born alive, while 40.0% were fresh stillbirths (FSB). In comparison, in the severe preeclampsia (PE) group, 94.2% of neonates survived, with 5.8% resulting in FSB.

Regarding gestational age at birth, 80.0% of neonates from eclampsia cases were preterm, compared to 75.4% in severe PE cases. Term deliveries were observed in 20.0% of eclampsia cases and 24.6% of severe PE cases.

In terms of mode of delivery Lower segment caesarean section (LSCS) was performed in 80.0% of eclampsia cases and 87.0% of severe PE cases. Normal vaginal delivery (NVD) occurred in 20.0% of eclampsia cases and 10.1% of severe PE cases. Ventouse-assisted delivery was performed in 2.9% of severe PE cases.

The mean birth weight of neonates born to eclampsia mothers was significantly lower (1.275 ± 0.75 kg) compared to 1.972 ± 0.70 kg in severe PE cases.

Prakash (2013) found that preterm births were common in hypertensive pregnancies, with rates exceeding 70%, consistent with the 80.0% preterm rate in eclampsia cases and 75.4% in severe PE cases in this study. [12] Bhutani et al. (2022) reported higher rates of perinatal mortality in eclampsia compared to severe PE, reinforcing this study's findings that FSB was more frequent in eclampsia cases (40.0%) vs. severe PE (5.8%). [1]

Tadu et al. (2023) observed that caesarean section was the preferred mode of delivery in severe PE and eclampsia to reduce maternal and neonatal complications. The same can be seen in our study.[4]

This study confirms that eclampsia is associated with significantly higher perinatal mortality compared to severe PE. Preterm birth is common in both groups, and LSCS remains the predominant mode of delivery.

The study underscores the importance of early diagnosis, timely intervention, and close monitoring in managing severe preeclampsia and eclampsia. Severe

preeclampsia is associated with a higher incidence of maternal and neonatal complications than eclampsia, although both conditions require careful management to prevent severe outcomes.

CONCLUSION

The study provides important insights into the coagulation. While the study identified significant abnormalities in coagulation markers like PT-INR and APTT, no clear association was observed with D-Dimer and fibrinogen levels. These findings highlight the complexity of coagulation disorders in hypertensive pregnancy conditions and the need for further investigation. With this study we infer that no single modality can be taken as prognostic marker for severe pre-eclampsia and eclampsia, a Multi-Marker Modal approach is the latest go. A multi marker-modal approach allows for: earlier detection, risk stratification, monitoring disease progression, better clinical decision-making. The combination of clinical findings, biomarkers and advanced imaging techniques offers a comprehensive approach to managing preeclampsia and eclampsia. These new tools provide a more accurate and early diagnosis, leading to better risk stratification and tailored interventions.

SUMMARY

This study aimed to evaluate coagulation profile and outcomes of patients diagnosed with severe preeclampsia (PE) and eclampsia. The study found that the majority (66.2%) of participants were between 20 and 30 years of age. In terms of gestational age, most participants (73%) were diagnosed with severe preeclampsia between 28+1 and 36+6 weeks. Regarding maternal risk factors, the majority of participants had an overweight BMI (54.1%), followed by those with a normal BMI (41.9%). Overweight and obese women are known to have a higher risk of developing preeclampsia, and this study supports those findings. Most patients had stable maternal outcomes, with only one patient in the severe pre-eclampsia group experiencing maternal mortality (1.4%). This is in line with the lower mortality rates found in well-equipped healthcare settings. The study also observed that coagulation abnormalities, including altered PT-INR and APTT levels, were present in both eclampsia and severe preeclampsia. While there were differences in these values, they were not statistically significant between the two groups, suggesting that coagulation monitoring should be individualized. Similarly, liver function abnormalities were more common in severe preeclampsia cases (94.2%) than in eclampsia cases (60%), while renal function abnormalities were less frequently observed in both groups. In terms of complications, severe preeclampsia was associated with a wider range of maternal complications compared to eclampsia. These included HELLP syndrome (14.9%), pulmonary oedema (4.1%), and acute kidney injury (AKI, 1.4%), which were absent in eclampsia cases. Postpartum haemorrhage (PPH) was noted in 2.7% of severe preeclampsia cases, further highlighting the need for close monitoring in these patients. As for neonatal outcomes, eclampsia was associated with significantly higher perinatal mortality, as 40% of neonates were fresh stillbirths, compared to just 5.8%

in the severe preeclampsia group. Preterm births were common in both groups, with 80% of eclampsia cases and 75.4% of severe preeclampsia cases being preterm. Caesarean section was the most common mode of delivery in both groups. The study also highlighted the importance of early diagnosis and timely intervention in improving maternal and neonatal outcomes. The results highlight the importance of careful coagulation monitoring in hypertensive disorders during pregnancy, as coagulation abnormalities can contribute to severe complications.

