"PLATELET COUNT AND PLATELET INDICES IN PREGNANCY WITH PREECLAMPSIA AND ECLAMPSIA – A PROSPECTIVE OBSERVATIONAL STUDY"

By

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

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LIST OF ABBREVIATIONS

PE	:	Preeclampsia
MPV	:	Mean platelet volume
PDW	:	Platelet distribution width
PC	:	Platelet count
HELLP	:	Haemolysis – elevated liver enzyme- Low platelet
DIC	:	Disseminated Coagulopathy
PRES	:	Posterior reversible encephalopathy syndrome
PAPP-A	:	Pregnancy–associated plasma protein A
PlGF	:	Placental growth factor
sFlT-1	:	Soluble FMS like tyrosinase kinase 1
LDH	:	Lactate Dehydrogenase
VEGF	:	Vascular endothelial growth factor
sENG	:	Soluble endoglin
HIF	:	Hypoxia-inducible transcription factor
ROS	:	Reactive oxygen species
AKI	:	Acute kidney injury
CKD	:	Chronic kidney disease

ABSTRACT

Introduction - Detecting pregnant women at higher risk of preeclampsia is a crucial objective in modern obstetrics. Developing a test that is sensitive, specific, cost-effective, and easy to administer would not only enable the identification of women prone to preeclampsia but also facilitate close monitoring, accurate diagnosis, and timely intervention. Although the exact pathophysiology of preeclampsia is not completely understood, the utility of different platelet indices can be utilized to predict preeclampsia.

Aims and objective – To study the association of platelet count and platelet indices in pregnancy with preeclampsia and Eclampsia.

Material and method – A prospective observational study was conducted in the Department of Obstetrics and Gynaecology at KAHER's Dr Prabhakar Kore Hospital and Medical Research Centre, Belagavi for a total duration of 1 year. A total of 174 women were included in the study .

Results -174 participants were analysed in the study. The mean platelet count of the study participant is 2.32± 0.73. In our study , we found that mean platelet count and plateletcrit showed a decreasing trend while MPV and PDW showed an increasing trend with increasing severity of the disease, although the results were statistically insignificant. Also we observed 85.45% of preeclamptic women without severe features and 79.66% of preeclamptic women with severe features and eclampsia were nonthrombocytopenic. PC/MPV ratio shows a decreasing trend as severity of the disease increases which was statistically significant with p -value of (<0.001) .

Conclusion – Platelet Indices such as MPV, PDW, Plateletcrit, PC/MPV, showed a significant variation along with the severity of the disease, Platelet indices, especially PC/MPV can be used along with platelet count to evaluate the severity of preeclampsia instead of relying on platelet count alone.

Keywords – Platelet indices , Plateletcrit ,MPV, PDW , PC/MPV , Preeclampsia , Eclampsia.

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1. INTRODUCTION

One of the grave complications of pregnancy is a hypertensive disorder which affects about 5-7% of the pregnancies and is the preeminent cause of maternal and foetal morbidity and mortality [1].

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 [2]. One of the prime causes of preterm birth is preeclampsia, In the newborn it is an early marker for future cardiovascular and metabolic disease in the newborns.[2,3,4]

Annually preeclampsia is responsible for 70,000 maternal deaths worldwide along with 500,000 foetal deaths. And is solely responsible for 14% of the deaths associated with pregnancy, rendering it the 2nd most common cause of pregnancy-related deaths [Centers for Disease Control and Prevention].

Pre-eclampsia is characterized by an acute increase in systolic blood pressure of $\geq$ 140 mmHg and/or diastolic blood pressure of $\geq$ 90 mmHg, along with the presence of proteinuria, diagnosed by the presence of 300 mg protein in 24 h urine sample or the presence of protein/creatinine ratio $\geq$ 0.3. In case of the absence of proteinuria, it is characterized by sudden onset of hypertension along with the emergence of any of the following functional disorders in the mother like thrombocytopenia with a platelet count below 100,000/µL, deranged liver function test confirmed by an increase of two-fold in the concentration of liver transaminases,

renal insufficiency diagnosed by the presence of a two-fold increase in the concentration of serum creatinine and pulmonary edema [5].

Eclampsia is characterized as preeclampsia with the onset of seizure or coma antenatally or postpartum, for which another neurological disorder is not the cause[5].Eclampsia has a recurrence rate of around 10% if not treated[6]

When interactions between trophoblasts and the decidua fail during the first trimester, it triggers a stress response in the placenta. This impacts the growth and development of the villous tree, which in turn affects the transfer of oxygen and nutrients to the foetus. The stress on the syncytiotrophoblast results in the release of various factors into the bloodstream. These factors trigger a systemic inflammatory response due to disturbances in the maternal endothelium's homeostatic functions, affecting clotting regulation, fluid transfer, and blood pressure [7].

The damaged vascular endothelium causes platelets to adhere more at the site of injury, resulting in increased consumption and subsequent platelet destruction, leading to thrombocytopenia.

Platelet activation is widespread, accompanied by heightened degranulation, resulting in a reduction in platelet lifespan and a rise in immature platelet count observed in peripheral blood smears [1,4]. Platelet count varies and could correlate with alterations in platelet indices such as Platelet Distribution Width (PDW), Mean Platelet Volume (MPV), Plateletcrit, and the Platelet Count Ratio Mean Platelet Volume (PC/MPV).

The MPV reflects the average platelet size, while PDW indicates the variation in platelet size. When peripheral platelet destruction results in reduced platelet count, the body stimulates platelet production, releasing larger platelets into circulation, thereby increasing the mean platelet volume[8].

Platelet distribution width reflects variations in platelet morphology caused by the presence of both large and normal-sized platelets. This parameter can be clinically linked to platelet activation. Large platelets, characterized by an increase in both number and size of pseudopodia, exhibit greater activity compared to smaller platelets, resulting in an elevated PDW[9].

Plateletcrit indicates the overall mass of platelets, akin to hematocrit for red blood cells. The hemostatic efficacy of platelets relies not only on their quantity but also on their size, as larger platelets tend to be more functionally active than smaller ones. Plateletcrit reflects alterations in both platelet size and count, as it is derived from both MPV and platelet count[10]

Plateletcrit, which signifies the total platelet mass, provides a more comprehensive indication of platelet hemostatic capacity compared to platelet count alone. It's the circulating platelet mass, not just the count, that the body regulates, so a low Plateletcrit indicates diminished platelet activity. A platelet count below 100,000/L suggests a severe disease condition [7-9].

A reduced platelet count is commonly considered a feature of deteriorating preeclampsia and eclampsia. However, various studies have shown that uncontrolled platelet activation and aggregation also occur in nonthrombocytopenic cases of preeclampsia and eclampsia[2,10].

The platelet-related parameters, known as platelet indices, have not been thoroughly investigated in cases of preeclampsia and eclampsia where the platelet count is normal. Therefore, there is a necessity to assess markers of platelet activation in nonthrombocytopenic instances of preeclampsia and eclampsia.

Detecting pregnant women at higher risk of preeclampsia is a crucial objective in modern obstetrics. Developing a test that is sensitive, specific, cost-effective, and easy to administer would not only enable the identification of women prone to preeclampsia but also facilitate close monitoring, accurate diagnosis, and timely intervention.

Due to the rapid advancement of the illness, it necessitates immediate intervention and the termination of pregnancy, either through labor induction or cesarean section, as it stands as the only cure for the condition[12,13,14].

As it affects multiple organs, no single, specific and cost-effective marker to predict PE has yet been proposed [9]. However, several models have been suggested but are found to be not clinically relevant[10-12].

Platelet indices, encompassing platelet count (PC), platelet distribution width (PDW), mean platelet volume (MPV), plateletcrit (PCT), and PC/MPV, are among the parameters captured in a complete blood count (CBC) test. Although there has been research into their relevance for understanding vascular diseases, including preeclampsia (PE), their precise value remains to be fully validated[15,16]

During the progression of preeclampsia (PE), a decline in platelet count (PC) is noted, indicating a potential indicator of deteriorating PE [9].

The decrease in platelet count during pregnancy typically returns rapidly to its baseline range after delivery. Additionally, there is an observed elevation in mean platelet volume (MPV) throughout pregnancy, particularly in women with preeclampsia (PE). This increase in MPV tends to precede the onset of PE symptoms, suggesting its potential as a valuable marker for the development of PE [13-15].Additionally, it has been suggested that PDW could serve as a useful tool for assessing the activation of coagulation or diseases associated with thrombocytosis . There are several studies conducted which shows conflicting results .

Thus , the purpose of this study was to find the association between platelet count and indices in pregnancy with preeclampsia and eclampsia and to look for new markers of platelet activation.

2 OBJECTIVES

Researchers have found variation in platelet indices in the early stage of preeclampsia, measurement of platelet indices can act as a predictor of maternal complication .Currently there are no specific and cost effective test that is done for prediction of preeclampsia and eclampsia progression .Various studies had been done previously for different platelet indices as predictor of preeclampsia , however report in this regard are controversial **.**

- **PRIMARY OBJECTIVE**

- To study the association of platelet count and platelet indices in pregnancy with preeclampsia and eclampsia

- **SECONDARY OBJECTIVE**

To assess maternal and foetal outcomes in patients with preeclampsia and eclampsia.

3 REVIEW OF LITERATURE

Background

Hypertensive disorders are the predominant medical complications during pregnancy, impacting approximately 7-15% of expectant mothers. Based on a systemic review conducted by the World Health Organization (WHO) on global maternal mortality, hypertensive disease continues to stand as a primary cause of direct maternal mortality. Together with hemorrhage and infection, hypertension comprises a lethal trio contributing to maternal morbidity and mortality during pregnancy and childbirth[4,18].

While maternal mortality rates are notably lower in developed nations compared to developing ones, the occurrence of preeclampsia is on the rise in developed countries[19].This trend could be linked to the growing prevalence of predisposing conditions like chronic hypertension, diabetes, and obesity[20].

Hypertension during pregnancy has lasting effects such as chronic hypertension and an elevated lifetime cardiovascular risk. Additionally, hypertensive disorders pose risks for the baby as well. Preeclampsia is significantly linked with fetal growth restriction, low birth weight, premature delivery either spontaneously or through medical intervention, respiratory distress syndrome, and admission to the neonatal intensive care unit[21].

Preeclampsia is a significant contributor to preterm birth rates, including both spontaneous and medically induced cases.

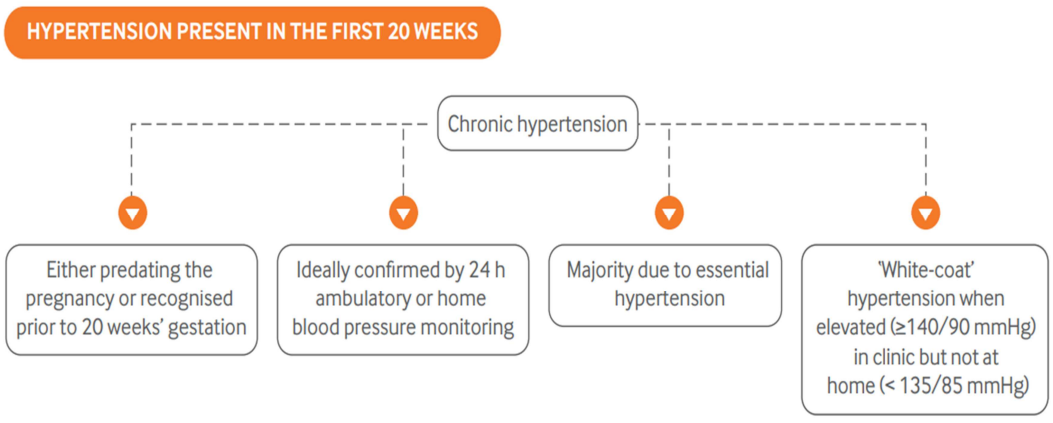
It's common for growth restriction to occur due to placental disorders, affecting approximately 20-25% of preterm births and 14-19% of term births in women with preeclampsia, resulting in birth weights falling below the 10th percentile for the gestational age. Hypertensive disorders increase the susceptibility of women and foetuses to a heightened risk of subsequent complications and long-term consequences[4,22,23]

Hypertension manifesting before 20 weeks comprises of

1) Essential chronic or secondary hypertension
2) White coat hypertension
3) Masked Hypertension

Hypertension manifesting beyond 20 weeks comprises of

1) Transient gestational hypertension
2) Gestational hypertension
3) Preeclampsia



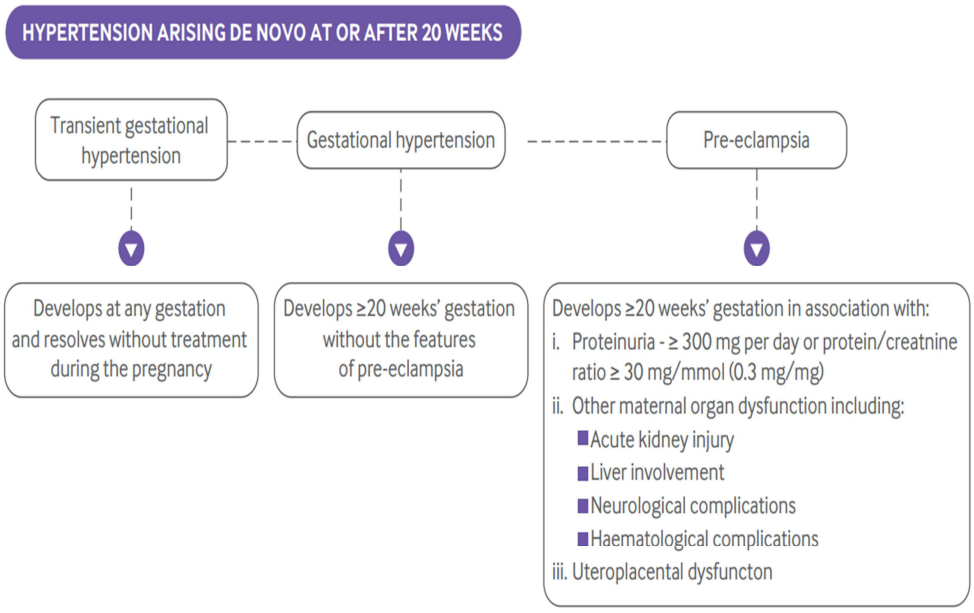


Figure 3-1 ***Category of hypertension in pregnancy acknowledged by the ISSHP***

Chronic hypertension refers to elevated blood pressure that is either detected before the 20th week of pregnancy, diagnosed before conception, or persists up to 12 weeks after childbirth. chronic hypertension results in complications in around 5 % of all pregnancies. Essential hypertension is diagnosed when there is no apparent underlying cause for chronic hypertension. secondary hypertension may be caused by renal parenchymal disease or scarring, renovascular disease, an endocrine disorder, or coarctation of aorta.

White coat hypertension is diagnosed when there is elevated office or clinic BP >= 140/90 mmHg, but normal BP at home or work (<135/85 mmHg) , it is not an entirely benign condition but conveys an increased risk for preeclampsia.

Masked hypertension is another form of hypertension more difficult to diagnose, characterized by BP that is normal at clinic or office visits but elevated at other time(4).

Gestational hypertension is defined as new onset hypertension developing after 20 weeks of gestation, during labour without proteinuria or any other systemic feature of preeclampsia in a previously normotensive, nonproteinuric woman and the blood pressure resolves within 3 months postpartum. It usually has a favourable outcome with a quarter progressing to preeclampsia. (4)

3.1 Definition of preeclampsia and 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 Gynecologists), 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 preeclampsia indicators may manifest in some women without proteinuria. Both organizations advise against using terms like "severe" and "mild" preeclampsia, as all cases pose potential clinical threats. Instead, they recommend categorizing cases as preeclampsia without severe features or with severe features[4,23].

Diagnostic criteria for preeclampsia without severe feature

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 preeclampsia 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 (4)

Eclampsia is characterized by the occurrence of seizures during the antepartum, intrapartum, or postpartum periods, in conjunction with the signs and symptoms of preeclampsia.

It is an obstetric emergency. In underdeveloped countries, a prevalence of 2.3 – 6 per 10,000 births has been reported. (24) Seizures before 20 weeks of gestation have been reported in case of prenatal trophoblastic disease.(25)

Eclampsia often presents with neurological symptoms like headache (80%) and visual disturbances (45%). Approximately 15% of women with eclampsia exhibit a diastolic blood pressure below 90 mmHg.Eclamptic seizures last for 60-90 seconds followed by postictal confusion, and agitation. They may also result in hypoxia-related bradycardia in the fetus.(26)

Preeclampsia contributes to 12-25% of cases of fetal growth restriction and is responsible for 15-20% of all preterm births. This, in turn, increases the risk of neonatal mortality and long-term health issues in adulthood, including stroke, coronary heart disease, and metabolic syndrome.

Preeclampsia and eclampsia are also linked to one-quarter of stillbirth and neonatal mortality in underdeveloped nation.(25,27)

3.2 Incidence of preeclampsia

Preeclampsia and eclampsia contribute to more than 50,000 maternal deaths globally each year. The incidence of preeclampsia ranges from 2 to 10% of pregnancies worldwide. According to the World Health Organization (WHO), preeclampsia is seven times more prevalent in developing countries (2.8% of live births) compared to developed countries (0.4% of live births)[28].

According to India's third National Family Health Survey (NFHS-3, 2005-06), the occurrence of preeclampsia and eclampsia in India (28% and 7.4-11.3%, respectively) surpasses the global average. India also reports the highest number of

preterm births globally, with preeclampsia (36%), chronic hypertension (5%), eclampsia (4.8%), and gestational hypertension (4.8%) being the most common risk factors. Consequently, India is currently prioritizing the provision of quality antenatal care to pregnant women, with a particular focus on addressing preeclampsia[29].

3.3 Risk factors for preeclampsia

Numerous factors contribute to the onset of preeclampsia during pregnancy, including but not limited to:

1. History of preeclampsia: A woman with a prior history of preeclampsia in a previous pregnancy faces an eightfold higher risk of developing the condition again. The severity of preeclampsia in the prior pregnancy significantly amplifies this risk. Preeclampsia occurring in the second trimester is linked to a higher recurrence rate, ranging from 25% to 65%. In pregnancies where there is no history of preeclampsia, the incidence is typically 5% to 7%.
2. Pre-existing medical conditions: These include pre-gestational diabetes, and chronic hypertension (which accounts for 5 to 10 percent of preeclampsia cases).
3. Autoimmune disorders: Some autoimmune diseases like thrombophilia systemic lupus erythematosus and antiphospholipid syndrome elevate the risk for preeclampsia.
4. High body mass index: For every rise in BMI of 5 to 7 kg/m2 before pregnancy, the likelihood of developing preeclampsia doubles. Globally, obesity and overweight contribute to more than 40% of preeclampsia cases, resulting in a two- to threefold increase in the risk of developing the condition.

5. Chronic kidney disease: The risk differs based on the degree of decline of glomerular filtration rate and whether or not hypertension is present. Chronic hypertension with superimposed Preeclampsia was found in the latter part of pregnancy in as high as 40 - 60 percent of women with advanced chronic renal disease.
6. Multifetal pregnancy: An incidence of 20 percent is reported in multifetal gestation.
7. Nulliparity: It heightens the likelihood of developing preeclampsia.
8. A family history of hypertension among first-degree relatives raises the likelihood of preeclampsia.
9. Prior pregnancy complications especially abnormalities in placentation: Fetal growth restriction, abruption, and stillbirth are outputs of placental insufficiency and pose as risk factors for the occurrence of preeclampsia,
10. Higher maternal age: Due to the presence of obesity, diabetes mellitus, and chronic hypertension, along with advancing age, the likelihood of developing preeclampsia is influenced[30].
11. Factors related to the couple include primiparity, limited sperm exposure, and pregnancies resulting from donor insemination, donor egg, or donor embryo.
12. Couple-related risk factors – Prim paternity, limited sperm exposure, pregnancy after donor insemination, donor egg, donor embryo.
13. Fetal gender is increasingly acknowledged as a significant risk factor. Research into sex differences in placental gene expression reveals that nearly half of these differences are x-linked and stem from the escape of X chromosome inactivation.

14. Smoking
15. Genetic factors – The protein produced by the s-FLT1 (FMS-like tyrosine kinase 1) gene is a recognized contributor to preeclampsia and is linked to a higher occurrence in late-onset cases. Its impact is solely on the fetal genome, not the maternal one[31]. The heritability of preeclampsia is estimated to be approximately 55%, with maternal and fetal genetic factors contributing around 30-35% and 20%, respectively, to the overall risk[32]

The National Institute for Health and Clinical Excellence (NICE) has categorized risk factors for preeclampsia into moderate and high risk, aiming to facilitate identification and management. If two moderate risk factors or one high-risk factor are present, prophylactic measures should be administered to pregnant women.

"The factors termed as high risks include:

- History of any hypertensive disorder in previous pregnancies
- Chronic kidney disease
- Autoimmune diseases like systemic lupus erythematosus or antiphospholipid antibody syndrome;
- Diabetes type 1 or 2;
- Chronic arterial hypertension"

"Factors termed moderate risk include:

- Primiparity
- Age > 40 years
- Interpregnancy interval greater than 10 years;
- Body mass index (BMI) higher than 35 kg/m^2

- A family history of preeclampsia
- Multiple pregnancies”

The "American College of Obstetricians and Gynecologists" (ACOG) also suggests the same risk factors as outlined by NICE, with the only distinction being the BMI threshold of 30 kg/m2, above which the risk for preeclampsia escalates. Furthermore, ACOG categorizes all these factors as high risk[33].

This approach led to the detection of 37% and 28.9% of cases in early and late preeclampsia, respectively. The most reliable predictor was found to be a previous history of preeclampsia.

3.4 Pathogenesis of preeclampsia

The main factors in the development of preeclampsia involve irregular invasion of spiral arteries by trophoblasts, improper activation of endothelial cells, and an overly intense inflammatory reaction. The evidence, such as the regression of symptoms and abnormalities post-delivery and the heightened occurrence of preeclampsia in multiple pregnancies and cases of hydatiform mole, strongly supports the placental origin of preeclampsia.

The preeclampsia syndrome has been hypothesized as a two-stage disorder

Primary stage

It involves abnormal placentation. In normal pregnancy, the wall of spiral arteries is invaded by endovascular trophoblastic cells. This migration transforms the small, Musculo-elastic arteries into large, torturous channels that carry a large amount of blood into the intervillous and are resistant to the effect of vasomotor agents.

The invasion starts during the first trimester, it reaches completion in the second trimester following a secondary wave of trophoblastic invasion.

In patients with preeclampsia, there are incomplete physiological changes, where trophoblastic invasion only impacts certain spiral arteries and does not extend to the myometrial portion, resulting in incomplete invasion [34].