There are some limitations of the study, it is conducted in single centre which may lead to biases due to the specific demographic or clinical practices at that location, relatively small sample size (74 participants), limits the generalizability of the findings to larger populations. As it is cross sectional study the population is studied for a single point of time and does not allow for the evaluation of changes over time, such as how coagulation profiles evolve with disease progression or response to treatment. Despite these limitations, the study provides valuable insights into the coagulation profiles of patients with severe PE and eclampsia and highlights the need for further research to confirm these findings and explore potential clinical applications.

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ANNEXURE – I - INFORMED CONSENT FORM

"AN OBSERVATIONAL STUDY OF COAGULATION PROFILE IN SEVERE PRE-ECLAMPSIA AND ECLAMPSIA PATIENTS."

Name of Student/Principal Investigator: Dr.

Name of Guide/Co Investigators: Dr.

Introduction: The above study is being conducted in the Department of Obstetrics and Gynaecology, under which blood samples of pregnant women with high Blood Pressure readings (hypertensive) along with headache, pain in abdomen (epigastric pain), with / without seizures are being collected. After taking permission of the subject, blood samples will be collected from them and will be sent to lab and studied for blood parameters – PT, APTT, D-Dimer, serum fibrinogen ,Platelet count. With the help of the blood report values we will be able to see how the disease will effect the blood components and the pregnancy. The details of the subject will not be disclosed, only the blood parameters values will be used.The values will be studied, analyse, described and the end result will be used for publication in journals, as research article. The subject confidentiality will be strictly maintained.

Explanation of procedure- I have been be explained about the procedure verbally in my own vernacular language. After applying tourniquet to hand ,5ml of blood will be collected. 2.9 ml of blood will be put in sodium citrate(blue) vacutainer and 1.9 ml blood will be put in EDTA (purple) vacutainer and these will be sent to lab.

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 Seeing the blood parameters, treatment will be given accordingly any alterations in management can be done. The data gathered will 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 These tests are routinely done for evaluating the pre-eclampsia and eclampsia patients. Patient is not doing any test additionally for the study. Only the parameters will be collected from the reports.

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:

Principal Investigator - Dr.

Phone no.

If you have any question or complaints with regard to your right as study participant you may 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 "**AN OBSERVATIONAL STUDY OF COAGULATION PROFILE IN SEVERE PRE-ECLAMPSIA AND ECLAMPSIA PATIENTS.**".

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 AND RECRUITMENT FORM

Screening number: ☐☐☐

Enrolment number: ☐☐☐

Date of screening (DD-MM-YYYY): ☐☐ ☐☐ ☐☐☐☐

First name: _____ Middle name: _____ Last name: _____

Age (years): ☐☐

OP/IP number: ☐☐☐☐☐☐

Husband's name: _____

Address: -_____

Phone number: _____

Gestational Age

1) Is the period of gestation above 20 weeks ☐

1-yes 2- no

BP reading – 1) ☐

2) ☐

Imminent sign – ☐

☐

Proteinuria -

2) History (1= yes, 2=no)

a) Patient with gestational hypertension, chronic hypertension, HELLP ☐

b) Preexisting renal or vascular disease ☐

c) Diabetes Mellitus ☐

d) ITP, TTP, APLA, SLE ☐

e) Intake of Drug affecting platelet count ☐

The woman is eligible to consent only if answer to 1 is yes and 2 is No ☐

Eligible ☐

Consented ☐

ANNEXURE – III - PROFORMA

An observational study of coagulation profile in severe pre-eclampsia and eclampsia patients.

Name	
Age	
Address	
Phone no	
Date of admission	
IP no.	
Registered	

COMPLAINTS AND HISTORY OF PRESENTING COMPLAINTS

Period of amenorrhea	
Imminent signs- 1.Headache 2.blurring of vision 3.epigastric pain 4.vomiting 5.seizures	 YES NO YES NO YES NO YES NO YES NO
Seizure No. of episodes	YES NO

HISTORY

Obstetric score	G P L A D
Last Menstrual Period	
EDD/CEDD	
Period of Gestation	

PAST HISTORY

Comorbidities

GENERAL PHYSICAL EXAMINATION

Height-		Weight-		BMI-	
Pulse Rate - Blood pressure-					
Pedal odema					
Systemic examination			CVS- RS-		
Per Abdominal examination					
CLINICAL DIAGNOSIS-					

INVESTIGATION-

Haemoglobin-	gm/dl
Peripheral Smear-	

PIH Profile-

Platelet count	Lakhs/cumm
LDH	U/L
Uric acid	mg/dl
RFT	
LFT	
Urine Albumin	

DIC profile -

1.D DIMER	ng/ml
2.Fibrinogen	mg/dl
3.PT/INR	
4.aPTT	

OBSTETRIC ULTRASOUND

Period of gestation	
Doppler findings	
Impression	

MANAGEMENT:

Mode of delivery-	Vaginal C-Section
	Induction Augmentation
Intrapartum Complications	Yes No
Blood Loss	
Indication for LSCS	
Intra-op findings:	

Peripartum Complications:	Yes	No
1) Convulsions		
2) DIC		
3) Abruptio placenta		
4) Peripartum cardiomyopathy		
5) Pulmonary oedema		
6) HELLP		
7) Acute kidney injury		
8) PPH		
9) Cerebro vascular complication		

ICU admission	Yes ☐ No ☐
Duration of stay	
Condition at discharge	
Cause of death if so	
Intervention if any	

PERINATAL OUTCOME

1) Term / Preterm -

2) Weight -

3) NICU admission - yes /no

ANNEXURE – IV

MASTER CHART

S.No	IP Number	Age	REGISTERED.(YES-1, NO-2)	Obstetric Score	Period Of Gestation(in weeks)	Diagnosis	Antenatal complication	Blood Pressure (mm/hg)	BMI (kg/m2)	Proteinuria	Platelet count (/mcl)	Hemoglobin (g/dL)	D-DIMER(ng/mL)	Fibrinogen(mg/dl)	PT-INR	APTT	LDH (U/L)	LFT(N-normal, ABN-anormal)	RFT(N-normal, ABN-anormal)	Serum creatinine (mg/dL)	Uric acid (mg/dL)	USG FINDING (N-normal)	INDUCED(YES-1,NO-2)	Mode of Delivery	Indication for LSCS	INTRAPARTUM COMPLICATIONS (YES-1,NO-2)	CONVULSIONS (YES-1,NO-2)	DISSEMINATED INTRAVASCULAR COAGULATION (YES-1,NO-2)	ABRUPTIO PLACENTAE (YES-1,NO-2)	PULMONARY ODEMA (YES-1,NO-2)	HELLP (YES-1,NO-2)	ACUTE KIDNEY INJURY (YES-1,NO-2)	POST PARTUM HAEMORRHAGE(YES-1,NO-2)	PERIPARTUM CARDIOYOPATHY (YES-1,NO-2)	ICU admission (YES-1,NO-2)	Maternal Outcome	Neonatal Outcome	TERM/PRETERM	Birth weight (in kg)	NICU admission (YES-1,NO-2)
1	10058751	22	2	PRIMI	37+5	SEVERE PE	imminent signs	160/110	29.2	3+	1,36,000	14.4	901	492	0.88	1.09	431	N	N	0.68	7.2	N	2	LSCS	SEVERE PE + IMMINENT SIGNS	2	2	2	2	2	2	2	2	1	stable	LIVE	TERM	2.6KG	2
2	10059465	20	2	PRIMI	37+5	SEVERE PE	thrombocytopenia	148/110	28	1+	72000	8.3	952	549	0.91	0.9	347	N	N	0.66	5.6	N	2	LSCS	SEVERE PE +ATYPICAL HELLP	1	2	2	2	2	1	2	2	2	1	stable	LIVE	TERM	3 KG	2
3	10054412	25	2	PRIMI	37+2	SEVERE PE	imminent signs	162/98	26	3+	147000	6.9	1146	335	0.87	0.93	414	N	ABN	1.13	8.8	ABN DOPPLER	1	LSCS	SEVERE PE	2	2	2	2	1	2	2	2	2	2	stable	LIVE	TERM	2.5KG	2
4	10055206	20	2	PRIMI	33	SEVERE PE	thrombocytopenia with fgr	170/100	24	2+	125000	12.2	5053	271	0.95	0.92	629	ABN	N	0.6	6.8	FGR	2	LSCS	HELLP	2	2	2	2	2	1	2	2	2	1	stable	LIVE	PRETERM	1.28KG	1
5	10055552	26	2	G3P2L2	34+2	SEVERE PE	imminent signs	170/130	25.9	2+	300000	10.2	825	424	0.87	0.88	360	N	N	0.53	5.5	N	2	LSCS	UNCONTROLLED HTN	2	2	2	2	2	2	2	2	2	1	stable	LIVE	PRETERM	1.68KG	1
6	10056130	28	2	PRIMI	31+2	SEVERE PE	FGR	148/110	30.2	2+	170000	10.1	393	478	0.92	0.81	590	N	N	0.94	8.2	FGR WITH AEDF	2	LSCS	SEVERE PE WITH AEDF	2	2	2	2	2	2	2	2	2	2	stable	LIVE	PRETERM	1.15KG	1