The outcome of this inadequacy is a reduction in uteroplacental blood flow. Moreover, due to the arteries retaining their muscular structure, they continue to respond to vasomotor stimuli.

The progression of preeclampsia to its second stage involves the shift from uteroplacental maladaptation to a systemic maternal syndrome. This stage is subject to alteration by preexisting maternal conditions such as cardiac or renal disease, diabetes, obesity, or genetic factors.

During this subsequent phase of maternal systemic disease, there is an intensified activation of endothelial cells and a widespread increase in inflammatory activity leading to a generalized hyperinflammatory state. Episodes of placental hypoxia or reperfusion lead to oxidative stress, followed by apoptosis and necrotic disruption of the syncytial structure.

The disturbance in both the anatomy and physiology of typical placental development is believed to trigger the release of placental debris from the intervillous space into the maternal bloodstream. This action then prompts a systemic inflammatory reaction by stimulating the production of inflammatory cytokines, substances that influence angiogenesis and abnormal lipid peroxidation. These

substances will impact the endothelial system, leading to the manifestation of signs and symptoms indicative of compromised function across multiple organs[35].

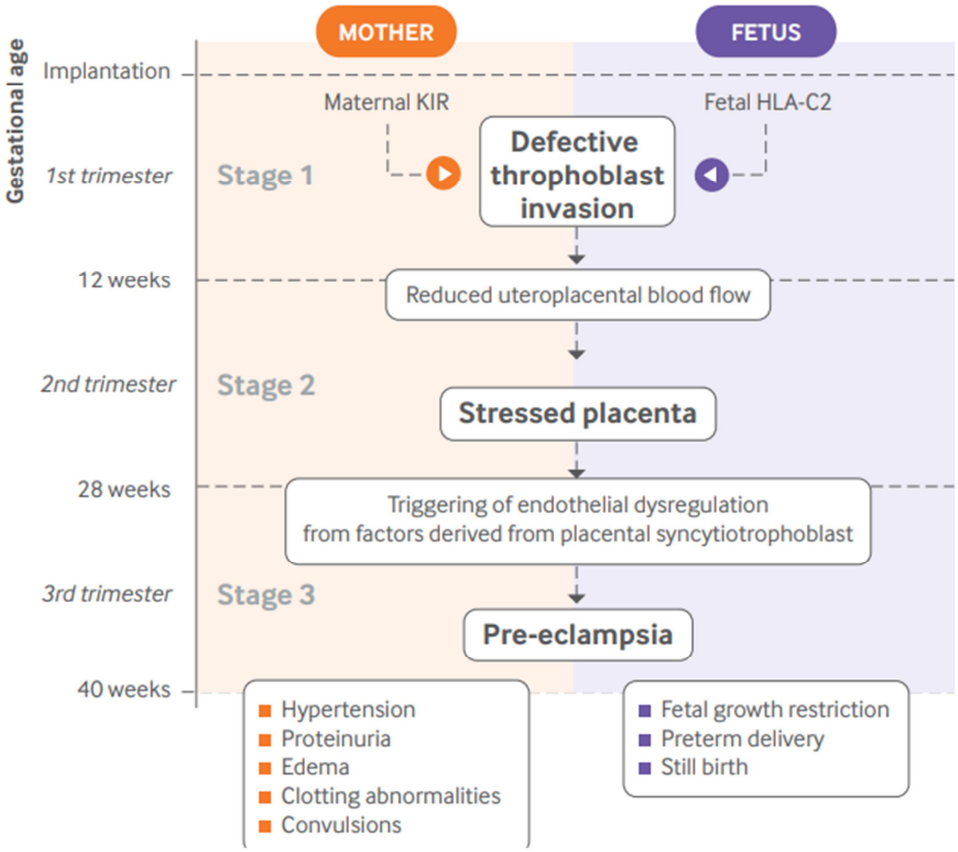


Figure 3-2 Pathogenesis of preeclampsia with subsequent affects on fetus and mother

The rheological and physiological consequence of the conversion of the maternal spiral arteries for uteroplacental blood flow during pregnancy

In a typical pregnancy, there's the development of significant arteriovenous shunts that remain present in the immediate postpartum period. Conversely, pregnancies affected by severe preeclampsia exhibit minimal arteriovenous shunts, resulting in narrower uterine arteries.

In normal pregnancy, the invasion of extra villous cytotrophoblasts extends from the decidua into the inner myometrium, leading to the creation of funnels at the outlets of spiral arteries. This differs from cases of severe preeclampsia. The depicted alterations are illustrated in the accompanying figure[36].

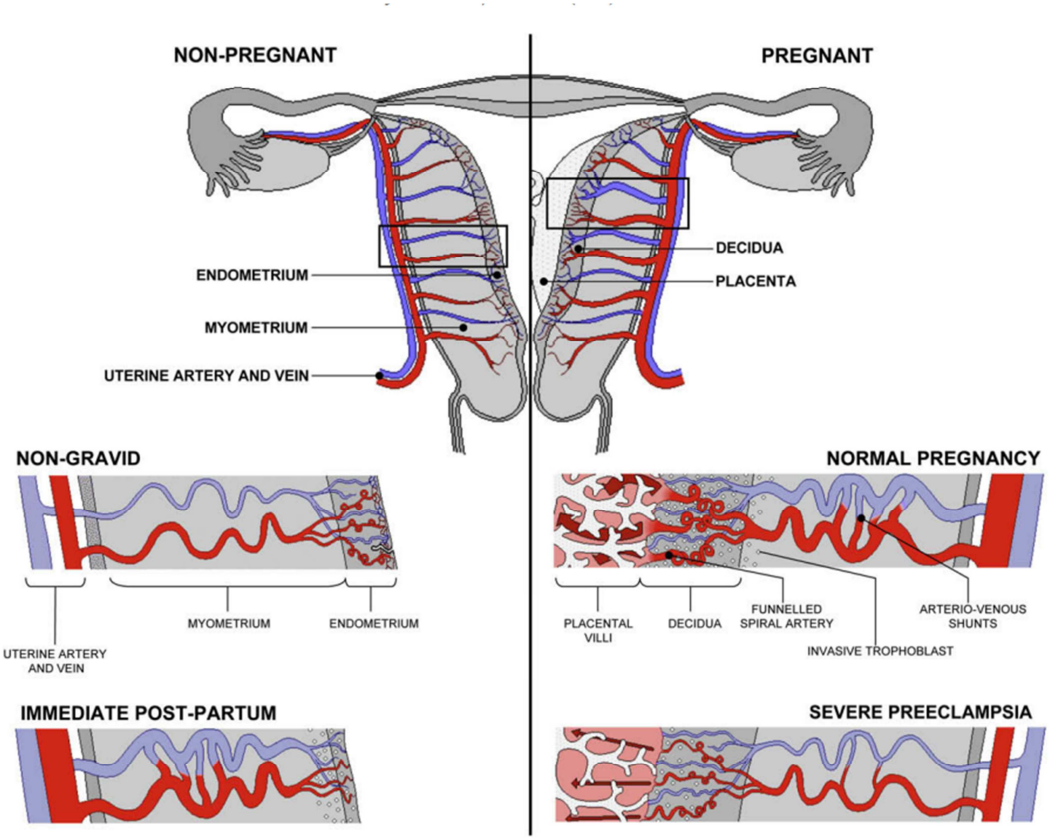


Figure 3-3 Diagrammatic representation of uterine and placental vasculature

Dilation of the distal segment in normal pregnancy will reduce the velocity of the incoming blood, and the residual momentum will carry the blood into the central cavity from where it will disperse evenly through the villous tree. In cases of Pathological pregnancy where there is insufficient or limited conversion, maternal blood will enter the intervillous space rapidly, resulting in turbulent flow.

The increased momentum will damage the structure of the villi, causing the rupture of the anchoring villi and the formation of echogenic cystic lesions, as indicated by ultrasound findings. Additionally, the shortened transit time will hinder oxygen exchange. Due to dislodgement of the trophoblastic microparticulate debris from the villous surface there is activation of maternal endothelial cell[37].

The persistence of smooth muscle also heightens the likelihood of spontaneous vasoconstriction and the occurrence of ischemia-reperfusion injury, leading to oxidative stress[38,39]

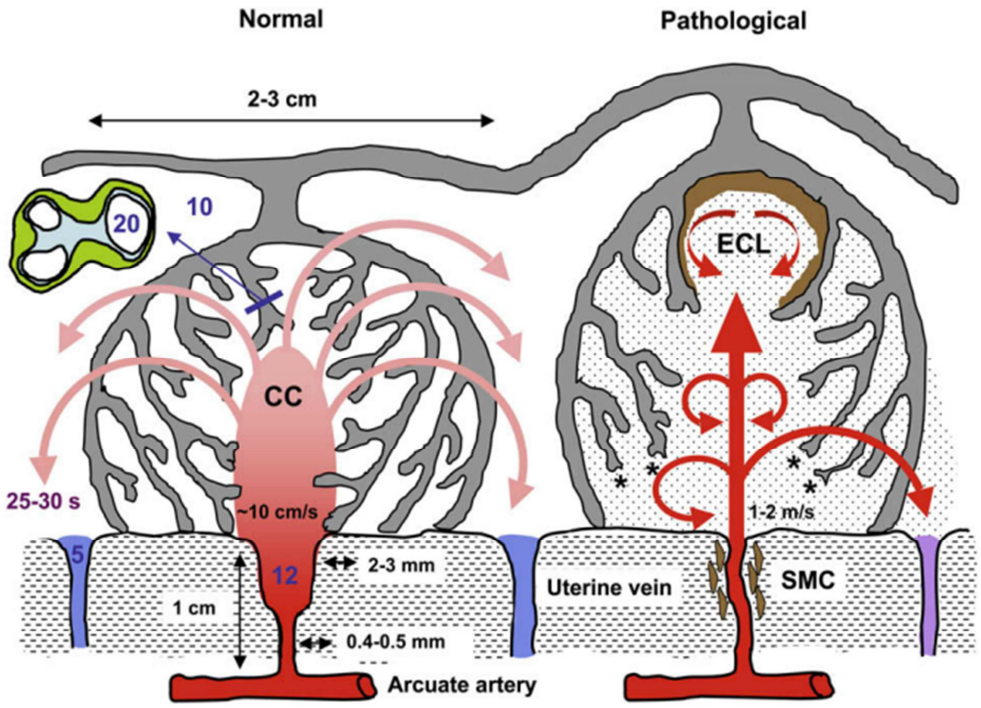


Figure 3-4 Diagrammatic representation of the effect of spiral artery conversion on the inflow of maternal blood into the intervillous space and on the lobule architecture .

Angiogenic imbalance

In individual which are prone to develope preeclampsia, trophoblastic tissue generates an excessive quantity of antiangiogenic factors, prompted by worsening hypoxia at the interface between the uterus and placenta.Soluble fms like tyrosine kinase 1 (sFlt-1) is a protein produced by placenta. It act by binding to the receptor binding domain of vascular endothelial growth factor (VEGF) and placental like growth factor (PLGF). Both PLGF and VEGF are proangiogenic protein .Increased maternal level of sFlt-1 inactivates and decrease circulating free PLGF and VEGF concentration resulting in endothelial cell dysfunction . The magnitude of sFlt-1 level correlates with disease severity .(40)

Soluble endoglin (sENG), a natural inhibitor of transforming growth factor 1 (TGF-1), is another extensively studied antiangiogenic protein in preeclampsia. Elevated levels of sENG are detected in the serum of women with preeclampsia two months before the onset of clinical symptoms, with its levels correlating with the severity of the condition and decreasing after delivery.

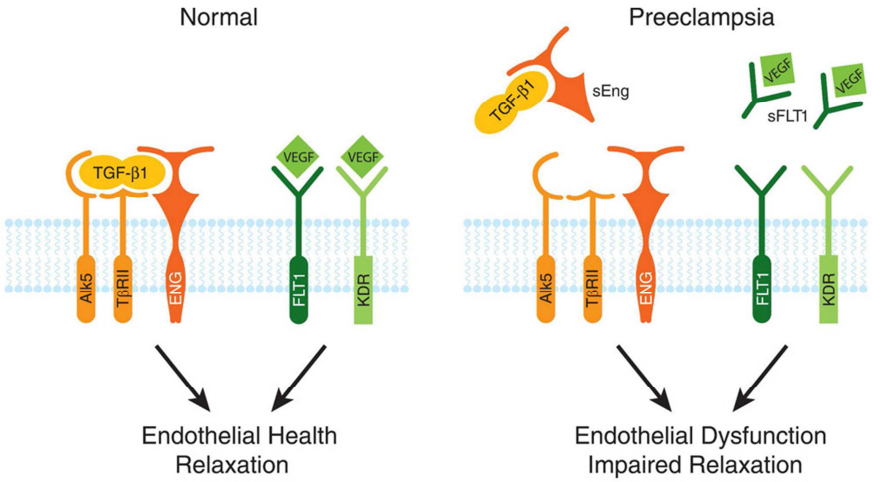


Figure 3-5 Endothelial dysfunction resulting due to sFLT-1 Seng

Vascular endothelial activation

Following the release of placental factors due to ischemia, a series of events is initiated, leading to endothelial cell damage. This dysfunction arises from heightened activation of leukocytes, interleukins, and tumor necrosis factor (TNF) in the maternal bloodstream. Oxidative stress manifests as lipid-laden macrophages, microvascular coagulation evident through thrombocytopenia, and increased permeability leading to edema and proteinuria[41].

Increased pressor response

In typical pregnancies, women tend to develop resistance to the blood pressure-raising effects of angiotensin 2. However, women at risk of developing preeclampsia exhibit heightened vascular responsiveness to both angiotensin and norepinephrine[41].

Nitric oxide

Nitric oxide, a strong dilator of blood vessels, is synthesized by endothelial cells from L-arginine. Preeclampsia is linked with reduced expression of endothelial nitric oxide synthase.

Prostaglandins

Proteinoids are also involved in the development of preeclampsia. In normal pregnancy, the reduced blood pressure response is attributed to heightened production of endothelial prostacyclin. However, in preeclampsia, prostacyclin production is mediated by phospholipase A2, while secretion of thromboxane A2 by platelets is simultaneously increased [41].

Genetic predisposition

It has been shown to influence the occurrence of preeclampsia. Even while both hereditary and environmental variables increase the risk of preeclampsia, having preeclampsia in first-degree relatives raises a woman's risk of preeclampsia by 2–4 times. It's possible that genetic variables play a role in the angiogenic imbalance seen in preeclampsia patients[42].

Certain researchers propose using the term "placental preeclampsia" for cases of preeclampsia characterized by abnormal placentation, and "maternal preeclampsia" to describe cases where the placenta is normal but there's an underlying chronic maternal condition associated with preeclampsia.

Preeclampsia develops in two parts (i) Initial phase which results in the generation of an unidentified signal due to a combination of factors like endothelial dysfunction, placental perfusion deficiency, defective implantation, high placental mass or oxidative stress; which are broadly termed abnormal placentation (ii) later phase where an abnormal maternal response results in clinical manifestation of preeclampsia characterized by hypertension and proteinuria.

Pathogenesis of preeclampsia is broadly divided into two phases,

- Abnormal placentation which occurs in the first trimester
- Development of maternal response, which develops in the 2^{nd} and 3^{rd} trimesters .

Factors contributing to abnormal placental development

1) Hypoxia – This was established by the upregulation of hypoxia–inducible transcription factor (TFs) and hypoxia-related genes in the placenta. HIF1 and HIF 2 are two proteins that are produced by the same oxygen-sensing mechanism and control the expression of hypoxia-induced genes such as erythropoietin, vascular endothelial growth factor (VEGF), and NO synthase circulation and hence oxygenation to the fetus increases, HIF1 expression in human placenta increases in the first trimester and thereafter decreases about 9 weeks. HIF 1 levels that remain consistently high may signal placental stress and the onset of preeclampsia. preeclampsia placenta has been demonstrated to overexpress HIF1 and HIF 2 and fail to downregulate their expression when exposed to oxygen.[43, 44]
2) Oxidative stress – In preeclampsia, antioxidant, and prooxidant mechanisms are not balanced, which is proposed to be the result of defective spiral artery remodeling (which causes repetitive ischemia-reperfusion injuries). Preeclampsia placenta has an imbalance of reactive oxygen species(ROS)-generating enzymes and antioxidants at the molecular level. ROS inhibits the wnt/catenin signaling pathway, which enhances trophoblast invasiveness. The transcription of antiangiogenic factors like Sflt 1 may be aided by oxidative stress.[45,46]
3) Role of heme oxygenase and other enzyme

   Heme oxygenase (HO), responsible for heme breakdown, plays a notable role in the vascular function of both the mother and fetus, as well as in the development and function of the placenta. There are three isoforms of HO, with HO-2 involved in spiral artery invasion and HO-1 strongly expressed in

non-invasive trophoblastic cells. In the rat model of reduced uterine perfusion pressure (RUPP), cobalt protoporphyrin induces a shift in the vascular endothelial growth factor (VEGF) gene[47].

Maternal syndrome

The second stage in the development of preeclampsia is characterized by an imbalance in proangiogenic and antiangiogenic factors, where an increase in antiangiogenic factors results in significant maternal consequences[48]

BIOMARKER IN PREECLALMPSIA

Due to unavailabity of adequate screening techniques and the serious consequences associated with the condition, all women at risk of preeclampsia must undergo thorough testing. The screening method developed by the Fetal Medicine Foundation, which considers maternal factors, mean arterial pressure, uterine artery pulsatility index, and PIGF, has been shown to be more effective than the screening methods typically recommended by national health authorities such as the National Institute for Health and Care Excellence and ACOG.

The ASPRE trial (Aspirin for evidence-based preeclampsia prevention) demonstrated the efficacy of such screening methods using PAPP-A (pregnancy-associated plasma protein A). This involved screening women in the first trimester, followed by administering 150 mg of aspirin or a placebo daily until 36 weeks gestation to those identified as high-risk. The trial resulted in a lower incidence of preterm preeclampsia compared to the placebo group, with an identification rate of 76.7% (138 out of 180 cases), including 43.1% for term preeclampsia, and a false

positive rate of 9.1%. These findings underscore the effectiveness of early screening utilizing plasma biomarkers and imaging studies in disease prevention[49].

An angiogenic biomarker could serve as a valuable tool in differentiating preeclampsia from other pregnancy-related conditions that exhibit similar signs and symptoms, such as chronic kidney disease, gestational thrombocytopenia, and chronic hypertension. This could potentially replace invasive diagnostic methods[50]. In a UK-based large-scale study, evaluating the plasma Sflt : PLGF ratio at 28 weeks gestation yielded a positive predictive value of 32% for preterm preeclampsia[51].

An elevated level of Sflt reduces bioavailability of VEGF and impare the production of nitric oxide which leads to vasoconstriction.VEGF induces NO production and neutralizes the effect of reactive oxygen species and vasoconstrictor signaling. The release of chemokine induces inflammation in maternal circulation and causes endothelial dysfunction. When platelets come into contact with damaged endothelium, it triggers the coagulation system, resulting in both increased production and consumption of platelets, ultimately leading to thrombocytopenia, a significant indicator of preeclampsia.(52,53)

Drugs affecting the platelet count

Numerous therapeutic medications have been linked to thrombocytopenia. Unlike immune-mediated thrombocytopenia, nonimmune drug-induced thrombocytopenia is characterized by the direct toxic effects of the drug molecule on platelets. This can result in impaired thrombopoiesis within the bone marrow or heightened platelet destruction in the bloodstream. Antineoplastic agents are a common cause of thrombocytopenia due to their direct toxicity to hematopoietic stem

cells. Specific drugs have been identified to directly induce antibody-independent apoptosis in platelets by triggering calcium signaling, mitochondrial depolarization, and exposure of phosphatidylserine. Examples of medications associated with nonimmune thrombocytopenia include chemotherapeutic agents, interferon-alpha, linezolid, bortezomib, thiazide diuretics, ethanol, tolbutamide, ganciclovir, tamoxifen, methotrexate, lovastatin, and vancomycin[54,55].

PLATELET COUNT AND PLATELET INDICES

PLATELETS

Platelets are also known as thrombocytes they have no nucleus, they are derived from the fragment of the cytoplasm of the megakaryocyte. Normal range of platelet is 1.5 lakho 4.5 lakh per microliter of blood .The major function of the platelet is hemostasis at the site of injured endothelium. The first step is the attachment of platelet outside the interrupted endothelium that is known as adhesion. After adhesion of the platelet, they secrete a chemical messenger that is known as activation.The third step is aggregation in which platelets connect through receptor bridges. Formation of platelet plug is known as primary hemostasis which in turn lead to activation of the coagulation cascade with deposition of fibrin is known as secondary hemostasis.(56)

PLATELET INDICES

Platelet indices are marker of platelet activation and these are parameter which are obtained as a part of an automated blood count. Platelet indices mostly are related to proliferation kinetics and platelet morphology. The most commonly assessed Platelet indices are mean platelet volume, platelet distribution width, and plateletcrit.

MPV (mean platelet volueme)

The most frequently investigated platelet parameter is MPV,, it depicts average size of the platelet . It normal value is between 7.2 fl – 11.7 fl . If mean platelet volume is greater than 13fl this indicate hyper destruction of the platelet , so there is release of new platelet which are bigger and there is increase in the activity, if the mean platelet volueme is <8 fl it denotes hypoproduction. Many factor like race , age , smoking , alcohol consumption and physical activity can alter MPV . (57,58)This has been analysed as a potential biomarker of the patient prognosis , in most of the studies its higher value is associated with worst clinical outcome .High level of platelet indicies above normal range are found in medical condition like immune thrombocytopenic purpura, diabetes mellitus, Low mean platelet value has been linked to various conditions including diabetes-related retinopathy and nephropathy, septic shock, heart disease, malignant tumors, and complicated acute appendicitis. Conversely, it's associated with milder inflammation such as rheumatoid arthritis. In acute cholecystitis, non-complicated acute appendicitis, and threatened preterm labor, its levels tend to be below normal[59,60].

Platelet distribution width indicates platelet anisocytosis, reflecting both the size and distribution of platelets produced by megakaryocytes. Its levels increase during platelet activation. Several studies have shown that this parameter typically ranges from 9% to 14% in healthy individuals. Alterations in PDW are observed in patients with various diseases, making this parameter a potential biomarker. Multiple studies have shown a proportional relationship between PDW and MPV. However, there are instances where PDW increases while MPV decreases under specific conditions.

Its value is above normal in conditions such as ST-elevation myocardial infarction, threatened preterm labour, vaso-occlusive crisis vaso-occlusive crisis.its level are decreased in non-malignant tumours.(61,62)

Plateletcrit (PCT) assesses the total platelet mass as a percentage of blood volume. Its normal range falls between 0.22% and 0.24%. It serves as an efficient screening tool for identifying abnormalities in platelet quantity. PCT is not linearly associated with platelet count and indices. Its levels rise in conditions such as active Crohn's disease and decrease in immune thrombocytopenic purpura[63]. Blood-based parameters, owing to their convenient accessibility and cost-effective measurement methods, are increasingly considered as promising new biomarkers for various diseases.