7	10055444	21	2	PRIMI	37+2	eclampsia	seizure	160/88	24	2+	2,50,000	12.1	1473	418	0.91	0.78	310	N	N	0.72	12.9	N	2	LSCS	ANTEPARTUM ECLAMPSIA	2	1	2	2	2	2	2	2	2	1	stable	LIVE	TERM	2.3kg	2
8	10059528	24	2	PRIMI	36+5	SEVERE PE	imminent signs	160/100	27.5	1+	200000	12.3	200	492	0.9	0.91	220	N	N	0.57	5.2	N	1	LSCS	FAILED INDUCTION	2	2	2	2	2	2	2	2	2	1	stable	LIVE	PRETERM	2.2KG	1
9	10056023	25	1	PRIMI	37+3	SEVERE PE	imminent signs	160/100	29.4	2+	105000	11.3	412	478	0.97	1.09	346	N	N	0.58	4	N	2	LSCS	SEVERE PE	2	2	2	2	2	2	2	2	2	2	stable	LIVE	TERM	3.1KG	2
10	10056441	29	2	PRIMI	36+3	SEVERE PE	imminent signs	160/92	29.5	Negative	181000	12.4	437	393	0.86	0.93	259	N	N	0.59	5.1	N	2	LSCS	SEVERE PE	2	2	2	2	1	2	2	2	1	stable	LIVE	PRETERM	2.5KG	2
11	10058697	45	2	PRIMI	32+2	SEVERE PE	AEDF	156/110	27.3	TRACE	172000	12		581	1.1	0.83	281	N	N	0.55	4.4	AEDF	2	LSCS	TRANSVERSE LIE	2	2	2	2	2	2	2	2	2	2	stable	FSB	PRE TERM	900GM	
12	10059465	35	2	G2P1L1	36+4	SEVERE PE	fgr	160/100	24	1+	73000	11.8	952	549	0.91	0.9	347	N	N	0.66	5.6	FGR + ABN DOPPLER	2	LSCS	SEVERE PE	2	2	2	2	2	2	2	2	2	2	stable	LIVE	PRETERM	1.8KG	2
13	10061938	29	2	G4P2L1A1	31+5	SEVERE PE	imminent signs	180/98	27.3	1+	228000	9.6	1245	417	0.92	0.76	335	N	N	0.49	335	N	1	LSCS	FETAL DISTRESS	2	2	2	1	2	2	2	2	2	1	stable	LIVE	PRETERM	1.36KG	1
14	10063822	39	2	PRIMI	34+1	SEVERE PE	thrombocytopenia	180/100	29.5	2+	90000	11.9	468	380	0.96	0.86	464	N	N	0.59	6.3	N	2	LSCS	UNCONTROLLED HTN	2	2	2	2	2	2	2	2	2	1	stable	LIVE	PRETERM	1.85KG	1
15	10065593	30	1	PRIMI	39+5	SEVERE PE	imminent signs	158/112	29.3	1+	137000	13	668	370	0.92	1.13	307	N	N	0.65	5.2	N	1	LSCS	SEVERE OLIGOHYDRAMNIOS	2	2	2	2	2	2	2	2	2	2	stable	LIVE	TERM	2.7KG	2
16	10077266	38	2	PRIMI	28+1	SEVERE PE	FGR+AEDF	158/110	27.7	3+	240000	12	1169	408	0.88	1.03	405	ABN	N	0.58	5.8	FGR+AEDF	1	LSCS	SEVERE PE + UNCONTROLLED HTN	2	2	2	2	2	2	2	2	2	2	stable	LIVE	PRETERM	622GM	1
17	10066733	27	2	G3P2L2	34+2	SEVERE PE	imminent signs	170/130	25.9	2+	194000	11.5	677	537	0.88	0.77	285	N	N	0.46	3.7	N	2	LSCS	UNCONTROLLED HTN	2	2	2	2	2	2	2	2	2	1	stable	LIVE	PRETERM	1.68KG	1
18	10066553	31	2	G2P1L1	32	SEVERE PE	imminent signs	160/100	26	2+	208000	10.1	800	441	0.93	0.88	329	N	N	0.8	7.8	N	2	LSCS	SEVERE PE +IMMINENT SIGNS	2	2	2	2	2	2	2	2	2	1	stable	LIVE	PRETERM	1.5KG	1
19	10066938	38	2	G4P2L1A1D1	35+1	SEVERE PE	UNCONTOLLED HTN	170/100	30.9	3+	200000	13.7	1129	690	1	1.22	561	ABN	N	0.82	5.7	N	2	LSCS	PREVIOUS LSCS	1	2	2	2	2	1	2	2	2	1	stable	LIVE	PRETERM	1.8	1
20	10070944	25	1	G2P1L1	32+2	SEVERE PE	AEFD	170/100	29.9	2+	324000	13.5	600	362	0.86	1.02	343	N	N	0.61	4.4	AEDF	2	LSCS	SEVERE PE WITH AEDF	1	2	2	2	2	2	2	1	2	1	stable	LIVE	PRETERM	1.6KG	1
21	10069958	25	2	PRIMI	38+1	SEVERE PE	imminent signs	172/100	23.6	Trace	222000	13	646	572	0.87	0.81	368	N	N	0.65	4.2	N	2	NVD		2	2	2	2	2	2	2	2	2	2	stable	LIVE	TERM	3.1KG	2
22	10078955	30	2	PRIMI	30+2	ECLAMPSIA	AEDF	160/100	23.6	Trace	303000	10.7	243	497	1.08	0.8	265	N	N	0.53	4.5	FGR+AEDF	2	LSCS	ANTEPARTUM ECLAMPSIA	1	1	2	2	2	2	2	2	2	1	stable	LIVE	PRETERM	945GM	1
23	10078434	33	1	PRIMI	32+6	SEVERE PE	FGR	186/110	23.4	Negative	505000	9.2	327	554	1.06	0.84	346	N	N	0.56	4.4	FGR+INCRESED RESISTANCE	1	LSCS	PRROM WITH CHORIOAMNIONITIS	2	2	2	2	2	2	2	2	2	2	stable	LIVE	PRETERM	1.4KG	1
24	10078446	30	1	g2a1	34+6	SEVERE PE	FGR	160/112	24.5	2+	90000	11.7	821	537	0.91	0.92	363	N	N	0.59	5.1	FGR	2	LSCS	PREV LSCS	1	2	2	2	2	2	2	2	2	2	stable	live	PRETERM	2KG	2
25	10079019	23	1	PRIMI	34+3	ECLAMPSIA	imminent signs	150/110	31	2+	214000	12.1	1609	461	0.92	0.76	368	ABN	N	0.62	6.5	N	2	LSCS	ECLAMPSIA+HELLP	2	1	2	2	2	2	2	2	2	1	stable	LIVE	PRETERM	1.8KG	1
26	10081484	24	1	g2p1l1	35+1	SEVERE PE	imminent signs	160/84	29	1	72000	12	697	497	0.96	0.89	252	N	N	0.56	5.3	n	2	LSCS	SEVERE PE	2	2	2	2	2	2	2	2	2	2	stable	LIVE	PRETERM	2.7KG	2
27	10086177	24	2	PRIMI	36+2	SEVERE PE	imminent signs	142/92	26.3	Trace	82000	14.3	7979	<50	10	180	954	ABN	ABN	1.3	9.6	N	2	LSCS	BREECH WITH FETAL DISTRESS	1	2	1	2	2	1	1	1	2	1	DEATH	LIVE	PRETERM	2.9KG	1
28	10086952	32	2	G3A2	33+4	SEVERE PE	imminent signs	162/100	24.6	2+	327000	9.2	1161	461	1.06	0.9	297	N	N	0.41	4.7	fgr	2	LSCS	ANAMNIOS	2	2	2	2	2	2	2	2	2	2	stable	LIVE	PRETERM	2.3KG	1
29	10085179	38	2	G7P4L1A2D3	30+1	SEVERE PE	imminent signs	164/110	26.4	1+	214000	11.1	378	475	1.06	0.8	253	N	N	0.5	4.1	FGR+INCRESED RESISTANCE	2	LSCS	BREECH IN LABOUR	2	2	2	2	2	2	2	2	2	2	stable	LIVE	PRETERM	1.2KG	1
30	10092085	20	1	PRIMI	39+2	SEVERE PE	imminent signs	160/110	24.4	1+	243000	10.9	787	200	0.95	1.2	277	N	N	0.76	6	N	1	VENTOUSE		2	2	2	2	2	2	2	2	2	stable	LIVE	TERM	3KG	2
31	10091957	28	2	G3P1L1	35+3	SEVERE PE	imminenet signs	156/110	24.6	2+	442000	9.5	559	675	0.7	0.8	233	N	N	0.42	3.8	MACROSOMIA	2	LSCS	PREVIOUS LSCS	2	2	2	2	2	2	2	2	2	2	stable	LIVE	PRETERM	3.4KG	2
32	10093265	24	1	G3P2L2	30+3	SEVERE PE	imminent signs	156/110	23	1+	243000	9.2	100	480	0.97	0.83	300	N	N	0.5	3.4	FGR	2	LSCS	PREVIOUS LSCS	2	2	2	2	2	2	2	2	2	2	stable	LIVE	PRETERM	1.2KG	1
33	10097803	26	2	G3P2L2	34+2	SEVERE PE	imminent signs	170/130	25.9	2+	74000	12.4	1459	205	1.25	1	1231	ABN	ABN	3.09	9.4	N	2	LSCS	UNCONTROLLED HTN	2	2	2	2	2	2	2	2	2	1	stable	LIVE	PRETERM	1.68KG	1