Studies examining the relationship between platelet parameters and preeclampsia have produced inconsistent findings. While certain well-conducted studies revealed a significant association between preeclampsia and platelet count, as well as between preeclampsia and MPV, other studies reported no significant variations in these parameters between preeclamptic and normotensive groups[64,65,66,67,68.69].

MANAGEMENT OF PREECLAMPSIA AND ECLAMPSIA

The revised NICE guideline advises treating hypertension if systolic blood pressure exceeds 140 mmHg or diastolic blood pressure is above 90 mmHg. The target blood pressure, once antihypertensive medication has been initiated, would be 135/85 mmHg (70). Previously the treatment would be started when the blood

pressure would be greater than 150/100 mmHg reflecting evidence from the CHIPS trial (Control of hypertension in pregnancy study).

Labetalol is the first line of management for hypertension in pregnancy, second line is nifedipine.If both nifedipine and labetalol are not sutibale , Methyldopa is recommended.

Termination of pregnancy -Both the NICE Guideline and ACOG recommend terminating pregnancy at 37 weeks of gestation for women with preeclampsia. Prior to 34 weeks, expectant management is preferred as induced preterm delivery before 34 weeks is linked to adverse neonatal outcomes such as respiratory distress.The HYPIPAT-2 Randomized Controlled Trial, comparing immediate delivery versus expectant monitoring for hypertensive disorders of pregnancy between 34 and 37 weeks, found that routine expedited delivery is not warranted[71].

- In a 2015 observational analytical study conducted in the Department of Obstetrics and Gynecology at Ambedkar Hospital, Pandit Jawaharlal Nehru Medical College, involving 150 women with a mean age of 26.3 ± 5.3 years, it was observed that mean platelet count and mean plateletcrit decreased significantly, while mean platelet volume and mean platelet distribution width (PDW) increased significantly with the severity of the disease. Additionally, it was noted that 66.7% of preeclampsia cases and 51.4% of eclampsia cases did not have thrombocytopenia. Among these women, a significantly higher number of eclampsia patients exhibited a decrease in platelet crit value and an increase in PDW[73].

- A prospective observational study was done in the pathology department with a study group comprising 235 pregnant women with a mean age group of 24±3.5 years in preeclampsia without severe features and 23±3.6 years in normal pregnancy. It also shows a decrease in platelet counts in preeclampsia with severe feature group 181±38.9, preeclampsia without severe feature 210±55.4 compared to platelet count in normal pregnancy group 355.59± 79 with a p-value of <0.001. It was observed that platelet value decreased as the severity of disease increased and was statistically significant when compared with preeclampsia cases without severe feature group when compared to normal pregnancy.(74)
- A case-control study conducted in 2016 on 120 women by Muneera A, Rafi S, et alat Qassim Hospital, Saudi Arabia, found in their study no significant difference in PDW and MPV between preeclamptic women compared with control, both PC and PC/MPV ratio were significantly lower in preeclampsia. PC of 248x103/uL was found for the diagnosis of preeclampsia with a p-value of 0.001. The PC/MPV cutoff was 31.2 with a p-value of 0.035 with the area under the ROC curve of 62.2%.(76)
- A retrospective study done in 2002 in Cukurova University Turkey among 2245 cases found that in preeclampsia there was a statistically significant relationship between maternal complication and low platelet level. (77)
- A retrospective case control done in 2004 among 199 women with low platelet count at sororal university , hospital of the negev found that moderate to severe maternal thrombocytopenia point to higher degree of severity of the primary disease , which increases perinatal complication (as preterm deliveries , Apgar score<7 in 5 min , IUGR , Stillbirth .(78)

- A study done by gioia et al found that in pregnant women with preeclampsia when MPV> 10fl this was significantly related to compromised foetus status (as neonate need oxygen support more than 2 days or intubation and/ or PH < 7.2 at umbilical blood gas analysis(79)
- A study by Muluken W, Fikir A in 2022 at the university of Gondor. A total of 126 pregnant women were analyzed out of which 63 were preeclamptic women. They found that Mean platelet volume, platelet distribution width, and platelet large cell ratio (P-LCR) were higher in preeclampsia women. MPV can differentiate preeclampsia patients from the normotensive patients at a cutoff value of >12.10 fl with 87.3% specificity, While platelet count can indicate preeclampsia at cutoff value of < 176.5 x 109 /L with 87.3 % specificity.
- A Retrospective analysis done among 936 pregnant women they found that the incidence of thrombocytopenia in pregnancy was 11.11%(104/936) . Thrombocytopenia represented a risk factor for premature delivery – highest risk for severe thrombocytopenia (RR=8.9 , p <0.001) . Thrombocytopenic preeclampsia or HELLP syndrome is associated with the highest rate of prematurity. Thrombocytopenia also represented a risk factor for low birth weight newborns, especially severe thrombocytopenia – birth weight 2047.50±938.98gm (p value <0.002) versus 3224.86±490 gm in control. Again thrombocytopenic preeclampsia was significantly associated with low birth weight newborn , with mean weight of 2462±794.54gm versus 2932±708.91gm in thrombocytopenic pregnancies respectively 3224.86±496 gm in normal pregnancies.(80)

MATERIALS AND METHODS

4.1 Study design

The present study was a hospital based descriptive observational study-(comparative cross-sectional study) to find an association between platelet count and platelet indices in pregnancy with preeclampsia and eclampsia. This study was conducted at KAHER'S Dr. Prabhakar Hospital, Belagavi for a period of 12 months. Data and samples were obtained from pregnant mothers presented in 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 KAHERS'S Dr. Prabhakar Kore Hospital, Belagavi, Karnataka. The hospital is a clinical training facility that provides free health care to the underprivileged in basic specialties. KAHER'S Dr. Prabhakar Kore Hospital is recognized by the medical council of India.

4.3 Study period

The study was conducted for a period of 12 months (February 2023 – March 2024). The study period included enrolment of participants , data collection , analysis and reporting.

4.4 Study Population

The study population consisted of antenatal women diagnosed with preeclampsia and eclampsia presented to the labor room at the Department of obstetrics and Gynaecology of KAHERS'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

$$n = \frac{2\left(Z_{1-\frac{\propto}{2*k}} + Z_{1-\beta}\right)^2}{f^2}$$

$$where, f = \left(\frac{\min(|\mu_i - \mu_j|}{\sigma}\right)$$

where, μ i is mean of i th group, μ j is mean of jth group, is the common error variance, is Z score adjusted for α level of significance (Bonferroni Correction), k is the number of pairwise comparisons and value is Z score for (1-β) % power.Assuming between group effect size to be 0.4 for platelet distribution width (PDW) according to (Singh et al) , at 5% level of significance, and 80% power, the sample size is obtained to be 44 subjects per group.Hence, total sample size required is 44 x 3 = 132 subjects. As sample size increases, accuracy of result also increases.

4.6 Selection Criteria

4.6.1 – Inclusion criteria

- **Group 1**
- **Normotensive -** Normal pregnant women of more than 20 weeks of gestational age.
- **Group 2**
- **Preeclampsia without severe features** —women with Systolic BP >=140 and or Diastolic BP >=90 mmHg on 2 occasion at least 4 hours apart after 20 week period of gestation along with presence of proteinuria.
- **Group 3**
- **Preeclampsia with severe features -** Systolic blood pressure >=160 mmHg or diastolic blood pressure >= 110 mmHg on 2 occasions 15 min apart or sign of maternal organ dysfunction with or without proteinuria.
- **Eclampsia**—Women with BP >=140/90 mm Hg with convulsions or coma.

4.6.2- Exclusion criteria

- Chronic hypertension.
- Gestational Hypertension.
- HELLP
- Known case of epilepsy .
- Pre- Existing renal disease.
- Diabetes Mellitus
- ITP, TTP, APLA, SLE
- Drugs affecting platelet count.

4.7 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. Pre-eclampsia without severe features was defined as an SBP>= 140mmHg and/or DBP>=90 mm Hg associated with proteinuria. Preeclampsia with severe features is defined as systolic blood pressure of >= 160 mmHg or diastolic blood pressure of >=110 mmHg on two occasions 15 minutes apart or signs of maternal organ dysfunction with or without proteinuria. Participants were also classified as PE with severe features if any of the premonitory symptoms such as occipital headache, visual disturbances, and epigastric pain, were present along with hypertension. The patients who were presenting with generalized – tonic-clonic convulsions in the absence of the other cause were diagnosed as eclampsia. Normotensive patients were included who were matched with pre-eclampsia without severe feature, preeclampsia with severe features and eclampsia patient.

After identifying the study participant, written informed consent was obtained for enrolment in the study.

Sampling techniques

After obtaining written informed consent and following all aseptic precautions, the anterior cubital vein was venipunctured into EDTA and a plain bulb. The samples will subsequently undergo testing for CBC, Platelet indices, LFT, and RFT.

Details of methods employed for sampling and investigation are as follows:

4.7.1 Socio-demographic characteristics

Data on socio-demographics like age, obstetrics history, and detailed history about other associated conditions were collected via interview, and structured questionnaires were used before blood samples were collected for clinical investigation. Findings of clinical examination and subsequent systemic examination were recorded on a predesigned and pretested proforma.

4.7.2 Complaints and history of presenting complaint

Duration of abdominal pain, duration of bleeding per vagina if present, duration of leak per vagina if present, duration of Data on period of amennorhea , last menstural period, expected due date, perception of fetal movement . Imminent signs of preeclampsia/eclampsia (headache , blurring of vision, epigastric pain) seizures (and in detail in case of seizures such as number of episodes, duration, loss of consciousness and lucid interval) and other associated medical history were collected by interview/examination of the patient ..

Obstetric history included information on married life/consanguinity, obstetric score (gravida,para,living, abortion, and death), data of last child birth and history of previous pregnancy which were noted down for study participant in the proforma .

Menstural history consisting of details on age of menarche, previous menstural cycle and period of gestation were collected from the study participants.

Past medical and surgical history ,family history, personal history were also assimilated from the patient .

4.7.3 Clinical examination

1) General physical examination

Height , weight , bodymass index (BMI) , pulse rate , blood pressure , pallor , icterus , pedal edema were noted for each of the participant .

2) Systemic examination

Examination of cardiovascular system , respiratory system , per abdomen examination for the size of the uterus , tenderness/tense , presentation and fetal heart sound were carried out for the study participant .

Per speculum examination for the active vaginal bleeding , leak were done and vaginal examination for the consistency of cervix , position , effacement , dilation and station were recorded.

4.7.4 Clinical diagnosis and clinical investigation

Clinical investigation includes blood grouping/rh typing , complete blood count, peripheral smear , urine routine examination , serological testing (HIV , HBsAg, VDRL) , PIH profile including platelet count , 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) and thrombin time (TT).

Obstetric ultrasound (Doppler) and fundoscopy were also carried out as a part of clinical investigation .

4.7.5 Management / Interventation

Details of the antihypertensive used , MgSO4 regimen , and other medication prescribed for the condition were collected via proforma .

4.7.6 Delivery related information

Data on the date of delivery , mode of delivery , induction /augmentation , duration of labour, intrapartum complication, blood loss , indication for lower segment Cesarean section (LSCS) and intraoperative finding were recorded for the study participants.

The placenta was observed post-partum and information on the weight of placenta , retroplacental clot was noted .

4.7.7 Maternal outcome

Information on the following peripartum complication was collected and recorded for the study participant .

- Central nervous system : Convulsion , Posterior reversible encephalopathy syndrome (PRES) and stroke
- Hematological complication – DIC
- Respiratory system – Respiratory distress, pulmonary edema .
- Hepatic dysfunction
- Renal complications

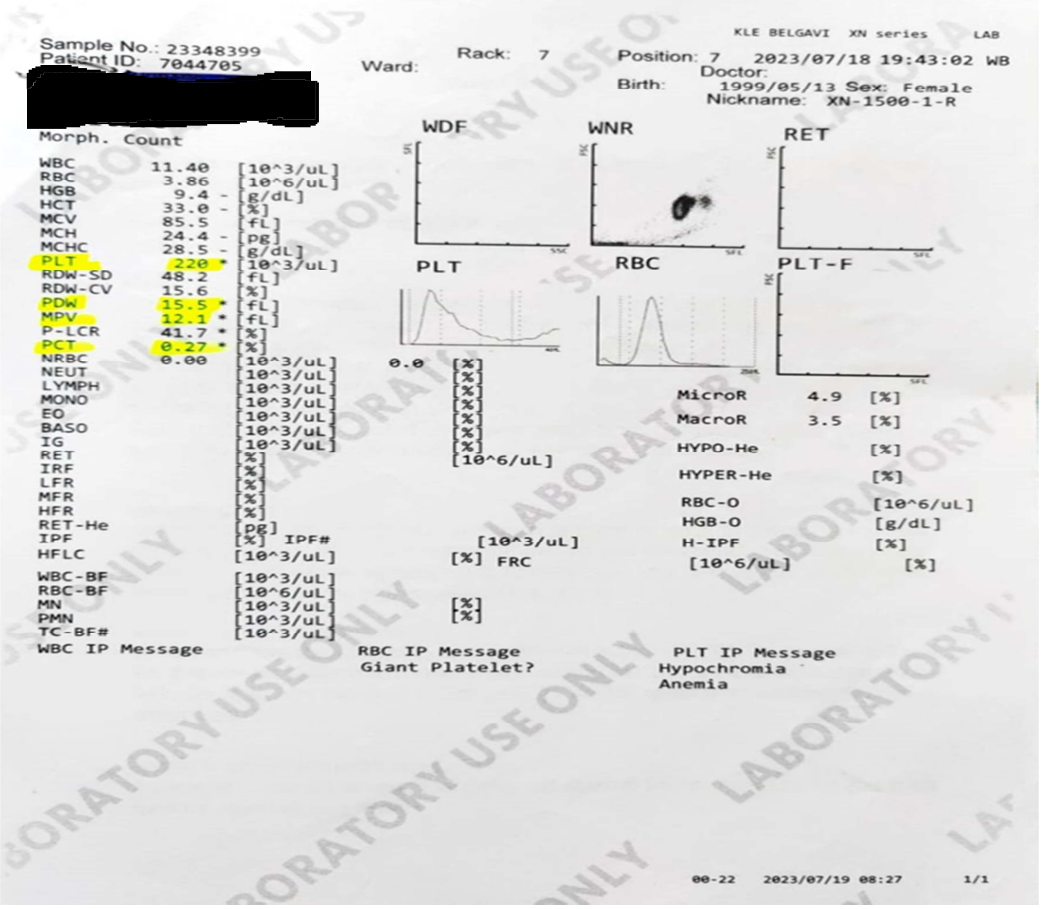
- HELLP syndrome (Haemolysis, elevated liver enzymes, low platelet count).
- Abruptio placenta
- Cerebro- vascular complication.

Other information related to admission to the intensive care unit (ICU) , duration of hospital stay , cause of death if any collected .

4.8 Method of Estimation of Platelet count and platelet indices

Platelet count and platelet indices was estimated by a standard technique using an automated analyser. 2 ml of venous blood was collected in EDTA bulb from patient. The samples were analysed in SYSMEX XN -1500/XN 10 and a blood report was obtained.

The following is the lab report obtained from which data of platelet indices is taken



4.9 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. Continuous variables given in Mean ± SD / Median (Min, Max) form. Normality of variable is checked by Shapiro Wilk test and QQ plot. Chi square test is used to check the association of categorical variables with groups. One way ANOVA is used to compare the mean of variables over groups. Tukey's HSD is used as post hoc analysis. Kruskal Wallis test is used to compare the distribution of variables over

groups. Dunn test is used as post hoc analysis. Applicability of various parameters to predict non severe PE, and severe PE and Eclampsia is checked by Logistic regression and Receiver Operating Characteristic (ROC) curves. Cut off values are obtained by simultaneously Youden index. Spearman's rank correlation test is used to check the correlation of different parameters with SBP and MAP. P-value less than or equal to 0.05 at 95% confidence interval indicates statistical significance.

4.10 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 participants 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, KAHER's Dr Prabhakar kore hospital, Belagavi, Karnataka in the prescribed format.

5 RESULTS

The study was conducted at the department of obstetrics and gynecology of KAHER's Dr. Prabhakar Kore Charitable hospital , Belagavi, Karnataka for a period of one year . This observational study was conducted among antenatal women with preeclampsia or eclampsia , admitted to the labour was from February 2023 to march 2024 , after the study was approved by the institute ethics committee.

Data obtained from structured questionnarires was analysed using graph pad prism version 9.0 and MS Excel. Continuous quantitative variables have been given as Mean ± SD / Median (Min, Max) form. Categorical data have been expressed in terms of frequencies and percentages and have been expressed using Chi-square test . Krushal -Wallis one way analysis of variance was used to compare distribution between the groups and pre and post treatment measures . P value < 0.05 was considered as significant in all the cases.

5.1 Recruitment of study participants

The total number of mother screened and enrolled for this study was 174. The details of inclusion of study participant have been given in figure 5-1. The age of the participant ranged from 18 to 40 years with the mean age of 25.86 ± 4.39 years. Average body mass index of study participant was found to be 24.18 ± 1.74 (ranging from 20 to 29.5) .

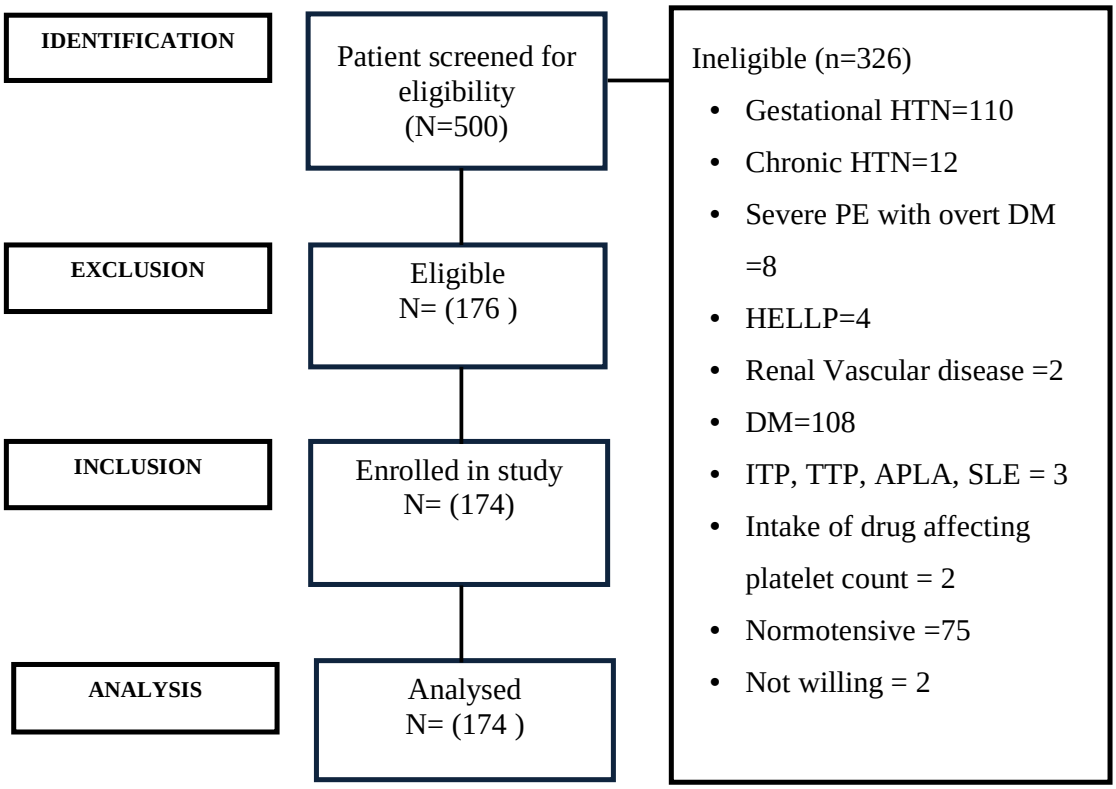


Figure 5-1 Enrolment of study participant

5.2 Distribution of study participants based on demographic and obstetric parameters

The distribution of study participant based on demographic and obstetric parameter has been given in table 5-1

Table 5-1 : Distribution of study participants based on demographic and obstetric parameter

S.NO	Parameter	Subgroup	No of study participant	Percentage
1.	Age (years)	<20 years	9	5.17%
		21-25 years	89	51.15%
		26-30 years	50	28.74%
		>30 years	26	14.94%
2.	Parity	Primigravida	94	54.1%
		Multigravida	80	45.9%
3.	Gestational age	<37 weeks	73	41.95%
		>37 weeks	101	58.05%
4	BMI	18.5-24.9	110	63.22%
		25-29.9	64	36.78%

(1) Age: Maximum number of the study participant were in the age group of 21 to 25 years (51.15%), followed by the age group 26 to 30 years (28.74%). A smaller proportion of subject were younger than 20 years (5.17%) or older than 30 years (14.94%).

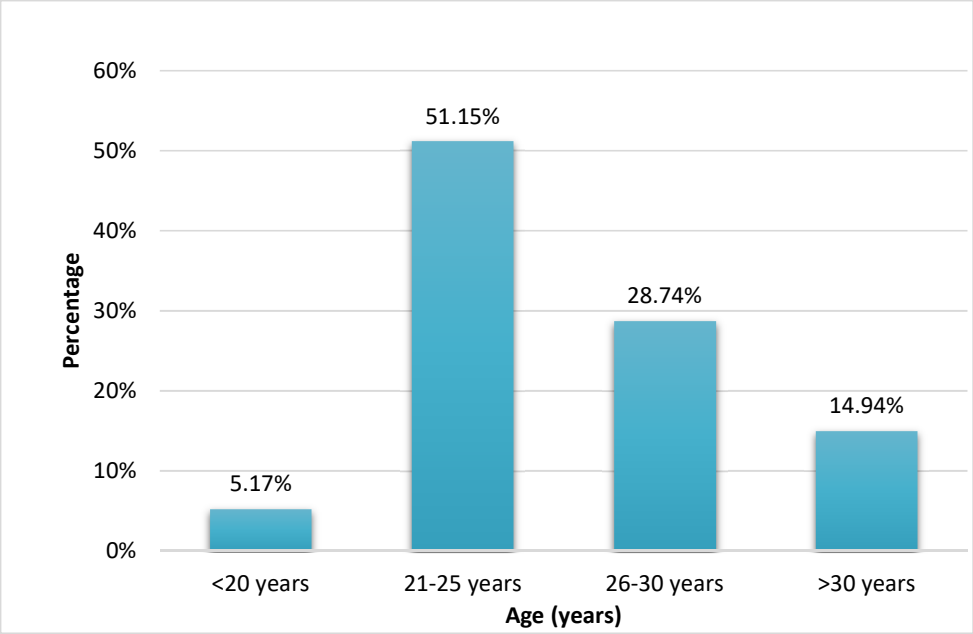


Figure 5-2 Distribution of study participant based on age

(2) Distribution of study participant based on parity

Maximum number of study participant were primigravida (54.1%). The percentage of multigravida is 45.9%.