34	10098880	30	1	G3P2L2	36+5	SEVERE PE	imminent signs	152/110	23	Trace	220000	11.3	555	367	0.89	0.86	290	N	N	0.65	8.6	FGR+AEDF	1	LSCS	FETAL DISTRESS	2	2	2	2	2	2	2	2	2	2	stable	LIVE	PRETERM	1.7KG	1
35	10099436	34	1	G3P1L1A1	40+2	SEVERE PE	imminent signs	146/110	28	1+	139000	9.4	1412	373	1.05	1.04	935	N	N	0.42	7.8	N	1	NVD		2	2	2	2	2	2	2	2	2	2	stable	LIVE	TERM	2.7KG	2
36	10101640	29	2	G2A1	30+1	SEVERE PE	imminent signs	170/100	24.8	1+	206000	10.1	824	314	0.83	0.82	677	N	N	0.98	8.9	AEDF	2	LSCS	SEVERE PE WITH AEDF	2	2	2	2	1	2	2	2	2	1	stable	LIVE	PRETERM	1.06KG	1
37	10101510	36	1	G4A3	29+1	SEVERE PE	FGR	164/96	24.5	1+	223000	12.5	825	353	0.92	0.96	229	N	N	0.58	5.5	REDF	2	LSCS	REDF	2	2	2	2	2	2	2	2	2	2	stable	LIVE	PRETERM	750GM	1
38	10103388	40	2	PRIMI	30+5	SEVERE PE	imminent signs	150/110	23.6	1+	328000	12	1518	497	0.94	1.02	328	N	N	0.86	7.2	N	2	LSCS	SEVERE PE	2	2	2	2	2	2	2	2	2	1	stable	LIVE	PRETERM	1.1KG	1
39	10104279	34	1	PRIMI	37+5	SEVERE PE	FGR	160/100	25	Negative	237000	11.1	700	475	0.96	0.97	235	N	N	0.46	3.1	fgr	2	LSCS	SEVERE PE	2	2	2	2	2	2	2	2	2	2	stable	LIVE	TERM	2.3KG	1
40	10107705	22	1	G2P1L1	36	SEVERE PE	imminent signs	162/102	26.8	1+	192000	12.1	416	418	0.93	0.94	303	N	N	0.49	5.1	FGR	2	LSCS	SEVERE PE	2	2	2	2	2	2	2	2	2	2	stable	LIVE	PRETERM	2.6KG	2
41	10110581	36	2	PRIMI	38	SEVERE PE	imminent signs	166/110	23	1+	155000	11.9	269	410	1.05	0.92	269	N	N	0.51	5.2	FGR	2	LSCS	FETAL DISTRESS	2	2	2	2	2	2	2	2	2	2	stable	LIVE	TERM	2.9KG	2
42	10111389	46	2	PRIMI	31+6	SEVERE PE	imminent signs	180/100	24.8	1+	375000	10.6	1126	451	0.83	0.98	342	ABN	ABN	1.07	7.9	N	2	LSCS	SEVERE PE	2	2	2	2	2	2	2	2	2	1	stable	LIVE	PRETERM	1.7KG	1
43	10110499	27	2	PRIMI	26+1	SEVERE PE	imminent signs	160/108	24.9	3+	95000	6.7	448	299	1.05	1.06	405	N	N	0.77	7.6	N	1	LSCS	SEVERE PE+ FAILED INDUCTION	2	2	2	2	2	2	2	2	2	1	stable	FSB	PRETERM	698G	
44	10115157	30	1	PRIMI	28+6	SEVERE PE	imminent signs+FGR	162/110	22	2+	89000	11.1	1043	355	0.78	1.09	326	N	N	0.63	4.5	FGR	2	LSCS	SEVERE PE	2	2	2	2	2	1	2	2	2	1	stable	FSB	PRETERM	875G	
45	10118198	24	1	G3P2L2	36+6	SEVERE PE	imminent signs	166/100	26	Trace	64000	8.1	989	464	1.14	1.25	1437	N	N	0.64	7.6	FGR	2	LSCS	SEVERE PE +HELLP	1	2	2	2	2	1	2	2	2		stable	LIVE	PRETERM	2.2KG	Y
46	10102801	23	1	G4P1L1A3	27+2	ECLAMPSIA	imminent signs	154/112	28	1+	233000	9.6	344	344	1.1	0.83	682	N	ABN	1.53	6.6	FGR	2	LSCS	ECLAMPSIA+HELLP	1	1	2	2	2	1	2	2	2	1	stable	FSB	PRETERM	900G	
47	10109006	28	2	G2A1	26+3	ECLAMPSIA	imminent signs	160/110	25	3+	178000	11.8		451	0.9	0.99	460	N	N	0.53	3.1	FGR	1	NVD		2	1	2	2	2	2	2	2	2	1	stable	FSB	PRETERM	434G	

48	10118910	20	2	PRIMI	32	SEVERE PE	imminent signs	144/112	29	2+	77000	14.8	1314	284	0.96	0.86	593	ABN	N	0.57	4.8	FGR	2	LSCS	SEVERE PE +HELLP	2	1	2	2	2	1	2	2	2	1	stable	LIVE	PRETERM	1.13KG	Y
49	10112842	34	1	PRIMI	28+4	SEVERE PE	imminent signs	168/112	23	Trace	333000	13.4	196	464	0.93	0.8	128	N	N	0.56	5.1	AEDF	1	LSCS	SEVERE PE+HELLP	2	2	2	2	2	1	2	2	2	1	stable	LIVE	PRETERM	828G	Y
50	10112925	23	2	G2P1L1	24+2	SEVERE PE	imminent signs	156/114	22	1+	273000	11.3	203	124	0.98	1	252	N	N	0.92	6.8	FGR	1	LSCS	PREV	2	2	2	2	2	2	2	2	2	2	stable	LIVE	PRETERM	2KG	N
51	10121661	19	1	PRIMI	30+1	SEVERE PE	imminent signs	142/110	28	1+	300000	10.9	773	353	0.9	1	315	N	N	0.47	4.2	FGR	1	NVD		1	2	2	1	2	2	2	2	2	1	stable	FSB	PRETERM	730GM	
52	10121953	20	1	PRIMI	38+4	SEVERE PE	imminent signs	164/110	29	2+	259000	8.7	500	589	0.84	0.86	527	ABN	N	0.59	6.3	N	2	VENTOUSE		1	2	2	2	2	1	2	2	2	1	stable	LIVE	TERM	3KG	N
53	10118125	28	2	G2P1L1	30+2	SEVERE PE	imminent signs	146/112	25	Trace	160000	11.3	408	372	0.92	0.88	219	N	N	0.68	7.1	FGR	2	LSCS	SEVERE PE	2	2	2	2	2	2	2	2	2	2	stable	LIVE	PRETERM	2KG	Y
54	10122743	22	2	G1L1P1	34+4	SEVERE PE	imminent signs	140/110	26	1+	66000	6.3	938	118	2.75	1.89	1383	ABN	ABN	2.13	9	FGR	2	LSCS	SEVERE PE +HELLP	2	2	2	2	2	1	2	2	2	1	stable	LIVE	PRETERM	1.8KG	N
55	10122908	22	2	PRIMI	38	SEVERE PE	imminent signs	150/110	28	2+	186000	7.5	700	355	0.93	0.88	1796	ABN	N	0.7	9.6	FGR	1	LSCS	MSL	2	2	2	2	2	1	2	2	2	2	stable	LIVE	TERM	2.6KG	N
56	10106405	29	2	G3P2L2	37+2	severe pe	FGR	154/114	23	1+	261000	12.4		332	0.93	0.83	288	N	N	0.57	5	FGR	2	NVD		2	2	2	2	2	2	2	2	2	2	stable	LIVE	TERM	2.6KG	N
57	10108924	26	2	PRIMI	28+1	SEVERE PE	imminent signs	150/110	25	3+	231000	10.2	700	300	0.9	0.91	312	N	N	0.59	5.3	FGR + ABN DOPPLER	2	LSCS	REDF	2	2	2	2	2	2	2	2	2	2	stable	LIVE	PRETERM	680G	Y
58	10110917	28	2	PRIMI	37	SEVERE PE	FGR	160/112	27	1+	269000	10.2	500	496	0.84	0.75	1208	N	N	0.58	4.5	FGR	1	LSCS	CPD	2	2	2	2	2	2	2	2	2	2	stable	LIVE	TERM	2.4KG	N
59	10114071	24	2	G2P1L1	36+1	SEVERE PE	FGR	164/110	26	Trace	145000	13.4	400	240	0.83	0.72	606	N	N	0.4	5.5	FGR	2	LSCS	MSL	2	2	2	2	2	2	2	2	2	2	stable	LIVE	PRETRM	2.2KG	Y
60	10118018	34	2	G3P1L1	35+3	SEVERE PE	FGR	152/110	29	2+	372000	12.9	897	123	0.78	0.85	428	N	N	0.5	3.8	FGR+AEDF	2	LSCS	FETAL DISTRESS	2	2	2	2	2	2	2	2	2	2	stable	LIVE	PRETERM	2KG	Y
61	10102268	29	1	G2A1	35	SEVERE PE	imminent signs	180/110	24.9	2+	188000	10.3		475	0.83	0.84	333	N	ABN	1.37	7.8	FGR	2	LSCS	SEVERE PE	2	2	2	2	2	2	2	2	2	2	stable	LIVE	PRETERM	2.2KG	Y