(3) Gestational age- Average gestational age of the study participant was observed to be 35.68 ±3.68 weeks . Ranging from 25 weeks to 41 weeks . Gestational age of maximum proportion of study participants 58.05% was < 37 weeks .While 41.95% of the women delivered after 37 weeks .

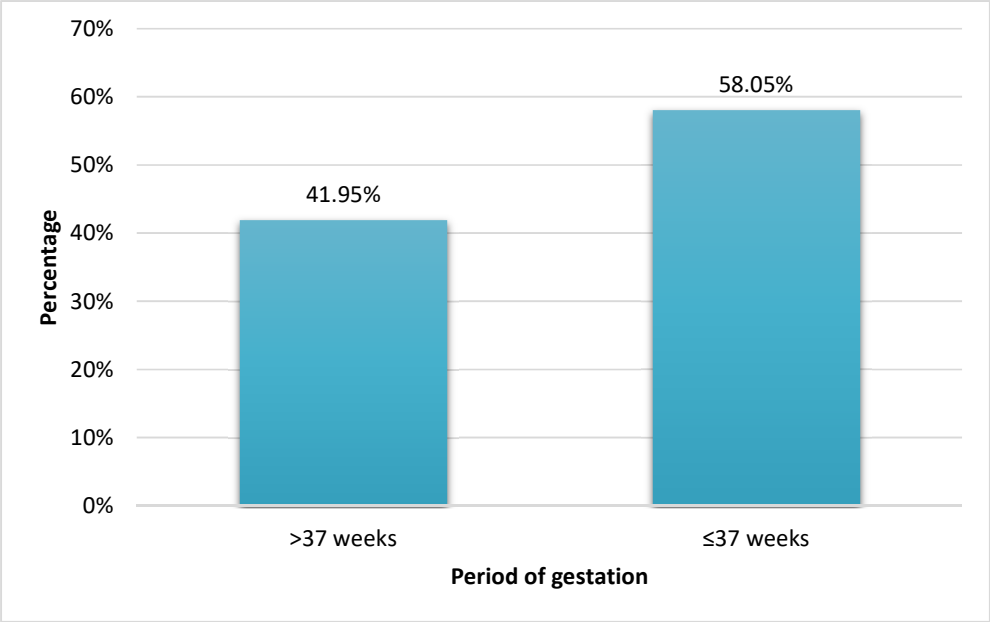


Figure 5-3 Distribution of study participant based on period of gestation

(4) BMI - The distribution of BMI among participant shows that most subjects fell within the normal weight range (18.5-24.9 BMI, 63.22%), while a significant proportion were in the overweight range (25-29.9 BMI, 36.78%). The mean BMI was 24.18 with a standard deviation of 1.74, and the median BMI was 24 (ranging from 20 to 29.5).

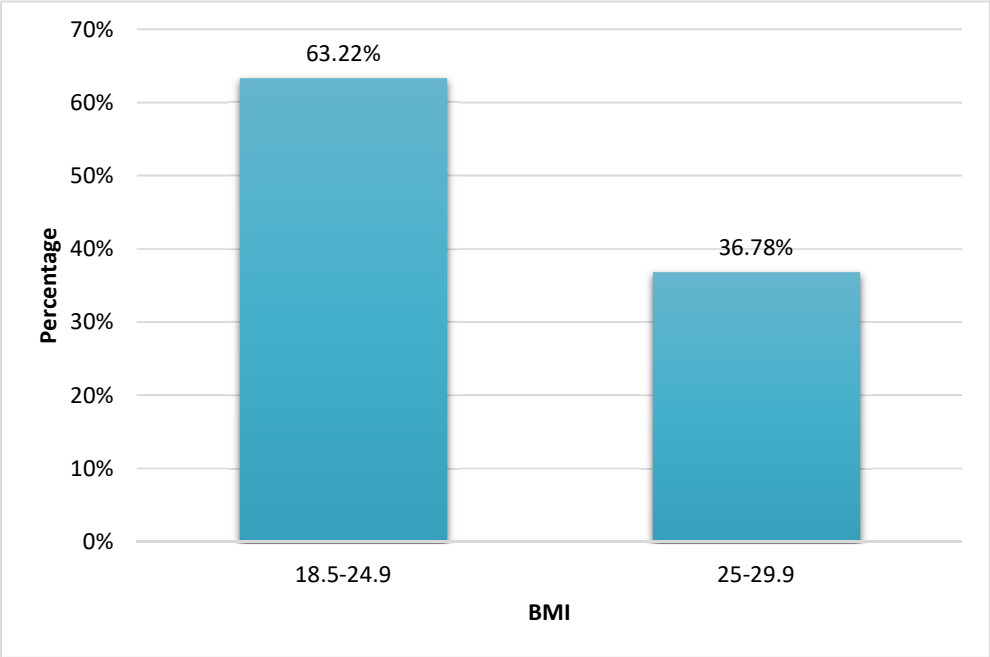


Figure 5-4 Distribution of study participant according to BMI

5.3 Distribution of study participant based on severity of condition

Among a total of 174 subjects, 60 participant (34.48%) belonged to group 1 (normotensive), 55 participant (31.61%) belonged to group 2 (PE without severe features), and 59 (33.91%) participant belonged to group 3 (eclampsia +PE with severe features).

Table 5-2 Distribution of subjects according to various group

Groups	Number of subjects (%)
Group 1	60 (34.48%)
Group 2	55 (31.61%)
Group 3	59 (33.91%)

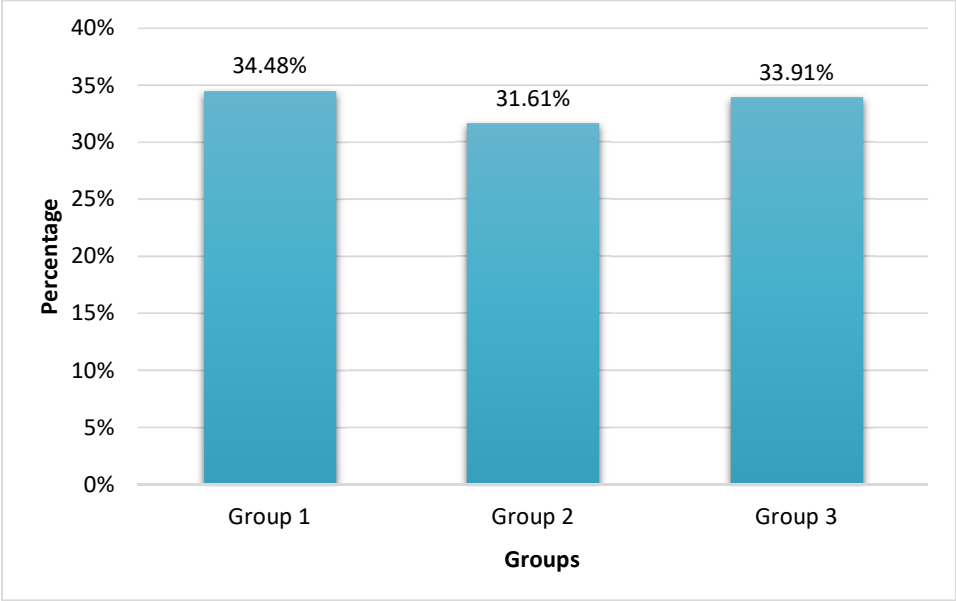


Figure 5-5 Distribution of subjects according to groups

5.4 Distribution of study participant based on clinical parameters

Average systolic and diastolic blood pressure of the study participant were 139.36 ±18.5 mmHg and 88.82 ± 11.5 mmHg respectively (Table 5-2) . The mean arterial blood pressure of the study participant was 105.69 ± 13.61 mmHg.). Proteinuria levels varied, with the majority of participant showing no proteinuria (32.76%), followed by trace (2.3%), 1+ (28.16%), 2+ (24.71%), 3+ (9.2%), and 4+ (2.87%) levels.

Table 5-3 Distribution of study participant based on clinical parameter

SI.NO	Parameter	Mean ± SD	Median (IQR)
1.	Systolic blood pressure	139.36 ± 18.5	140 (110, 220)
2.	Diastolic blood pressure	88.82 ± 11.5	90 (60, 120)
3.	Mean arterial pressure	105.69 ± 13.61	106.6 (76.6, 153.3)
4.	Protenuria	Subgroup	N %
		Nil	57 (32.76%)
		Trace	4 (2.3%)
		1+	49 (28.16%)
		2+	43 (24.71%)
		3+	16 (9.2%)
		4+	5 (2.87%)

SD- Standard deviation , IQR- Interquartile range

5.5 Comprasion of different demographic and obstetric parameter over groups

Table 5-4 Shows the distribution of study participant based on demographic and obstetrics parameter over different groups . Major proportion of study participant were primigravida 20.6% in group 3 which is preeclampsia with severe feature with eclampsia .

Table 5-4 Distribution of study participant based on demographic and obstetrics parameter over groups .

Variable	Subcategory	Group 1 (Normotensive)	Group 2 (Preeclampsia without severe feature)	Group 3 (Preeclampsia with severe feature)
1. Age	<20 years	4 (2.29%)	1 (0.5%)	4 (2.29%)
	21-25 years	34 (19.54%)	26 (14.94%)	29 (16.6%)
	26-30 years	15 (8.62%)	18 (10.3%)	17 (9.77%)
	>30 years	7 (4.02%)	10 (5.7%)	9 (5.17%)
2.Parity	Primigravida	28(16%)	30 (17.2%)	36(20.6%)
	Multigravida	32(18.3%)	25(14.3%)	23(13.2%)
3. Gestational age	>37 weeks	24 (13.7%)	34 (19.5%)	15 (8.6%)
	≤37 weeks	36 (20.6%)	21 (12%)	44 (25.2%)
4.BMI	18.5-24.9	48 (27.5%)	31 (17.8%)	31 (17.8%)
	25-29.9	12 (6.8%)	24 (13.7%)	28 (16%)

5.6 Distribution of study participants based on clinical parameter in different groups

Table 5-5 shows significant correlation between systolic blood pressure and severity of preeclampsia p- value <0.001 . In Group 1 that is normotensive the mean of SBP is 119.9 mmHg , in group 2 that is preeclampsia without severe feature the mean of SBP is 141.75 mmHg whereas in group 3 that preeclampsia with severe feature along with antepartum eclampsia it is 156.92 mmHg . Significant difference is observed in the distrubition of DBP, MAP and proteinuria over all pairs of group (p-value <0.001).

Table 5-5 Distribution of study participant base on clinical parameter in different groups .

S.no	Variables	Subcategory	Group 1 (Normotensive)	Group 2 (Preeclampsia without severe feature)	Group 3 (Preeclampsia with severe feature)	P-Value
1.	SBP		119.9 ± 5.55	141.75 ± 3.57	156.92 ± 16.35	**< 0.001^{K}***
2	DBP		77.13 ± 5.7	90.8 ± 3.5	98.85 ± 10.15	**< 0.001^{K}***
3.	MAP		91.3 ± 5.01	107.7 ± 3.08	118.47 ± 11.56	**< 0.001^{K}***
4.	Proteinuria	NIL	53 (88.33%)	3 (5.45%)	1 (1.69%)	**< 0.001^{MC}***
		Trace	1 (1.67%)	1 (1.82%)	2 (3.39%)	
		1+	6 (10%)	32 (58.18%)	11 (18.64%)	
		2+	0	17 (30.91%)	26 (44.07%)	
		3+	0	2 (3.64%)	14 (23.73%)	
		4+	0	0	5 (8.47%)	

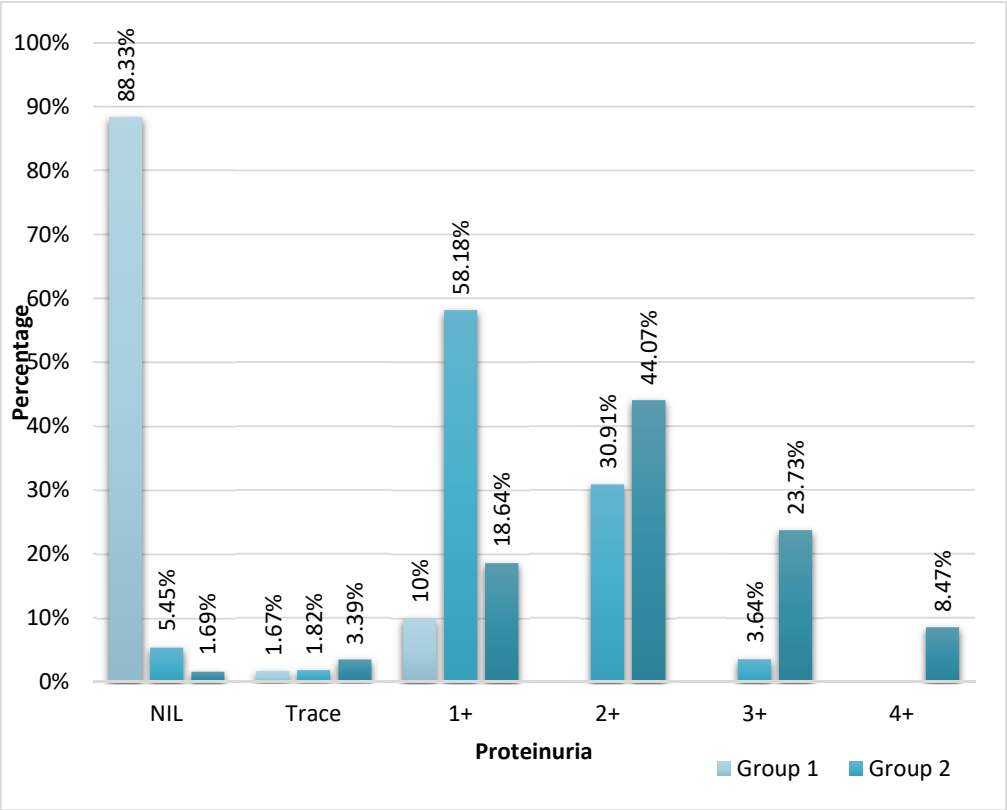


Figure- 5-6 Distribution of study participant based on proteinuria over different groups .

5.7 Comparison of Platelet count and platelet indices over different study groups

Significant correlation between PC/MPV and severity of preeclampsia was found (p value < 0.001) . In Group 1 with include normotensive participants mean of PC/MPV =25.47 where as in group 2 and group 3 that values are 21.8 , 21.06 respectively . There is a decreasing trend of platelet count and plateletcrit over group 1 group2 and group 3 , However it is statistically not significant .Also there is a increasing trend in values of PDW and MPV seen in group 1 , group 2 and group 3 which is statistically not significant .

Table 5-6 Comparison of platelet count and platelet indices over different study groups

S.no	Variable	Group 1		Group 2		Group 3		P value
		Mean ±SD	Median (IQR)	Mean ±SD	Median (IQR)	Mean ±SD	Median (IQR)	
1.	Platelet	2.42 ± 0.54	2.32(1.37-3.91)	2.28 ± 0.82	2.19(0.57-4.25)	2.27±0.81	2.25 (0.58-4.34)	0.4376[K]
2.	PDW	11.82 ±2.37	11.55 (7.9-17.7)	12.48 ± 2.28	11.8 (8.4-20.5)	12.91± 2.87	11.8 (8.2-19.9)	0.1700[K]
3.	Plateletcrit	0.25±0.06	0.24(0.13-0.4)	0.24 ± 0.08	0.23 (0.06-0.49)	0.23±0.07	0.23 (0.07-0.37)	0.3534[K]
4.	MPV	10.28 ± 1	10.1 (8.7 – 12.6)	10.3 ± 0.9	10.3 (8.7-12.5)	10.47 ± 1.18	10.2 (7.8 – 13.2)	0.5420[A]
5.	PC/MPV	25.47 ± 5.23	24 (13.6-40)	21.8 ± 7.53	20.7 (5.7-41.95)	21.06 ± 7.32	22 (6.3-37.6)	< **0.001[K]***

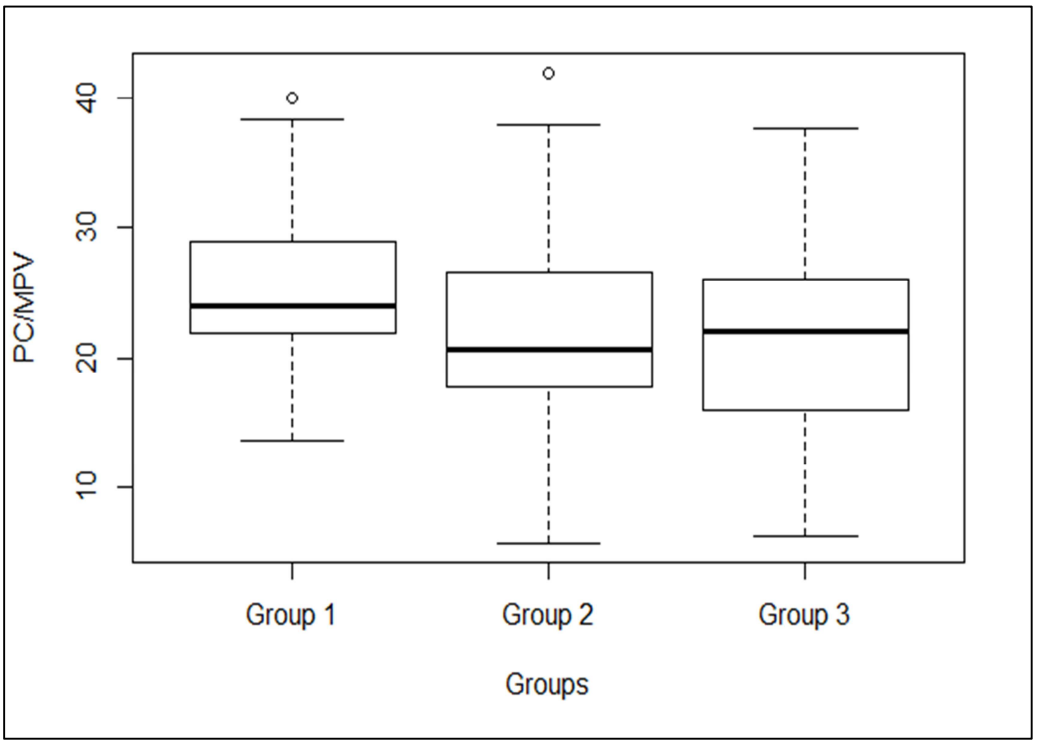


Figure 5-7 Mean plot of PC/MPV over groups

5.8 Comparison of incidence of change in platelet count and platelet indices in different study groups

In group 2 that is preeclampsia without severe feature 85.7% have normal platelet count whereas in group 3 that is preeclampsia with severe feature and eclampsia 79.66% have normal platelet count .

Table 5-7 Comparison of incidence of change in platelet count and platelet indices in different study group

S.no	Variables	Subcategory	Group 1	Group 2	Group 3
1.	Platelet	Decreased	1 (1.67%)	8 (14.55%)	12 (20.34%)
		Normal	59 (98.33%)	47 (85.45%)	47(79.66%)
2.	PDW	Increased	12 (20%)	15 (27.27%)	21 (35.59%)
		Normal	48 (80%)	40(72.72%)	38 (64.4%)
3.	Plateletcrit	Decreased	18 (30%)	24 (43.64%)	22 (37.29%)
		Normal	42(70%)	31(56.3%)	37 (62.7%)
4.	MPV	Increased	26 (43.33%)	24 (43.64%)	24(40.68%)
		Normal	34(56.67%)	31 (56.36%)	35(59.32%)

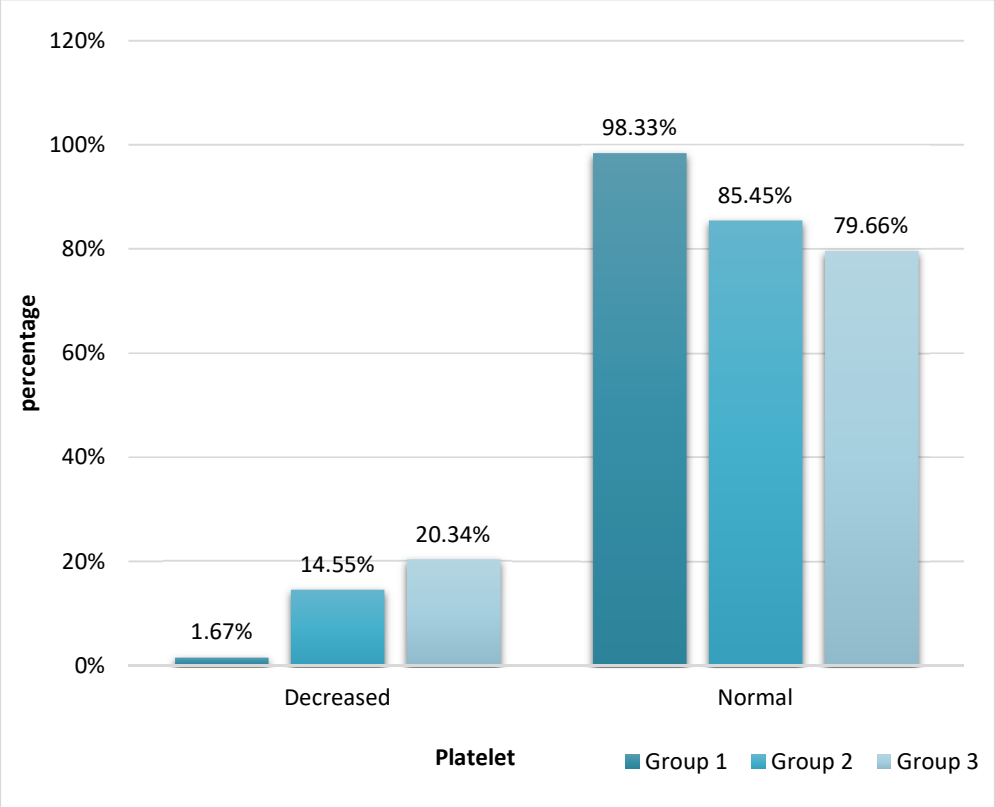


Figure 5-8 Distrubition of decreased versus normal platelet count in study groups

5.9 Comparison of incidence of change in PDW and plateletcrit in preeclampsia and eclampsia.

In group 3 that is preeclampsia with severe feature and eclampsia , study participants that were nonthrombocytopenic , 40.4% of them have increased PDW, whereas 21.2% have increased PDW in nonthrombocytopenic PE without severe feature. Similarly 31.91% of nonthrombocytopenic PE with severe feature and eclampsia and 34% of nonthrombocytopenic PE without severe feature have decreased plateletcrit.