62	10093265	24	1	G3P2L2	30+3	SEVERE PE	imminent signs	154/110	22.8	Trace	243000	10.1	100	474	0.83	0.97	221	N	N	0.52	3.4	FGR WITH ABN DOPPLER	2	LSCS	SEVERE PE	2	2	2	2	2	2	2	2	2	1	stable	LIVE	PRETERM	1.2KG	Y
63	10095737	25	2	G4P1L1A1	35+4	SEVERE PE	NONE	154/110	22	1+	135000	11.8	400	489	0.88	0.92	324	N	N	0.49	4.4	FGR	2	LSCS	PREVIOUS LSCS+PROM	2	2	2	2	2	2	2	2	2	2	stable	LIVE	PRETRM	2.4KG	N
64	10096304	31	2	G4P2L2	37+6	SEVERE PE	NONE	148/110	25.9	1+	252000	8.6	415	402	0.96	0.85	243	N	N	0.56	4.7	FGR	2	LSCS	PREVIOUS LSCS	2	2	2	2	2	2	2	2	2	2	stable	LIVE	TERM	2.9KG	N
65	10096718	23	1	PRIMI	37	SEVERE PE	FGR	150/110	24.9	1+	160000	10.9	407	371	0.92	1	284	N	N	0.48	2.7	FGR	2	NVD		2	2	2	2	2	2	2	2	2	2	stable	LIVE	TERM	2.5KG	N
66	10091705	29	2	G3P2L2	38+3	SEVERE PE	imminent signs	150/110	23	Trace	161000	11.4	422	371	0.93	1.02	484	N	N	0.48	5	ABN DOPPLER	2	LSCS	SEVERE PE	2	2	2	2	2	2	2	2	2	2	stable	LIVE	TERM	2.56KG	N
67	10099481	22	2	PRIMI	28+1	SEVERE PE	FGR	164/110	24	Trace	219000	11.5	347	454	0.91	0.57		N	N	0.4	5	FGR	1	LSCS	MSL	2	2	2	2	2	2	2	2	2	1	stable	LIVE	PRETERM	2.KG	Y
68	10100785	29	1	G3P1L1A1	30+2	SEVERE PE	imminent signs	144/114	24.9	1+	253000	9.9	884	576	1.12	0.95	200	N	N	0.44	4.1	FGR	1	LSCS	FETAL DISTRESS	2	2	2	2	2	2	2	2	2	1	stable	LIVE	PRETERM	1.5	Y
69	10101766	25	2	PRIMI	29+2	SEVERE PE	imminent signs	169/116	26.9	2+	268000	11.1	803	425	0.95	0.93	157	N	N	0.42	5.2	N	1	NVD		2	2	2	2	2	2	2	2	2	2	stable	LIVE	PRETERM	1.9KG	Y
70	10102307	26	2	G4P1L1A2	31	SEVERE PE	imminent signs	180/116	27.9	1+	242000	7.9	389	577	0.99	0.85	557	N	N	0.62	6.3	N	2	LSCS	PREVIOUS LSCS	2	2	2	2	2	2	2	2	2	2	stable	LIVE	PRETERM	1.87KG	Y
71	10126306	31	1	G3A2	32+4	SEVERE PE	FGR	160/94	24.6	Trace	306000	12.9	95	245	1	0.94	200	N	N	0.59	7.2	ABN DOPPLER	1	LSCS	SEVERE PE	2	2	2	2	2	2	2	2	2	1	stable	LIVE	PRETERM	2KG	Y
72	10103654	27	2	PRIMI	32	SEVERE PE	imminent signs	148/112	24.4	1+	229000	11.6	200	200	1	0.92	235	N	N	0.46	8.4	N	2	LSCS	FETAL DISTRESS	2	2	2	2	2	2	2	2	2	2	stable	LIVE	PRETERM	2.1KG	Y
73	10103368	29	2	PRIMI	31+4	SEVERE PE	imminent signs	142/110	26.9	Trace	232000	11.9	119	513	1.01	0.8	212	N	N	0.54	6.7	N	1	NVD		2	2	2	2	2	2	2	2	2	1	stable	LIVE	PRETERM	2KG	A
74	10103281	21	2	G3A2	34	SEVERE PE	imminent signs	160/110	26.7	1+	157000	9.9	1219	325	0.96	0.79	245	N	N	0.55	5.9	ABN DOPPLER	2	LSCS	PREV LSCS	2	2	2	2	2	2	2	2	2	2	stable	LIVE	PRETERM	2.1KG	Y