Table 5-8 Comparision of incidence of change in PDW and plateletcrit in preeclampsia and eclampsia .

S.no	Groups	Parameter		P-value
		Increased PDW	normal PDW	
1	Nonthrombocytopenic PE with severe features and eclampsia (n=47)	19(40.4%)	28 (59.57%)	<0.03
2	Nonthrombocytopenic PE without severe feature (n=47)	10 (21.2%)	37 (78.7%)	
		Decreased Plateletcrit	Normal plateletcrit	
3	Nonthrombocytopenic PE with severe feature and eclampsia (n=47)	15 (31.91%)	32 (68%)	<0.04
4	Nonthrombocytopenic PE without severe feature (n=47)	16 (34%)	31 (65.9%)	

5.10 Diagnostic cutoff value of various parameter for preeclampsia without severe feature and preclampsia with severe feature and eclampsia.

Table 5-9 Diagnostic cutoff value of various parameter for preeclampsia without severe feature and preeclampsia with severe feature and eclampsia .

S.no	Groups	Parameter	SN%	SP%	PPV	NPV	AU-ROC	Cutoff	P-value
1	Precelampsia without severe features	Platelet	96.67%	21.82%	57.43%	85.71%	0.567	<1.68	0.299
2		PDW	93.33%	21.82%	56.57%	75%	0.556	>15.5	0.181
3		Plateletcrit	85%	32.73%	57.95%	66.67%	0.557	<0.20	0.375
4		MPV	41.67%	65.45%	56.82%	50.70%	0.508	>10.60	0.933
5		PC/MPV	86.76%	50.91%	65.82%	77.78%	0.676	<20.75	**0.004**
1	Preeclampsia with severe feature + eclampsia	Platelet	98.33%	22.03%	56.19%	92.86%	0.550	<1.6	0.263
2		PDW	90%	33.9%	58.06%	76.92%	0.602	**>15**	**0.0291**
3		Plateletcrit	93.33%	28.81%	57.14%	80.95%	0.572	<0.19	0.09
4		MPV	98.33%	20.34%	55.66%	92.31%	0.534	>11.8	0.359
5		PC/MPV	95%	40.68%	61.96%	88.89%	0.669	**<19.7**	**<0.001**

From the above table it is observed that area under the ROC curve ranges from 0.5083 to 0.6761 , with the PC/MPV ratio for Preeclampsia demonstrating the highest discriminatory ability (AU-ROC =0.6761). From the logistic regression , it is observed that PC/MPV is significantly predicting preeclampsia with non severe feature (p=0.0047) with the diagnostic cutoff value of <20.75.

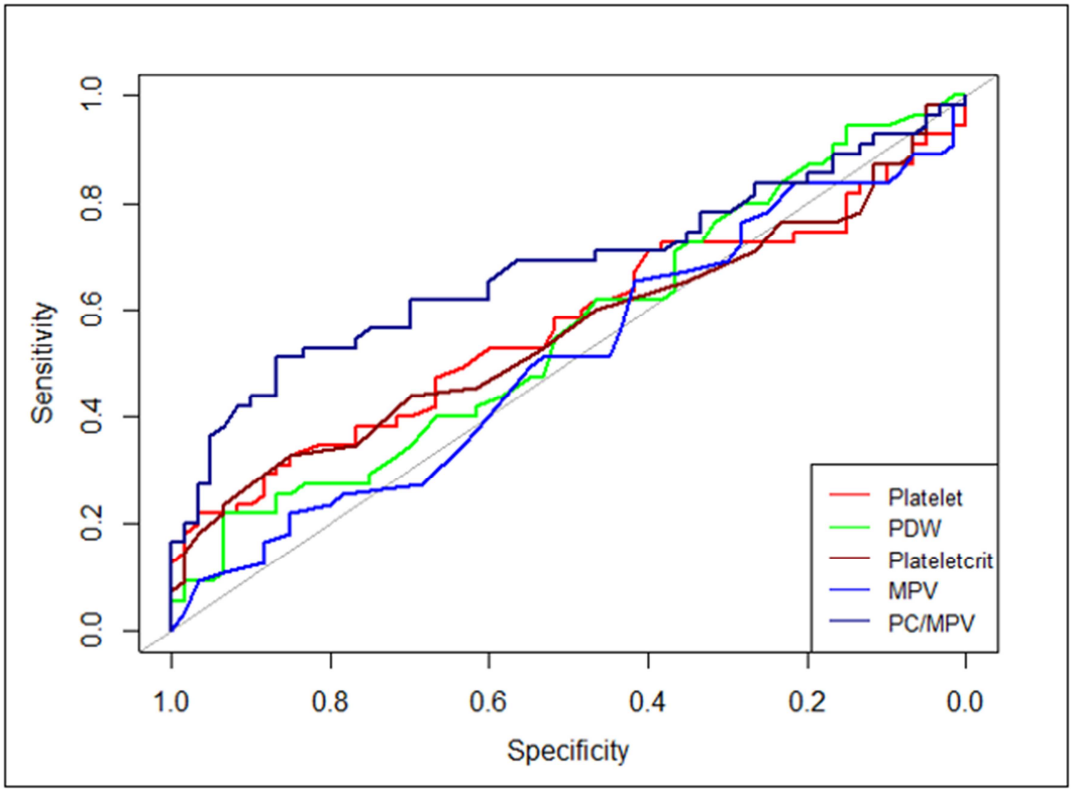


Figure 5-9 ROC curve of various parameter for predicting preeclampsia with non severe feature

Further it has been observed for preeclampsia with severe feature +eclampsia that PC/MPV ratio has the highest discriminatory ability (AU-ROC =0.6692). Statistically significant p- values are observed for PDW (p=0.0291) and PC/MPV ratio (p<0.001), indicating these parameter potential as useful diagnostic marker for preeclampsia with severe feature +eclampsia .

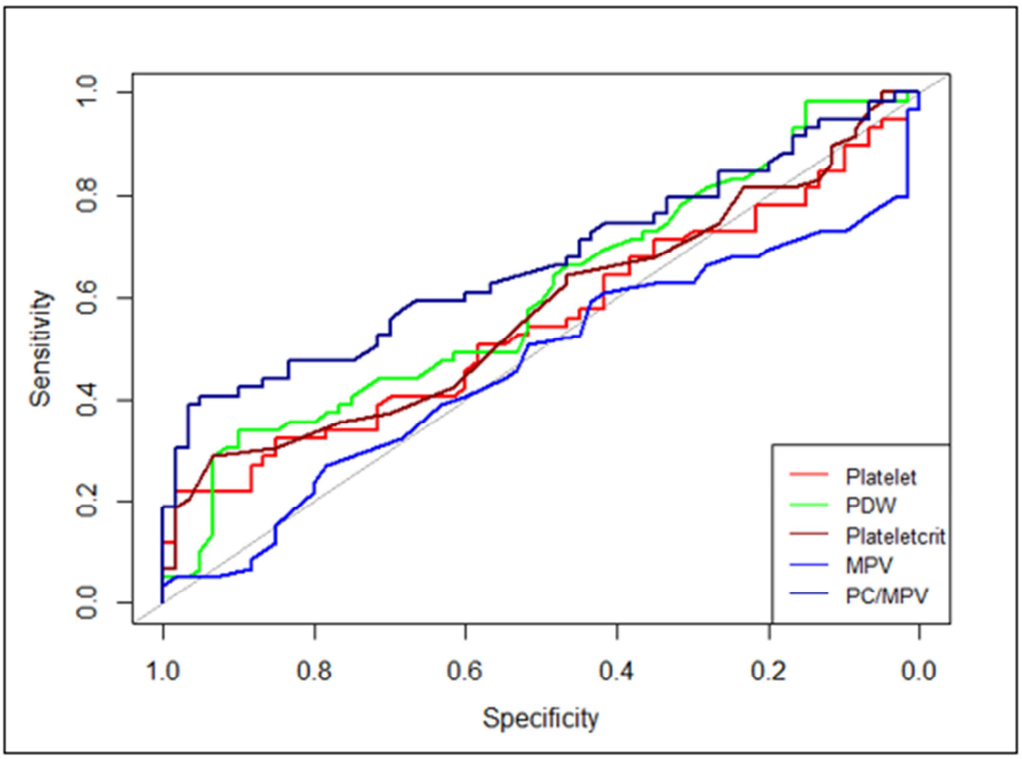


Figure 5-10 The ROC curve of various parameter for predicting Preeclampsia with severe feature + eclampsia

5.11 Comparison of Doppler study, Number of antihypertensive used and uses of MgSo4 in different groups.

Table 5-10 Comparison of Doppler study , Number of antihypertensive used and uses of MgSo4 in different groups .

S.no	Parameter	Variables	Group1	Group 2	Group 3
1.	Dopplers	Normal	58 (96.6%)	45(81.8%)	39(66.1%)
		Increased	2 (3.33%)	5(9.09%)	9(15.25%)
		AEDF*	0	4(7.27%)	5(8.47%)
		REDF*	0	1(1.81%)	2(3.38%)
2.	No of anti hypertensive used	None	60 (100%)	3 (5.45%)	1(1.69%)
		One	0	47(85.45%)	34(57.63%)
		Two	0	5 (9.09%)	24(40.68%)
3.	Use of MgSo4	No	60(100%)	51(92.73%)	9(15.25%)
		Yes	0	4(7.27%)	50(84.75%)

AEDF- Absent end diastolic flow

REDF – Reverse end diastolic flow

From the above table it is observed that abnormal doppler study were found more in group 2 and group 3 . Increased resistance were found in 3.33% of study participant in group 1 as compared to 9.09% in group 2 and 15.23% in group 3 respectively .

Use of antihypertensive drug in management of preeclmapsia increase as severity of disease increases . In group 1 which include normotensive women none of the participant used anti antihypertensive medication as compared to group 2 in which 9.09% and group 3 in which 40.68% used dual antihypertensive medication .

Similar the used of magnesium sulphate is more in group 3 that is 84.75% in contrast to group 2 that is 7.27 % respectively .

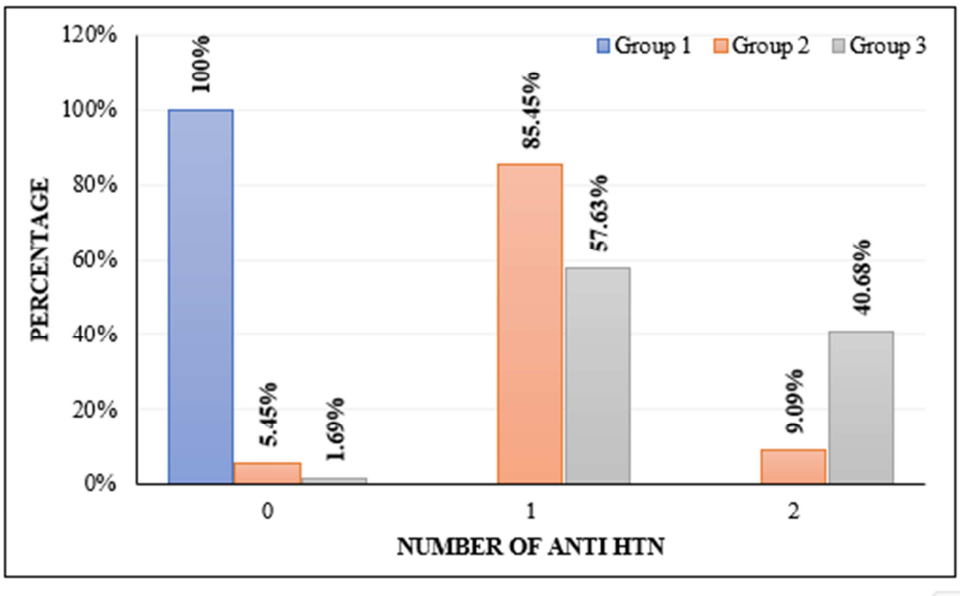


Figure 5-11- Distribution of number of Anti-hypertensive used over different group

5-12 Comparison of mode of delivery over different group

Table 5-12 shows the distribution of study participants on the basis of mode of delivery .It has been observed as the severity of disease increase more number of study participant underwent Caesarean section . In group 1 The rate of caesarean section is 19.5 % which is almost comparable to group 2 (20.11%) and in group 3 it is significantly more that is 27.4% .

Table 5-11 Comparison of mode of delivery over different group

S.no	Mode of delivery	Group 1	Group 2	Group 3
1.	LSCS	34 (19.5%)	35 (20.11%)	48 (27.5%)
2.	Vaginal delivery	26 (14.9%)	20 (11.4%)	11(6.3%)

5-13 Comparison of maternal outcome in study participant over different groups

From table 5-13 it is observed that more number of study participant were having adverse maternal outcome in group 3 which is preeclampsia with severe feature and eclampsia in comparison to group 2 and group 3 .HELLP syndrome were seen in 3.64% of study participant in group 2 in comparison to 6.78% in group 3.

Table 5-12 comparison of maternal outcome in study participant over different groups

S.no	Variables	Group 1	Group 2	Group 3
1.	CNS complication	0 %	0 %	2(3.39%)
2.	DIC	0%	2 (3.64%)	1(1.69%)
3.	PPH	1(1.6%)	1(1.82%)	2(3.39%)
4.	Abruption	1(1.6%)	3(5.45%)	3(5.08%)
5.	HELLP	0	2(3.64%)	4 (6.78%)
6.	Renal complication	0	0	0
7.	ICU admission	0	0	1(1.69%)

5-14 Comparison of neonatal outcome in study participant over different groups

Table 5-13 comparison of neonatal outcome over different groups

S.no	Variables		Group 1	Group 2	Group3	p-Value
1.	Neonatal outcome	Death	1 (1.67%)	5 (9.1%)	8 (13.56%)	0.0630^{MC}
		Live	59 (98.33%)	50 (90.9%)	51 (86.44%)	
2.	Term/Preterm	Preterm	33 (55%)	18 (32.7%)	40 (67.8%)	**< 0.001^{C}***
		Term	27 (45%)	37 (67.2%)	19 (32.2%)	
3.	Birth weight	Mean ± SD	2.32 ± 0.85	2.23 ± 0.65	1.76 ± 0.73	**< 0.001^{K}***
4.	NICU admission	No	50(83.3%)	47(85.45%)	32(54.24%)	0.0014^{C}*
		Yes	10(16.6%)	8(14.55%)	27(45.76%)	

From the above table it is observed that there were more preterm deliveries in group 3 that is 67.8% in group 2 Number of preterm delivery is 32.7%, as the severity of the disease increase, preterm delivery rate increases. The mean birth weight in group 1 is 2.32 kg, in group 2 is 2.23 kg in group 3 it is 1.76kg, which corresponds to the number of preterm delivery There were more number of NICU admission in Group 3 than in group 2 and group 1 . There were more perinatal mortality in group 3 as compared to group 2 and group 1 .

6 DISCUSSION

Preeclampsia which is defined as sudden onset hypertension with or without proteinuria and or other maternal organ dysfunction after 20 week of pregnancy, is associated with serious adverse maternal and neonatal outcomes .Several studies have been conducted using a variety of biochemical indicator as predictors and prognosticator for preeclmapsia in pregnant women . Decrease in platelet count may be due to gestation thrombocytopenia itself rather than Preeclampsia .Thus plalelet count although an important parameter in PE cannot be used as a definitive marker for the same . The utility of different platelets indices as predictors of preeclampsia has been studied previously; however, reports in this regard are controversial. The current study was conducted with the objective to evaluate platelets indices in women with preeclampsia and eclampsia.

Currently there are no specific test done for prediction of preeclampsia and eclampsia , there is no cost effective test that is a potential predictor for preeclampsia and eclampsia progression .

This study was a hospital based observational study conducted at KAHER'S Dr. Prabhakar Kore Hospital , Belagavi for a period of 12 months. Data on Demographic and clinical parameter was collected from 174 antenatal women , with a gestational age of >20 weeks and diagnoised with new onset of hypertension after obtaining written consent from the participant . Statistical tools were employed to understand asses the platelet count and platelet indices in association with preeclampsia and eclampsia .

Age distribution – The age of the participant ranged from 18 years to 39 years with mean maternal age of 25.86 with the standard deviation of 4.39 years . This indicates that preeclampsia is more common in younger women .51.15% of the total study participant were in age group of 21-25 out of which 31.54 % were having preeclampsia and eclampsia .This is in corelation with previously reported study by Saxena et al that 71% of the study population were in the age group 20-30 years.(81)

Parity – Primigravida has been found to be associated with preeclampsia . In the present study it has been observed that preeclampsia and eclampsia occurred more in primigravida (57.8%) as compared to Multigravida (42.2%). Out of 174 women 114 women pregnancy were complicated with Preeclampsia and eclampsia .Out of those 114 women 66 were primigravida 48 were multigravida .Similar study conducted by Kazt et al (82)and swamy et al (83) reported 50% and 61% of study population as primigravida .

Gestational age distribution – Gestational age of maximum proportion of study participant in Group 2 and group 3 of our study which include preeclampsia and eclampsia were < 37 weeks (57%) which indicate preterm deliveries are more prevalent in preeclamptic women .Similar studies were conducted by Ngwenya S et al Which report 50 % of the study population had gestational age <37 weeks .(84)

Majority of the preeclampsia and eclampsia patient in our study (54%) women had a average BMI . These results correlate with the finding of Qublan HS et al (85).

Both systolic and diastolic blood pressure were elevated in the women with preeclampsia and eclampsia (group 2 and 3) of our study with the mean systolic blood pressure of 141.75 mmHg with a standard deviation of 3.57 in group 2 (preeclampsia without severe feature) and a mean systolic blood pressure of 156.92 mmHg with a standard deviation of 16.35mmHg in group 3 (preeclampsia with sever feature + eclampsia) which is similar to the study conducted by Ratan et al .(86)

Level of proteinuria were elevated for most of the study participant in group 2 (preeclampsia without severe feature) and group 3 (preeclampsia with severe features +eclampsia) with most of the participant 37% had a proteinuria of 2+ in group 2 and 37% had proteinuria of 3+ in group 3 . Although the association between level of proteinuria and severity of preeclampsia has not been found in previous studies(87) , the likelihood of severity of preeclampsia increases increase when proteinuria is detected in the patient.

In this study 52% of the women reported to be PE with severe feature and eclampsia out of group 2 and group 3 in our study . The prevelance of PE with severe feature was found to be 26.3% in the study conducted by Zanzibar et al . Since the present study was conducted in referral hospital , patient were admitted during the latter stages of preeclampsia and not during initial stages , which contributes to the high number severe cases . When compared to prior studies on preeclampsia in the global south , which found prevalence rate ranging from 1.8 percent to 16.7 percent (88, 89), the current study prevelance rate is high . Also considering this to be the referral center , these result can be generalized for the whole region .

In this study it is found that as the severity of disease increases the platelet count decreases.

With mean **platelet count** of 2.42 ± 0.54 in group 1 , 2.28 ± 0.82 in group 2 and 2.27 ±0.81 . the platelet count shows decreasing in this study but most of the subjects were nonthrombocytopenic . 85% of the participant in group 2 (preeclampsia without severe feature) and 71% of participant in group 3 (preeclampsia with severe feature

+ eclampsia) were having normal platelet count which indicated that plaletet alone cannot be used as definite marker for the same . similar result were also found in the study conducted by Abha S , Ruchi V .(90).

The **MPV** show a increasing trend in our study with a mean of 10.28 ± 1in group 1 (normotensive) , 10.3 ± 0.9 in group 2 and 10.47 ± 1.18 but results were not statically significant (p- value – 0.5) . Similar studies conducted by Kurtoglu E et al and Kurt R et al found no significant association between preeclampsia and MPV . (91,92)

The level of **PDW** shows an increasing trend in our study with the mean of 11.82 ± 2.37 in group 1 , 12.48 ± 2.82 in group 2 , 12.91 ± 2.87 in group 3 in contrast to the study done by yang S et al .(93)

The level of Level of plateletcrit also shows a decreasing trend with mean of 0.25 ±0.06 , 0.24±0.08, 0.23±0.07 in group 1, group 2 and group 3 respectively but the result were statistically not significant (p value 0.3) in contrast to the study done by Karateke A et al . (94)

PC/MPV has come as a reliable marker for preeclampsia in this study . As the severity of the disease increase the value decreases. That ratio of 25.47 ±5.23 was found in group 1 , and in group 2 and group 3 it was found to be 21.8±1 , 21.06 ± 7.32 respectively which was found statistically significant with a p value of (< 0.001), similar results were found in the study conducted by Muneera A, Rafi S et al with platelet count less than 2.48 and PC/MPV ratio less than 31.2 for the diagnosis of preeclampsia (95) In contrast to our study in which the diagnostic cutoff for group 2 (preeclampsia without severe feature) was found to be < 20.75 with a senisivity of 86.67% and PPV of 65.82%. for group the diagnostic cutoff was <19.7 with sensitivity of 95% .

From our study it has been observed that 40.4% of non thrombocytopenic preeclampsia with severe feature, eclampsia and 21.2% of nonthrombocytopenic preeclampsia without severe features had increased PDW with p value of <0.03 .

Further 31.91%of nonthrombocytopenic preeclampsia with severe feature , eclampsia and 34% of nonthrombocytopenic preeclampsia without severe features had decreased plateletcrit with p-value of <0.04.

This was consistent with the study done by abha s, Ruchi V et al in which they found 57.9% of nonthrombocytopenic eclampsia had increased PDW and 33.3% of nonthrombocytopenic preeclampsia had increased PDW, also 63.3% of nonthrombocytopenic eclampsia and 25% of nonthrombocytopenic preclampsia had decreased plateletcrit[73].In eclampsia patients having normal platelet count , in a higher number of patient plateletcrit was decreased (63,3%), while PDW was increased (57.9%),

This further reinforces our understanding that relying solely on platelet count is insufficient for evaluating the severity of this condition.

In this study, maximum maternal complications were reported in preeclampsia with severe features/eclampsia, as compared to preeclampsia without severe features. Pregnancy complications such as preeclampsia with severe features, eclampsia, and HELLP syndrome are linked to a higher risk of maternal morbidity and mortality. Severe bleeding from abruption, pulmonary edema, cerebral hemorrhage, and liver rupture are among the complications that cause maternal mortality. Women who acquire Preeclampsia with severe features, and eclampsia syndrome before 32 weeks of pregnancy are more likely to have this problem. Women who have Preeclampsia with severe features/eclampsia have a higher risk of unfavorable maternal outcomes [96,97]. Similar outcomes have been observed in our study.

In this study it is observed that doppler abnormalities were found more in group 3 (preeclampsia with severe feature and eclampsia), and use of dual anti hypertensive medication were found to be more in group 3 with 40.68% of the study participant as compared to group 2 with 9.09% of the study participant .Similary the use of MgSo4 were more in group 3 [98]

In this study, the mean birth weight of neonates both to patients with preeclampsia with severe feature and eclampsia were lower than 2.5 kg and this than been observed in previous studies. Contrary to a previous study conducted in Canada where 61.2% of neonates born to women with preeclampsia were delivered before 37 weeks. In this study, 67.8% of pregnant women delivered before 37 weeks in group 3[99].

In our study, we noted that as the severity of the disease increases, the termination of pregnancy via cesarean section is more common, with a rate of 27.5% in group 3 compared to 19.5% in group 1. Conversely, the rate of vaginal delivery is lower, at 6.3% in group 3 compared to 14.9% in group 1 .

Pre-term newborns are more susceptible to the effects of low birth weight. This could explain why prior epidemiologic investigations on the relationship between preeclampsia or prenatal hypertension and birth weight came up with inconsistent results. The incidence of preterm deliveries among patients with pre-eclampsia was high (27.5% percent) in one study, where it was observed that babies born to pre-eclamptic women had a substantially lower mean birth weight (2.9 kg). The total effect on birth weight seen in the study could be influenced by the proportions of full-term and preterm deliveries among preeclampsia patients. The fact that most babies born patients with pre-eclampsia at term have normal foetal growth cannot be reconciled with the widely held idea that preeclampsia is caused by a reduction in uteroplacental perfusion . A growing body of evidence supports the idea that preeclampsia has significant pathophysiologic heterogeneity.

Studies have shown that premature births, NICU admissions and neonatal morbidity, and neonatal fatalities are all higher among neonates of pre-eclamptic women [32–34]. It was observed in this study that 51% of the neonates were born pre-term and the rate of NICU admissions was around 38%, which is higher that literature reported findings. The small cohort and stringent inclusion criteria can be a possible reason for this high incidence of both prematurity and NICU admissions. Also, since this study was conducted at a tertiary referral hospital, so most patients were admitted for complicated or preterm deliveries.

It is widely established that pregnancies complicated by severe preeclampsia or eclampsia, increase maternal and neonatal morbidity and mortality. In the literature, different prenatal morbidity and mortality rates are given .

7. LIMITATION OF THE STUDY

Following are the limitation of the study

- This study was conducted in a tertiary care centre therefore the finding may not adequately reflect the entire Belagavi region.
- The study did not take into account the participant’s socioeconomic status including income, living standard , and nutritional status , which could be an important factor for preeclampsia .
- The platelet count and indices used in the current study were measured at a single gestational age; however, values obtained at various gestational ages should also be taken into account to reinforce the platelet indices' predictive power for preeclampsia.

8) CONCLUSION

In obstetrics, determination of cost effective test that can predict development of maternal complication , could help in management of Preeclampsia , thus improving both maternal and foetal outcome . Platelet indices specifically ratio of PC/MPV could be used as a marker for severity of the condition. However large scale study wth large sample size is required to strengthen the role of platelet indices as a predictor of preeclampsia .Furthermore, use of additional test along with platelet indices could provide more accurate result. Identification of maternal and fetal outcomes in preeclampsia with this simple, cost effective test can help in early reduction of unfavorable events associated with preeclampsia and eclampsia.

The key conclusion of the study are as follows

1) Incidence of preeclampsia and eclampsia were higher in case of primigravida and women of younger age group.
2) There is a positive association of PC/MPV and severity of preeclampsia and eclampsia.
3) Higher incidence of maternal complication were likely to be in patient with PE with severe feature and eclampsia compared to patient with PE without severe feature.
4) Higher incidence of preterm birth, birth weight less than <2.5 kg and NICU admission were likely in case of women preeclampsia with severe features and eclampsia.

9) SUMMARY

The present study which was conducted to evaluate the association between the platelet count and platelet indices with the severity of preeclampsia and eclampsia as well as the risk of developing maternal complications and to asses maternal and perinatal outcome of preeclampsia and eclampsia .The study was conducted out at the department of Obstetrics and gynecology of KAHER's Dr Prabhakar kore hospital , Belagavi for a period of one year. Patient population consisted of antenatal women more than 20 week period of gestation who presented with preeclampsia and eclampsia and normotensive women , presented to labour room ward .A total of 174 women were selected for enrolment based on inclusion criteria .Data regarding sociodemographic and clinical characteristic were collected in the form of structured questionnaires and analysed statistically .

Key finding of this study have been summarized as follows –

1) The age of the participant ranged from 18 years to 39 years with mean maternal age of 25.86 with the SD of 4.39 years.
2) Average body mass index of the participant was found to be 24.18 with a SD of 1.74 and ranges from 20 to 29.5.
3) Maximum number of study participant in group 2 and group 3 were primigravida (57.8%) as compared to multigravida (42.2%).
4) Gestational age of maximum proportion of study participant in Group 2 and group 3 of our study which include preeclampsia and eclampsia were < 37 weeks (57%).
5) Both systolic and diastolic blood pressure were elevated for the study participant in group 2 and group 3 in comparison to group 1 (normotensive women) . With

mean arterial blood pressure of 91.3 ± 5.01 mmHg in group 1 and 107.7 ± 3.08 mmHg , 118.47 ± 11.56 mmHg in group 2 and group 3 respectively .

6) A significant level of proteinuria were found in most of the study participant in group 2 (preeclampsia without severe feature) and group 3 (preeclampsia with severe features and eclampsia) with most of the participant 37% had a proteinuria of 2+ in group 2 and 37% had proteinuria of 3+ in group 3 .
7) In the study we found a decreasing trend of platelet count and plateletcrit and an increasing trend of MPV and PDW but the results were statistically not significant.
8) This study showed a decline in PC/MPV values, which is shown to be statistically significant with a p value of (<0.001) in group 2, group 3, and group 1. The values in each group are 25.43, 21.8, and 21.06, respectively.
9) The diagnostic cutoff value of PC/MPV in Group 3 that is Preeclampsia with severe feature and eclampsia is found out to be <19.7 with a sensitivity of 95%.

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ANNEXURE -1

INFORMED CONSENT

Purpose of the study

I have been informed by _____, a post graduate in M.S. Obstetrics and Gynaecology under the guidance of _____, Professor & Unit Head, Department of Obstetrics and Gynaecology, J.N. Medical College, KLE University, Belagavi is conducting a study in KAHER's Dr. Prabhakar Kore Charitable Hospital, Belagavi.

Pre-eclampsia and eclampsia account for significant maternal morbidity and mortality including acute renal failure, visual impairment, pulmonary edema, cardiorespiratory arrest, and neurological manifestations including recurrent seizures and cerebrovascular accidents. Additionally, Preeclampsia and eclampsia are associated with increased perinatal mortality and morbidity accounting for 15% of preterm births and 10% stillbirths.

As the incidence of preeclampsia and eclampsia is high this study's aim is to find the association of platelet count and platelet indices in pregnancy with preeclampsia and eclampsia.

Study procedure:

Once I have signed the informed consent form, the personal details like name, age, place, address, my education, my health, reproductive history and other information will be noted down.

Potential Risks

There are no observable risks associated with the study.

Alternatives

If I decide not to participate in the study, my health care provider will provide the usual standard care during my delivery.

Privacy

To protect my privacy, all the collected information will be given a number rather than using my name. Any information collected during the study will remain confidential. My medical files will be reviewed only at the hospital (or study doctor's office) to check the information and verify the result without breaking my confidentiality. Only de-identified information on my pregnancy will be shared so as to learn the results of the study.

Authorisation to publish results

The information about me will be analysed together with other study participants.

Results of this study will be published and presented to scientific groups for scientific purposes, but I will never be individually identified in the presentation of the study results.

Questions: In case of any questions with regard to this study, you are free to contact: "Name of student/PI, mobile number, email ID" 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.

Person requesting consent, please check applicable boxes:

☐ Consent obtained (for adult respondent)

I have read the consent form or the consent form has been read to me. I understand the consent and the signature or sign below confirms that I agree to participate in this study (The participant will receive a copy of this form.)

Study identification number: ☐☐☐☐

_____ _____

Signature or thumbprint of participant Date

Name and signature of the Witness _____

Name and signature of the doctor _____

ANNEXURE -II

SCREENING AND RECRUITMENT FORM

Screening number: ☐☐☐

Enrollement 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 – ☐

Protenuria - ☐

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

"Platelet count and Platelet indices in pregnancy with Preeclampsia and Eclampsia" A Prospective Observational study .

Name	
Age	
Address	
Phone number	
Date of admission	
Date of discharge	
IP No.	
Registered/Unregistered	

COMPLAINTS AND HISTORY OF PRESENTING COMPLAINT:

Period of amenorrhea	
Duration of pain abdomen if present	
Duration of bleeding per vagina if present	
Perception of fetal movements (Yes/No)	
Imminent signs – 1. Headache 2. Blurring of vision 3. Epigastric discomfort 4. Vomiting 5. Seizures	☐ _____ ☐ _____ ☐ _____ ☐ _____ ☐ _____
Seizures (Yes/No) If Yes : 1. Number of episodes 2. Duration 3. Loss of consciousness 4. Lucid interval	☐

OBSTETRIC HISTORY:

Married Life / Consanguinity	
Obstetric Score	**G** ☐ **P** ☐ **L** ☐ **A** ☐
History of previous pregnancy	

MENSTRUAL HISTORY:

LMP	
EDD/CEDD	
Period of gestation	

PAST HISTORY :

Past medical and surgical history	

FAMILY HISTORY:

PERSONAL HISTORY :

GENERAL PHYSICAL EXAMINATION :

Height ☐		Weight ☐		BMI ☐	
Pulse rate ☐		Blood pressure ☐		MAP ☐	
Pallor		YES ☐		NO ☐	
Icterus		YES ☐		NO ☐	
Pedal edema		YES ☐		NO ☐	

SYSTEMIC EXAMINATION :

CVS :	
RS:	
Per Abdominal Examination	
Per Vaginal examination	

CLINICAL DIAGNOSIS :

INVESTIGATIONS :

Blood group/ Rh typing	
Hemoglobin (gm/dl)	
Peripheral smear	
Urine Routine	Protein - Sugar -
Serology : HIV/HbsAg/VDRL	
PLATELET INDICES	
1. Platelet count (10^3/uL)	
2. PDW (fL)	
3. Plateletcrit(%)	
4. MPV (fL)	

PIH profile : 1. Platelet count 2. Urea 3. Serum creatinine 4. Urine Albumin 5. LDH 6. Uric Acid 7. LFT : - Enzymes : SGOT/SGPT/ALP - Total Bilirubin - Serum Albumin	
DIC profile: 1. D-DIMER 2. Fibrinogen 3. PT/INR 4. aPTT 5. TT	

OBSTETRIC ULTRASOUND :

Date of Scan	
Doppler	

FUNDOSCOPY:

MANAGEMENT:

Antihypertensive used	
Mgso4 regimen	Yes ☐ No ☐
Other drugs if used	
Mode of delivery	Vaginal ☐ C-Section ☐
	Induction ☐ Augmentation ☐
Duration of Labour	
Intrapartum Complications	Yes ☐ No ☐
Blood Loss	
Indication for LSCS	
Intraop findings :	
EXAMINATION OF PLACENTA	
Weight of placenta	
Retroplacental clot	Yes ☐ No ☐

MATERNAL OUTCOME :

Peripartum Complications:	YES	NO
1) Convulsions	☐	☐
2) DIC	☐	☐
3) Abruption Placenta	☐	☐
4) Peripartum Cardiomyopathy	☐	☐
5) Respiratory Distress	☐	☐
6) Pulmonary edema	☐	☐
7) HELLP	☐	☐
8) Acute Kidney injury	☐	☐

9) PPH 10) Abruptio Placenta 11) Cerebro -Vascular Complication	
ICU admission	Yes ☐ No ☐
Duration of stay	
Condition at discharge	
Cause of death if so	
Intervention if any	

PERINATAL OUTCOME:

1) Condition at birth 2) Term / Preterm 3) Female /Male 4) Weight 5) APGAR 6) NICU admission - yes /no	Live ☐ IUD ☐ MSB ☐ FSB ☐
7) Indication for Nicu admission	Fetal Distress ☐
	MSL ☐ Low Birth weight ☐ Asphyxia ☐ Others ☐
Condition of baby at discharge	

ANNEXURE IV– MASTER CHART

S.No	IP Number	Age	Obs Score	POG	Diagnosis	Antenatal complication	BMI	BP	MAP	Protenuria	Platelet	PDW	Plateletcrit	MPV	PC/MPV	Doppler	No of anti HTN	Mgso4	Mode of delivery	Indication	Intrapartum complication	CNS complication	DIC, PPH	Abruption	Pulmonary edema	HELLP	Renal	ICU admission	Maternal Outcome	Neonatal Outcome	Term/Preterm	Birth weight	NICU admission	Indication	Fundoscopy
1	1162740	26	G2P1L1	32 week 3 day	Severe PE	IUD	25.3	160/110	126	1+	1.44	16.3	0.15	10.4	13.86	normal	1	Yes	PTVD		nil	nil	nil	nil	nil	nil	nil	no	stable	MSB still birth	preterm	1kg	no	no	normal
2	1162772	24	G2P1L1	32 week	Non severe pe	IUGR	26	150/100	116.6	3+	3.38	15.6	0.3	8.9	37.9	Increase resistane	1	Yes	pre term lscs	FGR, preeclampsia	nil	nil	nil	nil	nil	nil	nil	no	stable	Live	preterm	1.2 kg	yes	LBW	normal
3	1166656	20	Primigravida	35 week 1 day	Severe PE	nil	22.1	146/100	115.3	3+	4.34	15.6	0.339	7.8	30.5	IUD	1	no	PTVD		nil	nil	no,no	no	no	no	no	no	stable	IUFD,MSB	preterm	1.2kg	no		
4	1167062	21	Primigravida	40 week	Non severe PE	nil	28.7	140/90	106.6	1+	1.58	17.2	0.186	11.8	13.3	normal	1	no	TERM LSCS	Fetal distress	nil	nil	no,no	no	no	no	no	no	stable	live	Term	3.3kg	no		normal
5	1166457	23	primigravida	39 week 3 days	Eclampsia	nil	26.7	160/90	113.3	2+	2.69	16.1	0.23	8.6	31.27	normal	1	yes	TERM LSCS	Fetal distress	convulsion 1 episode	nil	no,no	no	no	no	no	no	stable	Live	Term	3.7kg	no		normal
6	1167067	30	primigravida	28 week 4 days	Severe PE	nil	25	160/90	113.3	2+	3.21	16.3	0.3	9.4	34.1	normal	2	yes	PTVD		nil	nil	no,no	no	no	no	no	no	stable	live, died 2 day	Preterm	1 kg	yes	Asphyxia	normal
7	1168636	25	G3P2L0	28 week 1 day	No severe PE	IUGR	26.4	134/84	100.3	2+	1.04	12	0.09	9.8	10.6	Incresed R	2	no	PRETERM LSCS	Prev Lscs with PE	nil	nil	n0,no	no	no	no	no	no	stable	Live	preterm	1 kg	yes	LBW	Normal
8	1169104	26	G2A1	28wek 2 day	Non severe PE	nil	23	138/90	106	1+	1.76	18	0.19	9.1	19.34	AEDF	1	no	PTVD		nil	nil	no,no	no	no	no	no	no	stable	abortion		750 gm	no		normal
9	1169125	27	G2A1	38 week 1 day	Non severe PE	nil	22.8	140/90	106.6	1+	3.15	16	0.27	8.8	35.7	normal	1	no	FTVD		nil	nil	no,no	no	no	no	no	no	stable	Live	Term	2.9kg	no		Normal
10	1170673	27	primigravida	33week 5 days	Nonsevere PE	nil	23.1	150/90	110	2+	1.87	16.2	0.18	9.4	19.8	normal	2	yes	PRETERM LSCS	Abruption placenta	nil	nil	no,no	yes	no	no	no	no	stable	live	Term	1.4kg	yes	LBW	normal
11	1170731	25	primigravida	36 week 6 days	severe PE	IUGR	23.4	160/90	113.3	2+	1.28	16.6	0.152	11.9	10.7	normal	1	yes	PRETERM LSCS	severe pe	nil	nil	no,no	no	no	yes	no	no	stable	live	preterm	2kg	yes	LBW	normal
12	1170999	26	primigravida	36 week3 day	Nonsevere PE	nil	23.6	140/90	106.6	2+	3.65	15.9	0.317	8.7	41.95	normal	1	no	PTVD		nil	nil	no,no	no	no	no	no	no	stable	live	Term	2.5kg	no		normal
13	1172026	22	G2P1L1	39week 1day	Eclampsia	nil	25	160/90	113.3	4+	3.01	16	0.26	9	33.44	normal	1	yes	TERM LSCS	Eclmapsia	Convulsion 3 episode	nil	no,no	no	no	no	no	no	stable	Live	Term	2.3kg	no		normal
14	1101861	24	G2P1L1	35 week 1 day	severe PE	nil	22.8	160/110	126.6	4+	2.43	11.8	0.26	10.5	23.14	increse resistance	1	yes	pre term lscs	severe pe	nil	nil	no,no	no	no	no	no	no	stable	live	preterm	1.7kg	yes	LBW	normal
15	1175611	26	Primigravida	36 week 3 days	Severe PE	IUGR	26.3	160/100	120	3+	2.37	16.5	0.247	11.5	20.6	Incresed R	1	no	pre term lscs	Failed induction	nil	nil	no,no	no	no	no	no	no	stable	Live	Term	2.1kg	No		normal
16	1176619	28	G3P1L1A1	37week 1 day	Eclampsia	nil	26.3	170/90	116.6	3+	3.65	15.9	0.37	9.7	37.6	normal	2	yes	TERM LSCS	Antepartum eclampsia	conulsion 2 episode	nil	no,no	no	no	no	no	no	stable	Live	Term	2.4kg	no		normal
17	1176623	21	Primigravida	40 week 5 days	Nonsevere PE	nil	25	140/90	106.6	2+	2.83	11.8	0.3	10.5	26.95	normal	0	no	TERM LSCS	MSL	nil	nil	no,no	no	no	no	no	no	stable	Live	Term	2.5kg	no		normal
18	1178516	22	primigravida	28 week 3 days	Eclampsia	NIL	22.2	160/110	126.6	1+	1.47	10.4	0.14	9.3	15.8	normal	1	yes	pre term lscs	Antepartum eclampsia	Convulsion 2 episode	nil	no,no	no	no	no	no	no	stable	Live	preterm	850gm	yes	Resp dis	normal
19	1179447	22	Primigravida	32 week 1 day	Eclampsia	IUD	24.5	140/94	109.3	1+	2.13	11.8	0.21	9.6	22.187	IUD	1	yes	PTVD		Conuvlsion 3 episode	PRESS	no,no	no	no	no	no	no	stable	IUD,MSB	preterm	1 kg			normal
20	1177451	29	Primigravida	35week 1day	Severe PE	PPROM	26.5	160/100	120	3+	2.25	10.2	0.22	9.8	22.9	normal	1	no	FTVD		nil	nil	no,no	no	no	no	no	no	stable	Live	preterm	1.6kg	yes	LBW	normal
21	1181077	30	G2P1L1	39week 5 days	Nonsevere PE	nil	25.5	140/90	106.1	1+	1.25	12.8	0.14	10.8	11.57	normal	1	no	TERM LSCS	previous lscs in labour	nil	nil	no,no	no	no	no	no	no	stbalr	live	Term	2.6 kg	no		normal
22	1182378	29	primigravida	28 week 1 day	non severe PE	abruption	25.3	144/90	108	1+	2.91	10.7	0.29	9.9	29.3	normal	1	no	PRETERM LSCS	Abruption placenta	nil	nil	no,no	no	no	no	no	no	stable	live	preterm	699 gm	yes	lbw	normal
23	1183141	22	G2P1L1	32week 1 day	Eclampsia	NIL	23.6	110/70	83.3	2+	2.55	9.2	0.24	9.4	27.12	normal	2	Yes	PRETERM LSCS	Eclampsia ,	convulsion 1 episode	nil	no,no	no	no	no	no	no	stable	Live	preterm	1.05kg	yes	LBW	normal
24	1184731	26	G2P1L1	39week 5day	Eclampsia	nil	23.2	160/100	120	2+	2.12	16.2	0.25	11.7	18.11	normal	2	yes	TERM LSCS	Eclampsia	convulsion 1 episode	Gliosis in brain	no,no	no	no	no	no	no	stable	Live	Term	2.7kg	no		normal
25	1185251	32	G3P2L2	38Week 6 day	Non severe PE	nil	23.4	140/90	106.6	2+	2.12	11.3	0.21	10.1	20.9	normal	2	no	FTVD		nil	nil	no,no	no	no	no	no	no	stable	Live	Term	3kg	no		normal
26	11186271	30	primigravida	37 week 3 days	non sever PE	nil	23.2	144/86	105.3	1+	2.29	10.3	0.49	9.3	24.6	normal	1	no	FTVD		nil	nil	no,no	no	no	no	no	no	stable	live	Term	2.3kg	no		normal
27	1187240	23	primigravida	38 week 6 days	non severe PE	nil	25.6	140/94	109.3	1+	2.31	11.5	0.23	11.5	20.08	normal	1	no	TERM LSCS	MSL	nil	nil	n,no	no	no	no	no	no	stable	live	Term	3kg	no		normal
28	1187067	29	G2A1	38 week 5 days	non severe PE	nil	25	144/84	104	2+	1.1	14.2	0.12	10.9	10.09	normal	0	no	TERM LSCS	Failed induction	nil	nil	no,no	no	no	no	no	no	stable	live	Term	2.5kg	no		normal
29	1188642	23	G2A1	36 week 3 days	Severe PE	nil	28.5	170/108	128.6	2+	2.7	10.3	0.26	9.6	28.12	AEDF	2	yes	PRETERM LSCS	Severe PE	nil	nil	no,no	no	no	no	no	no	stable	Live	Term	2.1kg	no		normal
30	1187909	33	G4P2L2A1	39 week 3 days	Non severe PE	Hypothyrodism	22.5	140/90	106.6	1+	1.92	10.8	0.19	9.9	19.3	normal	2	no	TERM LSCS	uncontrolled HTN	nil	nil	no,no	no	no	no	no	no	stable	Live	Term	2.5kg	no		normal
31	1166806	23	Primgravida	36week 4 day	Normotensive	nil	25.1	130/90	103.3	trace	2.57	16.3	0.25	9.8	26.22	IUD	0	no	PTVD		nil	nil	no,no	no	no	no	no	no	stable	IUFD, FSB	Term	3kg	no		
32	1176596	25	primigravida	37 week 1 day	Nonsevere PE	late IUGR	25.3	140/90	106.1	1+	2.51	10.6	0.25	8.8	28.5	normal	1	no	FTVD		nil	nil	no, yes	no	no	no	no	no	stable	Live	Term	2.2 kg	no		normal
33	1176649	31	Primigravida	39week 4 day	Normotensive	nil	23.1	112/74	86.6	nil	2.36	13	0.27	11.3	20.8	normal	0	no	FTND		nil	nil	no,no	no	no	no	no	no	stable	Live	Term	2.9kg	no		
34	1179850	24	Primigravida	28week 4 day	Severe PE	Lowlying placenta, FGR	25.3	170/110	130	2+	1.1	16.1	0.14	12.6	8.73	AEDF	2	yes	PRETERM LSCS	severe PE and AEDF	headache , vomiting	nil	no,no	no	no	Yes	no	no	stables	Live	preterm	960gm	Yes	LBW	normal
35	1171859	22	primigravida	38week 3 days	Severe PE	Early IUGR,	25.3	160/100	120	4+	3.19	13	0.32	11.2	28.4	normal	1	yes	TERM LSCS	severe PE pathological trace	nil	nil	no,no	no	no	no	no	no	stable	Live	Term	1.2kg	yes	LBW	normal
36	1172913	25	Primigravida	40week 4 day	Non severe PE	nil	24.2	140/96	110.6	2+	1.67	17.4	0.18	10	16.7	normal	1	no	TERM LSCS	uncontrolled HTN	nil	nil	no,no	no	no	no	no	no	stable	Live	Term	2.9kg	no		normal

S.No	IP Number	Age	Obs Score	POG	Diagnosis	Antenatal complication	BMI	BP	MAP	Protenuria	Platelet	PDW	Plateletcrit	MPV	PC/MPV	Doppler	No of anti HTN	Mgso4	Mode of delivery	Indication	Intrapartum complication	CNS complication	DIC, PPH	Abruption	Pulmonary edema	HELLP	Renal	ICU admission	Maternal Outcome	Neonatal Outcome	Term/Preterm	Birth weight	NICU admission	Indication	Fundoscopy
37	1183050	29	Primigravida	28week 5days	Non severe PE	Early IUGR	25.3	150/90	110	1+	2.71	10.3	0.26	9.7	27.9	AEDF	2	no	PTVD		nil	nil	no,no	no	no	no	no	no	stable	IUD,FSB	preterm	490gm	no		normal
38	1203509	32	G2P1L1	37 week 5 day	Severe PE	nil	28	200/120	146.6	2+	2.25	11	0.23	10.2	22	normal	1	yes	TERM LSCS	Imminent eclampsia	nil	nil	no,no	no	no	yes	no	no	stable	live	preterm	2.1kg	no		normal
39	1203396	32	G3P1L1A1	36 week 3 day	Severe PE	Hypothyrodism	23.9	200/120	146.6	1+	3	13.2	0.34	11.3	24.5	normal	1	YES	PRETERM LSCS	Severe PE	convulsion 1 episode	nil	no, yes	yes	no	no	no	yes	stable	Live	preterm	1.7kg	yes	LBW	normal
40	1169104	26	G2A1	28wek 2 day	Non severe PE	nil	28	138/90	106	1+	1.76	18	0.19	9.1	19.34	AEDF	1	no	PRETERM LSCS		nil	nil	no,no	no	no	no	no	no	stable	abortion		750 gm	no		normal
41	1203678	19	Primigravida	35 week 6 day	Severe PE	Hypothyrodism	27.3	160/110	126.6	2+	3.7	9.6	0.36	9.7	30.14	normal	2	yes	PRETERM LSCS	severe PE with oligo	nil	nil	no, no	no	no	no	no	no	stable	Live	preterm	1.6 kg	yes	LBW	normal
42	1203613	24	Primigravida	37 week	Severe PE	Fetal Macrosomia	27	144/110	144	3+	2.2	15.5	0.27	12.1	18.18	normal	1	no	TERM LSCS	Severe PE	nil	nil	no, no	no	no	no	no	no	stable	Live	Term	2.9kg	no		normal
43	1204164	23	Primigravida	34 week 6 days	Non severe	IUD	29.5	150/100	116.6	1+	0.57	12.8	0.06	10	5.7	IUD	1	no	PTVD	PE	nil	nil	yes, no	yes	no	no	no	no	stable	IUD	preterm , FSB	2 KG	no		Grade 2 retinopathy
44	1204261	36	primigravida	33 week	Severe PE	Early IUGR, Oligo	26.1	160/110	126.6	4+	2.14	11.8	0.22	10.2	20.9	normal	2	yes	PRETERM LSCS	severe PE	nil	nil	no,no	no	no	no	no	no	stable	Live	Term	1kg	yes	LBW	Grade 2 retinopathy
45	1204753	23	primigravida	36 week 6 day	Eclampsia	nil	24.2	220/120	153.3	2+	2.28	15.8	0.27	11.7	19.48	normal	1	yes	PRETERM LSCS	Eclampsia	convulsion 1 episode	nil	no,no	no	no	no	no	no	stable	live	preterm	1.2kg	yes	lbw	normal
46	1205417	20	primigravida	28 week	Eclampsia	IUGR	24.6	150/100	116.6	3+	2.23	12.7	0.3	9.9	22.5	REDF	1	YES	PRETERM LSCS	Eclampsia , Early onset IUGR	Convulsion 2 episode	nil	no, no	no	no	no	no	no	stable	Live	preterm	550 gm	yes	LBW	normal
47	1205708	21	Primigravida	34 week 5 days	severe PE	DCDA twin	24.1	150/90	110	Trace	0.88	8.2	0.08	8.7	10.11	normal	2	yes	PRETERM LSCS	Eclampsia	Convulsion 2 episode	nil	no,no	no	no	no	no	no	stable	live	preterm	2.8kg	no		
48	1205636	28	G3P2L2	26 week 5 days	Severe PE	nil	23.4	170/100	123.3	3+	0.85	11.7	0.09	10.6	8.01	AEDF	1	no	PTVD			nil	no,no	no	no	no	no	no	stable	IUD, fsb	Preterm	820gm	no		Normal
49	1206109	23	Primigravida	38 Week 6 days	Eclampsia	nil	25.4	170/100	123.3	2+	1.59	13.6	0.16	10.4	15.28	Normal	1	yes	TERM LSCS	Antepartum eclampsia	Convulsion 2 episode	nil	no,no	no	no	no	no	no	stable	live	Term	3.2kg	no		Normal
50	10000359	29	primigravida	40 week 4 day	normotensive	nil	25	126/70	86.6	nil	3.07	12	0.22	11	27.9	normal	1	no	TERM LSCS	prev Lscs IN Labour	none	nil	no,no	no	nio	no	no	no	stable	live	term	2.7kg	no		Normal
51	10000368	34	G3P2L2	32 week 5 days	Severe PE	nil	25	180/100	126.6	3+	2.22	13	0.24	11	20.18	normal	1	yes	PRETERM LSCS	prv 2 lscs	none	nil	no,no	no	no	no	no	no	stable	live	preterm	1.5kg	no		normal
52	10001282	24	G2P1L1	41 week	Normotensive	nil	25.3	122/80	94	nil	2.56	11.3	0.25	9.7	26.39	normal	0	no	PRETERM LSCS	Macrosomia	none	nil	no,no	no	no	no	no	no	stable	live	Term	3.4kg	no		normal
53	10002355	33	G3P1L1A1	38 week 4 days	non severe PE	nil	25	140/90	106.6	1+	1.82	12.1	0.19	10.5	17.3	normal	1	no	TERM LSCS	prev LSCS scar tenderness	none	nil	no,no	no	no	no	no	no	stable	live	term	2.6 kg	no		normal
54	10001467	23	G2P1L1	38week 4 day	Severe PE	nil	26	160/110	126.6	2+	1.75	18.4	0.23	13.2	13.25	AEDF	1	no	TERM LSCS	prev LSCS	none	nil	no,no	no	no	no	no	no	stable	live	Term	1.8 kg	yes	LBW	normal
55	10002535	25	G5P1L1A3	34 week 5 days	Nonsevere PE	IUGR	26.7	150/100	116.6	1+	4.13	9.2	0.37	9	19.8	normal	1	no	PRETERM LSCS	non reassuring NST	none	nil	no,no	no	no	no	no	no	stable	Live	Term	1.6kg	yes	LBW	normal
56	10002798	28	G3P1L1A1	37 week 5 day	normotensive	nil	25	116/74	88	nil	2.26	14.1	0.26	11.4	35	normal	0	no	FTVD			nil	no,no	no	no	no	no	no	stable	live	Term	2.8 kg	yes	LBW	normal
57	10003009	23	G2A1	35week 1day	SeverePE	nil	26.4	140/90	106.6	2+	1.87	11.5	0.19	10.4	17.9	normal	1	yes	PRETERM LSCS	severe PE	none	nil	no,no	no	no	no	no	no	stable	live	preterm	2.7kg	no		normal
58	10003173	34	G4P2L2A1	39 week 2 day	Non severe PE	nil	26	150/100	116.6	nil	2.16	8.8	0.21	9.6	22.5	normal	1	no	FTVD		None	nil	no,no	no	no	no	no	no	stable	live	term	2.8kg	no		normal
59	10006182	25	G2P1L1	39 week 3 days	Nonsevere PE	nil	25.3	140/94	109.3	nil	2.2	12.2	0.23	10.7	20.5	norml	0	no	FTVD		NONE	nil	no,no	no	no	no	no	no	stable	live	Term	3.2kg	no		normal
60	10007283	24	primigravida	40 week	non severe PE	IUGR	21.1	140/86	104	trace	2.15	9.2	0.2	9.2	23.3	normal	1	no	TERM LSCS	fetal distress	none	nil	no,no	no	no	no	no	no	stable	live	Term	2.5kg	no		normal
61	10606560	26	G3P2L2	28 weeks 5 days	Non severe PE	Anemia	27	140/90	106.6	nil	2.18	10.2	0.24	11.1	19.6	AEDF	1	no	PRETERM LSCS	Prev2 lscs with AEDF	none	nil	no,no	no	no	no	no	no	stable	live	preterm	600gms	yes	LBW	normal
62	10007834	24	G2P1L1	28week 3 day	Normotensive	Oilgihydramnios	22.1	114/74	87.3	nil	3.29	11.2	0.33	10	32.9	normal	0	no	PTVD	oligohydramnios	none	nil	no,no	no	no	no	no	no	stable	live	preterm	820gms	yes	LBW	normal
63	10007891	26	Primigravida	36 week 2 day	Severe PE	IUGR	23.2	160/110	126.6	trace	1.4	19.9	0.17	12.3	11.3	normal	1	yes	PRETERM LSCS	imminent sign	none	nil	no,no	no	no	no	no	no	stable	live	preterm	1.7kg	yes	LBW	noraml
64	10007921	20	primgravida	32 week 3 days	Eclampsia	IUGR late onset	21.6	160/90	113.3	3+	2.58	11.4	0.25	9.8	26.3	normal	2	yes	PRETERM LSCS	Eclampsia	Convulsion 2 episode	nil	no,no	no	no	no	no	no	stable	live	preterm	1.3 kg	Yes	LBW	normal
65	10007921	25	Primigravida	33 week 6 days	Normotensive	preterm labour , PV leak	23.4	110/70	83.3	1+	2.6	8.6	0.23	8.9	29.21	normal	0	no	PTVD		none	nil	no,no	no	no	no	no	no	stable	live	preterm	1.68kg	Yes	LBW	normal
66	10008128	28	Primigravida	30 week 5 day	Severe PE	IUD	27.4	160/110	126.6	3+	0.77	11.5	0.09	12.1	6.3	IUD	1	no	PRETERM LSCS	Severe PE , Abruption	Abruption	nil	no,Yes	yes	no	no	no	no	stable	IUD,MSB	preterm	1 kg	no		normal
67	10006724	24	primigravida	35 week 5 day	Severe PE	IUGR late onset	26.3	164/90	114.6	2+	1.76	11.1	0.18	10.4	16.92	increse resistance	2	yes	PTVD		None	nil	no,no	no	no	no	no	no	stable	live	preterm	2.3kg	no		normal
68	10008732	23	G3P1L1A1	34 week	Severe PE	oligihydramnios	25.3	164/90	114.6	2+	2.47	9.4	0.23	9.3	29.4	normal	2	yes	PRETERM LSCS	SeverePE , oligo	none	nil	yes, no	no	no	yes	no	no	stable	live	preterm	2.1kg	no		normal
69	10010493	22	G2P1L1	33 week	normotensive		25.6	122/78	91.3	negative	2.44	14.9	0.28	11.4	21.4	normal	0	0	PTVD		none	nil	no,yes	no	no	no	no	no	stable	live	preterm	2kg	no		normal
70	10010628	25	primigravida	39week 2 day	Severe PE	nil	25.3	150/110	123.3	negative	2.21	9.2	0.2	9.2	24	increased resistance	2	yes	TERM LSCS	imminent sign	none	nil	no,no	no	no	no	no	no	stable	live	term	1.8kg	yes	LBW	normal
71	10010089	21	G2P1L1	32 week 2 day	Severe PE	nil	25.5	152/112	125.3	1+	2.61	10.7	0.26	10.1	25.8	normal	1	yes	PRETERM LSCS	severe PE	none	nil	no,no	no	no	no	no	no	stable	live	preterm	1.7 kg	yes	LBW	normal
72	10011612	24	G2P1L1	38 week	non severe PE	nil	26.1	140/94	109	1+	4.25	8.5	0.37	8.7	35	normmal	1	no	TERM LSCS	previous lscs with PE	none	nil	no,no	no	no	no	no	no	stable	live	Term	2.9kg	no		normal
73	10011542	26	G3P2L2	40 week	normotensive	nil	24.9	120/76	90.6	nil	2.67	10.3	0.26	9.7	27.5	normal	0	no	FTVD		none	nil	no,no	no	no	no	no	no	stable	live	Term	3 kg	no		normal
74	10011662	26	G3P2L2	40 week	normotensive	nil	24.2	118/82	94	nil	2.95	11.1	0.29	10	29.5	normal	0	no	FTVD		none	nil	no,no	no	no	no	no	no	stable	live	Term	2.2kg	no		normal
75	10012341	18	primigravida	28 week 3 days	normotensive	nil	22.4	122/74	90	nil	1.73	11.5	0.17	10.1	17.12	normal	0	no	PTVD		none	nil	no,no	no	no	no	no	no	stable	live	preterm	1.5kg	Yes	LBW	normal
76	10012350	24	primigravida	35 week 1 day	Normotensive	nil	21.9	116/78	90.6	nil	2.6	8.6	0.23	8.9	29.21	normal	0	no	PTVD		none	nil	no,no	no	no	no	no	no	stable	live	preterm	2kg	Yes	distress	normal

S.No	IP Number	Age	Obs Score	POG	Diagnosis	Antenatal complication	BMI	BP	MAP	Protenuria	Platelet	PDW	Plateletcrit	MPV	PC/MPV	Doppler	No of anti HTN	Mgso4	Mode of delivery	Indication	Intrapartum complication	CNS complication	DIC, PPH	Abruption	Pulmonary edema	HELLP	Renal	ICU admission	Maternal Outcome	Neonatal Outcome	Term/Preterm	Birth weight	NICU admission	Indication	Fundoscopy
77	10013080	23	Primigravida	36 week 6 day	Severe PE	Stage 4 FGR	22.4	162/100	120.6	1+	2.72	14.2	0.31	11.3	24	REDF	1	Yes	PRETERM LSCS	REDF	none	nil	no,no	no	no	no	no	no	no	stable	live	preterm	1.14kg	yes	Distress	normal
78	10013112	24	G4P1L1A1DI	32week 2 day	Severe PE	IUGR	23.2	142/86	104.6	2+	1.44	15.6	0.18	12.2	11.8	normal	1	yes	PRETERM LSCS	severePE ,imminent sign	none	nil	no,no	no	no	no	no	no	no	stable	live	preterm	1.6kg	yes	LBW	normal
79	10013382	38	G2P1L1	30 Week 3 day	Severe PE	IUGR	22.7	160/112	128	1+	4.14	9.2	0.18	9.1	25	Increase resistane	1	yes	PRETERM LSCS	previous lscs with AEDF	none	nil	no,no	no	no	no	no	no	no	stable	live	preterm	900gms	yes	LBW	normal
80	10013398	28	Primigravida	39 week	Eclampsia	IUGR	23.2	140/90	106.6	2+	1.38	18.6	0.16	11.6	11.89	normal	2	yes	TERM LSCS	eclampsia	none	nil	no,no	no	no	no	no	no	no	stable	live	Term	2.7kg	no		normal
81	10013448	21	primigravida	34 week 2 day	Eclampsia	nil	22.7	150/90	110	3+	2.13	11.6	0.22	10.2	20.88	increase resistance	2	yes	LSCS	eclampsia	none	nil	no,no	no	no	no	no	no	no	stable	live	preterm	2.1kg	yes	Distress	normal
82	10013650	30	G2P1L1	37week 6 day	Severe PE	IUGR	24	160/94	116	1+	2.58	12.2	0.2	10.3	25.04	IUD	1	yes	LSCS	Severe PE withIUD	none	nil	no,no	no	no	no	no	no	no	stable	IUD,MSB	Term	1.2kg	no		normal
83	10013908	21	Primigravida	32 week 2 day	normotensive	nil	23.8	126/80	95.3	nil	2.1	12.6	0.22	10.6	19.8	normal	0	no	LSCS	PROM , Pathological trace	none	nil	no,no	no	no	no	no	no	no	stable	live	preterm	1.6kg	yes	Distress	normal
84	10013608	30	primigravida	36 week 4 day	normotensive	IUGR	23	128/78	94.3	nil	1.6	15	0.19	11.7	13.6	normal	0	no	PTVD		none	nil	no,no	no	no	no	no	no	no	stable	live	preterm	2.1kg	no		normal
85	10011475	34	G3P2L1D1	29 week 3 day	Severe PE	IUGR	25.2	140/92	108	2+	3.11	12.6	0.34	10.8	28.7	AEDF	1	yes	LSCS	AEDF	none	nil	no,no	no	no	no	no	no	no	stable	live	preterm	920gm	yes	VLBW	normal
86	10013932	25	primigravida	36week 6 day	Normotensive	nil	24.2	122/86	98	1+	3.08	9.9	0.28	9	32	normal	1	yes	PTVD		none	nil	no,no	no	no	no	no	no	no	stable	live	preterm	2.5kg	no		normal
87	10011312	29	primigravida	36 week 6 day	non severe PE	nil	26.8	140/90	106.6	2+	3.39	10.5	0.35	10.4	28	normal	1	yes	LSCS	Non progress of labour	none	nil	no,no	no	no	no	no	no	no	stable	live	preterm	2.6kg	no		normal
88	10014345	31	G3P1L1A1	34 week 1 day	severe PE	IUGR	26.9	162/90	114	2+	2.55	9.9	0.24	9.4	27.12	incresed resistance	1	yes	LSCS	imminent sign	none	nil	no,no	no	no	no	no	no	no	stable	live	Preterm	1.5 kg	yes	LBW	normal
89	10014410	28	Primigravida	40 week 5 days	non severe PE	IUD	25	140/90	106.6	1+	2.05	12.8	0.21	10.4	19.7	increased resistance	1	no	FTVD		none	nil	no,no	no	no	no	no	no	no	stabe	IUD,MSB	Term	3.1kg	no		normal
90	10015002	28	G2P1D1	36week 5 day	Severe PE	IUGR	26.7	142/86	104.3	3+	2.31	9.8	0.23	9.8	23.57	increased resistance	1	yes	PRETERM LSCS	prevs lscs imminent sign	headache , vomiting	nil	no,no	no	no	no	no	no	no	stable	live	preterm	2 kg	no		normal
91	10015340	21	primigravida	36week 5 day	Eclampsia	nil	25.5	140/94	109.3	1+	1	14	0.14	12	8.3	normal	2	yes	PRETERM LSCS	Imminent eclampsia	2 convulsion	nil	no,no	no	no	no	no	no	no	stable	live	preterm	2.3kg	no		normal
92	10015531	23	G4P1L1A2	33week 4 day	normotensive	nil	24.9	128/82	97.3	nil	2.15	14.4	0.24	11.3	19.02	normal	0	no	PRETERM LSCS	previous lscs in labour	none	nil	no,no	no	no	no	no	no	no	stable	live	preterm	1.9kg	no		normal
93	10015689	21	G4P2L1A1D1	35 week 2 day	normotensive	nil	23.6	126/84	98	nil	3.67	10.4	0.36	9.8	37.44	normal	0	no	PRETERM LSCS	noreassuring NST	none	nil	no,no	no	no	no	no	no	no	stable	live	preterm	2.06kg	no		normal
94	10015893	38	primigravida	29 week 2 day	normotensive	Hypothyrodism	25.3	118/78	91.3	nil	2.62	8.4	0.23	8.8	29.77	normal	0	no	PRETERM LSCS	anhydramnios	none	nil	no,no	no	no	no	no	no	no	stable	live	preterm	1.1kg	yes	VLBW	normal
95	10015963	32	G3P1L1A1	34week 4 day	Normotensive	nil	24.7	128/82	97.3	nil	2.74	8.8	0.25	9	30.44	normal	0	no	PRETERM LSCS	Anamnios,scarten der	none	nil	no,no	no	no	no	no	no	no	stable	live	preterm	1.65kg	no		normal
96	10015232	21	primigravida	36week 5 day	normotensive	IUGR	22.2	114/70	84.6	nil	1.8	11.8	0.19	10.6	26	normal	0	no	PRETERM LSCS	Failed induction	none	nil	no,no	no	no	no	no	no	no	stable	live	preterm	2.1kg	no		normal
97	10016999	28	G2P1L1	40week 2 day	Severe PE	Nil	22.7	140/92	108	2+	3.33	15.7	0.2	12	22	normal	1	no	FTVD		none	nil	no,no	no	no	no	no	no	no	stable	live	Term	3.3kg	no		normal
98	10017492	26	G3P2L2	36 week 2 day	Severe PE	nil	21.6	146/94	107.3	2+	2.47	11.3	0.24	9.8	25.2	normal	1	yes	PRETERM LSCS	Severe PE ,imminent sign	none	nil	no,no	no	no	no	no	no	no	stable	live	preterm	2.6kg	no		normal
99	10013855	23	G2P1L1	28 week 5 day	normotensive	nil	23.5	126/82	96.6	nil	2.63	11.6	0.28	10.5	29	normal	0	no	PRETERM LSCS	prevs lscs AEDF			no,no	no	no	no	no	no	no	stable	live	preterm	980gms	yes	lbw	normal
100	10017745	22	G3P1L1A1	37 week	eclampsia	IUGR	23.7	146/100	115.3	2+	2.86	10.9	0.29	10	22.6	normal	2	yes	PRETERM LSCS	eeclampsia	2 episode of convulsion	nil	no,no	no	no	no	no	no	no	stable	live	Term	2kg	no		normal
101	10017748	21	primigravida	36 week 5 days	normotensive	nil	24	128/74	92	nil	2.54	10.5	0.25	9.9	28	normal	0	no	PTVD		none	nil	no,no	no	no	no	no	no	no	stable	live	preterm	2.7kg	no		normal
102	10018090	29	G2P1L1	31 week	Severe PE	nil	21.6	160/90	113.3	2+	0.58	16.5	0.07	12.3	10	normal	2	yes	PRETERM LSCS	abruption placenta	none	nil	no,no	yes	no	no	no	no	no	stable	IUD, FSB	preterm	870 gm	no		normal
103	10018045	21	G2P1L1	31 week 4 days	normotensive	nil, placenta previa	22	124/80	94.6	nil	1.37	7.9	0.13	9.2	24.8	normal	0	no	PRETERM LSCS	Antepartum haeorrhage	none	nil	no,yes	no	no	no	no	no	no	stable	Live	preterm	1.3 kg	no		
104	10018573	25	primigravida	31 week 4 days	non severe PE	IUGR	25.5	140/90	106.6	2+	0.9	16.2	0.11	11.6	20.7	increased resistance	1	no	PRETERM LSCS	HELLP	none		no	no	no	yes	no	no	stable	live	preterm	1.2kg	yes	LBW	normal
105	10019132	35	G3P1L1A1	32 week 6 days	normotensive	nil DCDA twins	23.4	122/80	94	nil	2.4	11	0.4	10.3	23.3	normal	0	no	PRETERM LSCS	prev lscs DCDA twins	none	nil	no,no	no	no	no	no	no	no	stable	live	preterm	1.8/2.1 kg	yes	LBW	normal
106	10019084	28	G2P1L1	37week 4 days	normotensive	nil	25.3	118/84	95.3	nil	3.91	12.4	0.4	10.2	38.3	normal	0	no	TERM LSCS	prev lscs in labour	none	nil	no,no	no	no	no	no	no	no	stable	live	Term	3kg	no		normal
107	10018552	23	G2P1L1	35 week 4day	normotensive	IUGR	25.3	122/80	94	nil	2.66	10.2	0.2	10.3	25.82	normal	0	no	PRETERM LSCS	prev lscs scar tender	none	nil	no,no	no	no	no	no	no	no	stable	live	preterm	1.8kg	yes	lbw	normal
108	10019108	25	G4P1L1A1	37 week 5 day	normotensive	nil	25.3	118/76	90	1+	2.19	8.8	0.19	8.7	25.17	normal	0	no	TERM LSCS	breech	none	nil	no,no	no	no	no	no	no	no	stable	Live	Term	2.4kg	no		normal
109	10018972	18	primigravida	34 week 3 days	normotensive	nil	26	126/80	95.3	nil	2.44	9.1	0.23	9.4	25.95	normal	0	no	PRETERM LSCS	breech in labour	none	nil	no,no	no	no	no	no	no	no	stable	live	preterm	1.7kg	yes	LBW	normal
110	10019651	25	primigravida	31 week	normotensive	nil DCDA twin	27	128/84	98.6	nil	3.64	14	0.38	11.1	32.7	normal	0	no	PRETERM LSCS	DCDA twin in labour	none	nil	no,no	no	no	no	no	no	no	stable	live	preterm	1.5/1.2kg	yes	LBW	normal
111	10019428	28	G2P1L1	34week 6 day	severe PE	IUGR, hypothyrodism	24.9	160/94	116	1+	1.77	10.3	0.18	10	17.7	normal	2	yes	PTVD		none	nil	no,no	no	no	no	no	no	no	stable	live	preterm	1.7kg	no		normal
112	10019340	26	primigravida	33 week 2 day	normotensive	nil	23.4	124/78	93.3	nil	3.3	9.9	0.33	10	33	normal	0	no	PRETERM LSCS	ProlongedPROM chroio	none	nil	no,no	no	no	no	no	no	no	stable	live	preterm	1.8kg	yes	LBW	normal
113	10020556	21	primigravida	32 week 5 days	Eclampsia	nil	24.9	144/96	112	1+	1.83	10.1	0.18	9.7	16	normal	0	no	PTVD	Eclampsia	none	nik	no,no	no	no	no	no	no	no	stabe	live	preterm	1.8kg	yes	LBW	normal
114	10020708	37	G3P1L1A1	38 week 4 day	severe PE	nil	24.2	140/96	110.6	2+	2.81	9.9	0.27	9.5	29.5	normal	2	yes	TERM LSCS	imminent sign	none	nil	no,no	no	no	no	no	no	no	stable	live	Term	1.85kg	no		normal
115	10020441	30	G5P3L3A1	25week	normotensive	nil	22.8	130/80	96.6	nil	2.79	9	0.25	9	31	normal	0	no	PTVD		none	nil	no,no	no	no	no	no	no	no	stable	Live	preterm	860 gms	yes	VLBW	normal

S.No	IP Number	Age	Obs Score	POG	Diagnosis	Antenatal complication	BMI	BP	MAP	Protenuria	Platelet	PDW	Plateletcrit	MPV	PC/MPV	Doppler	No of anti HTN	Mgso4	Mode of delivery	Indication	Intrapartum complication	CNS complication	DIC, PPH	Abruption	Pulmonary edema	HELLP	Renal	ICU admission	Maternal Outcome	Neonatal Outcome	Term/Preterm	Birth weight	NICU admission	Indication	Fundoscopy
116	10020814	25	primigravida	36 week 2 day	normotensive	nil	21.4	126/80	95.3	1+	1.68	16.6	0.2	11.8	24	normal	0	no	PTVD		none	nil	no,yes	yes	no	no	no	no	stable	live	preterm	2.24kg	no		normal
117	10019738	23	primigravida	33week 5 day	normotensive	IUGR, Polyhydramnios	21.6	118/70	86	nil	1.72	12.1	0.18	10.7	22	incresed resistance	0	no	PRETERM LSCS	APH	none	nil	no,no	yes	no	no	no	no	stable	live	preterm	1.4kg	yes	distress	normal
118	10020785	22	primigravida	35 week 5 days	normotensive	oligohydramnios	22.7	122/80	94	nil	2.28	15.4	0.26	11.6	24	normal	0	no	PTVD		none	nil	no,no	no	no	no	no	no	stable	live	preterm	2.7 kg	no		normal
119	10021972	22	G4P1L1A2	35week 6 day	normotensive	nil	22.7	118/76	90	nil	1.93	14.1	0.22	11.5	23	normal	0	no	PRETERM LSCS	Prev lscs , anhydramnios	none	nil	no,no	no	no	no	no	no	stable	live	preterm	2.6kg	no		normal
120	10022019	25	G2P1L1	33week 6 day	normotensive	nil	21.6	122/70	87.3	nil	1.92	9.7	0.19	9.7	19.7	normal	0	no	PTVD		none	nil	no,no	no	no	no	no	no	stable	live	preterm	1.8kg	no		normal
121	10022320	29	primigravida	39week 1 day	severe PE	nil	21.9	146/100	115.3	3+	2.02	15.2	0.24	11.9	16.97	normal	2	yes	TERM LSCS	imminent sign	headache , vomiting	PRESS	no,no	no	no	no	no	no	stable	live	Term	2.9kg	no		normal
122	10022492	26	G3P2L2	37week 5 day	normotensive	nil	22.8	124/78	93.3	nil	1.69	13.4	0.19	11.3	23	normal	0	no	TERM LSCS	prevlscs not w/f VBAC	none	nil	no,no	no	no	no	no	no	stable	live	Term	2.6kg	no		normal
123	10022655	26	G2P1L1	38 week 5 day	Normotensive	nil	23.8	118/76	90	nil	2.32	11.9	0.25	10.6	21.8	normal	0	no	TERM LSCS	prev LSCS in labour	none	nil	no,no	no	no	no	no	no	stable	live	Term	3.1kg	no		normal
124	10022704	23	primigravida	37week 2day	normotensive	nil	22.2	120/80	93.3	nil	1.84	12.9	0.2	11.1	24	normal	0	no	FTND		none	nil	no,no	no	no	no	no	no	stable	live	Term	2.3kg	no		normal
125	10022710	23	primigravida	38week 1 day	normotensive	nl	22.4	116/84	94.6	nil	2.27	17.7	0.28	12.5	21	normal	0	no	FTND		None	nil	no,no	no	no	no	no	no	stable	live	Term	2.8kg	no		normal
126	10022885	40	primigravida	36 week 1 day	severe PE	severe FGR , Anmanios	22.4	160/110	126.6	3+	2.45	9.4	0.23	9.4	19	increased resistance	2	yes	Pt emergency LSCS	severe PE , Anamnios	none	nil	no,no	no	no	no	no	no	stable	live	preterm	1.5kg	yes	LBW	normal
127	10022906	30	primigravida	39 week 3 day	normotensive	oligohydramnios	22.2	122/80	94	nil	2.18	14.2	0.24	10.8	23	normal	0	no	TERM LSCS	Pathological trace	none	nil	no,no	no	no	no	no	no	stable	live	Term	3kg	no		normal
128	10022916	20	primigravida	39 week 5 day	Normotensive	nil	24.5	118/80	92.6	1+	2.09	10.2	0.21	9.9	21	normal	0	no	FTND		none	nil	no,no	no	no	no	no	no	stable	live	Term	3kg	no		normal
129	10023015	31	G2P1L1	40 week	non severe PE	nil	20.8	144/90	108	1+	3.04	11.8	0.34	11.1	27.3	normal	1	no	FTND		none	nil	no,no	no	no	no	no	no	stable	live	Term	2.9kg	no		normal
130	10023027	24	primigravida	39 week 5 days	normotensive	nil	21.1	120/78	92	nil	1.7	17.1	0.2	11.7	24	normal	0	no	TERM LSCS	Thick MSL	none	nil	no,no	no	no	no	no	no	stable	live	Term	2.8kg	no		normal
131	10023181	31	primigravida	36 week 6 day	normotensive	nil	21.1	112/68	82.6	nil	2.15	12.8	0.24	11	20	normal	0	no	PRETERM LSCS	Thick MSL	none	nil	no,no	no	no	no	no	no	stable	live	Term	2.4kg	no		normal
132	10023081	24	G2P1L1	38 week 3 day	Normotensive	nil	20	118/78	91.3	nil	2.77	12.8	0.3	10.7	25.8	normal	0	no	FTND		none	nil	no,no	no	no	no	no	no	stable	live	Term	3.2kg	no		normal
133	10021867	27	primigravida	34 week 3 days	normotensive	IUGR	21.4	122/80	94	nil	2.1	10	0.2	9.7	22	increased resistance	0	no	PRETERM LSCS	oligohydramnios	none	nil	no,no	no	no	no	no	no	stable	live	preterm	1.5kg	Yes	LBW	normal
134	10023261	19	Primigravida	38 week 2 day	normotensive	nil	22.8	110/68	82	nil	2.32	12.1	0.24	10.4	22.8	normal	0	no	TERM LSCS	oligohydramnios	none	nil	no,no	no	no	no	no	no	stable	live	Term	2.9kg	No		normal
135	10022904	24	G2P1L1	37 week 3 day	Normotensive	polyhydramnios	23.5	122/60	80.6	nil	3.68	8.9	0.34	9.2	40	normal	0	no	TERM LSCS	prev LSCS in labour	none	nil	no,no	no	no	no	no	no	stable	live	Term	2.9kg	no		normal
136	10022641	26	G2P1L1	38 week 2 day	Normotensive	nil	22.8	118/70	86	nil	1.92	10.1	0.18	9.4	20.4	normal	0	no	TERM LSCS	prev LSCS in labour	none	nil	no,no	no	no	no	no	no	stable	Live	Term	2.8kg	no		normal
137	10023343	31	G3P1L1A1	38 week 4 day	Normotensive	nil	24.9	110/60	76.6	nil	2.14	9.8	0.21	9.6	22.2	normal	0	no	FTND		none	nil	no,no	no	no	no	no	no	stable	Live	Term	2.9kg	no		normal
138	10023518	23	Primigravida	37 week 3 day	severe PE	nil	22.8	150/90	110	2+	1.88	15.2	0.23	12	15.6	normal	2	yes	TERM LSCS	severe PE , imminent sign	headache , vomiting	nil	no,no	no	no	no	no	no	stable	live	term	2.3kg	no		normal
139	10023727	28	G2A1	35 week 4 day	Normotensive	nil	20.8	112/78	89.3	nil	2.8	10.1	0.27	9.7	28.8	normal	0	no	PTVD		none	nil	no,no	no	no	no	no	no	stable	live	preterm	2.1kg	yes	LBW	normal
140	10023671	23	primigravida	37 week 6 day	Normotensive	nil	22.2	118/82	94	nil	2.62	9.3	0.25	9.4	27.87	normal	0	no	FTVD		none	nil	no,no	no	no	no	no	no	stable	live	Term	2.75kg	no		normal
141	10023383	27	G3P1L1D1	36 week 2 day	Normotensive	nil	22.8	120/80	93.3	nil	2.04	13	0.22	10.7	21	normal	0	no	PRETERM LSCS	Scar tenderness	none	nil	no,no	no	no	no	no	no	stable	live	preterm	2.9kg	no		normal
142	10022474	21	Primigravida	32 week 5 days	severe PE	nil	26.6	144/92	109.3	4+	2.47	11.7	0.27	10.8	22.8	normal	2	yes	PRETERM LSCS	Severe Distress	none	nil	no,no	no	no	no	no	no	stable	live	preterm	1.46kg	no		normal
143	10022213	31	G2P1L1	34 week 3 days	normotensive	hypothyrodism	24.7	112/80	90.6	nil	2.18	10.9	0.21	9.8	24	normal	0	no	PRETERM LSCS	prev lscs in labour	none	nil	no,no	no	no	no	no	no	stable	Live	preterm	2.9kg	no		normal
144	10023984	25	G2P1L1	37 week 3 day	normotensive	nil	23.1	110/70	83.3	nil	2	12	0.21	10.3	23	normal	0	no	FTND		none	nil	no,no	no	no	no	no	no	stable	Live	Term	2.6kg	no		normal
145	10024001	22	primigravida	27 week	severe PE	IUGR	24.1	144/90	108	2+	3.58	10.2	0.33	9.2	30	normal	1	yes	PTVD		none	nil	no,no	no	no	no	no	no	stable	live	preterm	530gm	yes	VLBW	normal
146	10024230	22	G2P1L1	37 week 2 day	normotensive	nil	24	122/80	94	nil	2.61	15.5	0.29	11.2	23.3	normal	0	no	FTND		NONE	Nil	no,no	no	no	no	no	no	stable	live	Term	3kg	no		normal
147	10023886	24	G2P1L1	38 week 2 day	normotensive	nil	23.1	110/70	83.3	nil	2.23	10.8	0.22	9.7	22.9	normal	0	no	TERM LSCS	prev lscs oligohydramnios	none	nil	no,no	no	no	no	no	no	stable	live	Term	2.4kg	no		normal
148	10023647	25	G2p1l1	34 week 3 days	normotensive	IUGR	23.7	112/80	90.6	nil	2.29	13.9	0.26	11.3	20.2	normal	0	no	PRETERM LSCS	MCDA twin	none	nil	no,no	no	no	no	no	no	stable	live	preterm	1.8kg, 2kg	yes	LBW	normal
149	10024226	21	primigravida	37 week	normotensive	IUGR	24	120/78	92	1+	2.2	12.7	0.23	10.6	20.75	normal	0	no	TERM LSCS	oligihydramnios	none	nil	no,no	no	no	no	no	no	stable	Live	Term	2.2kg	no		normal
150	10024542	23	G2P1L1	33 week 2 day	normotensive	IUD, MCDA	25.3	118/76	90	nil	2.51	10.9	0.26	10.3	24.3	IUD	0	no	PRETERM LSCS	MCDA twin	none	nil	no,no	no	no	no	no	no	stable	live	preterm	1.2kg , 1.7kg iud	no		nomal
151	10024537	24	G2P1L1	34 week 1 day	non severe PE	IUD	23	140/92	108	3+	1.57	13	0.17	11.1	14.1	normal	1	yes	PTVD		none	nil	yes,no	no	no	yes	no	no	stable	MSB	preterm	2kg	no		normal
152	10027081	24	G2P1L1	34 week 5 days	Non severe PE	nil	23.8	140/90	106.6	1+	1.23	20.5	0.15	12.6	9.76	normal	1	no	Pt emergency LSCS	MCDA twin	none	nil	no,no	no	no	no	no	no	stable	live	preterm	1.76kg/1.73kg	yes	fetal distress	normal
153	10027942	26	G4P1L1A2	37 week 4 days	Non severe PE	nil	24.5	142/90	107.3	1+	2.06	11.3	0.22	10.5	19.6	normal	1	no	FT emergency LSCS	Prevs LSCS non sev PE	none	nil	no,no	no	no	no	no	no	stable	live	Term	3.34 kg	no		normal
154	10027015	32	G2P1L1	36 week 6 day	Non severe PE	nil	25.5	140/90	106.3	1+	1.6	16.2	0.19	12.2	13.11	Increased resistance	1	no	Pt emergency LSCS	PE with IUGR	none	nil	no,no	no	no	no	no	no	stable	live	preterm	2.1 kg	no		normal

S.No	IP Number	Age	Obs Score	POG	Diagnosis	Antenatal complication	BMI	BP	MAP	Protenuria	Platelet	PDW	Plateletcrit	MPV	PC/MPV	Doppler	No of anti HTN	Mgso4	Mode of delivery	Indication	Intrapartum complication	CNS complication	DIC, PPH	Abruption	Pulmonary edema	HELLP	Renal	ICU admission	Maternal Outcome	Neonatal Outcome	Term/Preterm	Birth weight	NICU admission	Indication	Fundoscopy
155	10030054	24	G2P1L1	39 week 5 day	Non severe PE	nil	26.1	142/92	108.3	1+	2.46	13.2	0.27	11	22.3	normal	1	no	FT elective LSCS	Transvere lie with PE	none	nil	no,no	no	no	no	no	no	stable	live	Term	3.3 kg	no		normal
156	10029330	35	G2P1L1	28 week 2 day	Non severe PE	nil	24.7	140/90	106.6	2+	2.87	8.4	0.25	8.8	32.6	normal	1	no	Pt emergency LSCS	scar tenderness	none	nil	no,no	no	no	no	no	no	stable	live	preterm	900 gm	no		normal
157	10030730	26	G2P1L1	38 weeks 4 days	Non severe PE	nil	24.7	140/94	109.3	1+	1.71	10	0.17	9.8	17.44	normal	1	no	FT emergency LSCS	prev lscs with PE	none	nil	no,no	no	no	no	no	no	stable	live	Term	2.8 kg	no		normal
158	10043765	23	primigravida	30 week 3 days	non severe PE	nil	23.6	140/90	106.6	2+	2.33	10.6	0.23	10	23.3	normal	1	no	PTVD		none	nil	no,no	no	no	no	no	no	stable	FSB	preterm	800gm	no		
159	10045662	30	G3P1L1A1	37 week 1 day	non severe PE	nil	23	144/90	108	2+	3.11	9.7	0.3	9.8	31.73	normal	1	no	FTVD		none	nil	no,no	no	no	no	no	no	stable	live	term	2.6kg	no		normal
160	10045703	28	primigravida	38 week 1 day	non severe PE	nil	23.6	140/90	106.6	2+	1.73	10	0.23	10	17.3	normal	1	no	Ft emergency LSCS	non progress labour	none	nil	no,no	no	no	no	no	no	stable	live	term	2.8kg	no		normal
161	10045992	20	G2P1L1	37 week 5 days	non severe PE	oligohydramnios	23.2	142/82	102	1+	2.29	11.8	0.24	10.5	21.8	normal	1	no	FTVD		none	nil	no,no	no	no	no	no	no	stable	live	term	2.16kg	no		normal
162	10046376	24	G4P2L2A1	37 week 2 day	non severe PE	nil	22.2	140/94	109.3	1+	1.37	14.5	0.16	11.9	11.51	normal	1	no	FT elective LSCS	prev 2 LSCS	none	nil	no,no	no	no	no	no	no	stable	live	term	2.4kg	no		normal
163	10046932	31	G4P3L3	39 week	non severe PE	nil	23.2	140/92	108	1+	2.87	13	0.31	10.9	26.3	normal	1	no	FTND		none	nil	no,no	no	no	no	no	no	stable	live	Term	2.7kg	no		normal
164	10046941	37	G2P1L1	34 week 2 day	non severe PE	oligohydramnios	23.4	140/90	106.6	2+	2.16	11.6	0.21	10	21.6	normal	1	no	FT elective LSCS	prolonged PPROM	none	nil	no,no	no	no	no	no	no	stable	live	preterm	2.5kg	no		normal
165	10047108	23	G2P1L1	38 week 6 day	non severe PE	nil	24	142/90	107.3	2+	2.17	10.6	0.21	10.6	20.47	normal	1	no	FTND		none	nil	no,no	no	no	no	no	no	stable	live	Term	2.9kg	no		normal
166	10047187	21	primigravida	40 week	non severe PE	nil	26.1	140/90	106.6	1+	2.51	12.9	0.27	10.8	23.2	normal	1	no	Ft emergency LSCS	PE with NPL	none	nil	no,no	no	no	no	no	no	stable	live	term	3.2kg	no		normal
167	10047630	23	primigravida	41 week	non severe PE	nil	24.7	142/90	107.3	1+	1.1	14.5	0.13	11.6	9.4	normal	1	no	Ft emergency LSCS	fetal distress	none	nil	no,no	no	no	no	no	no	stable	live	Term	3.3kg	no		grade 3 retinopathy
168	10047602	24	primigravida	39 week 2 day	non severe PE	nil	24.5	140/90	106.6	1+	2.36	12	0.24	10.4	22.6	normal	1	no	Ftemergency LSCS	oligohydramnios	none	nil	no,no	no	no	no	no	no	stable	live	Term	2.6kg	no		normal
169	10047509	39	primigravida	36 week 4 days	non severe PE	nil	22.8	140/90	106.6	2+	3.92	9.9	0.37	9.9	30	normal	1	no	PT emergency LSCS	oligohydramnios	none	nil	no,no	no	no	no	no	no	stable	live	preterm	2.16kg	no		normal
170	10047699	25	G2A1	36 week 6 day	non severe PE	nil	23.8	142/90	107.3	2+	2.19	16.3	0.26	12.1	18	normal	1	no	PT emergency LSCS	CPD	none	nil	no,no	no	no	no	no	no	stable	live	preterm	2.37kg	no		normal
171	10050497	24	primigravida	38 week 6 days	non severe PE	nil	21.6	150/90	110	1+	2.55	10.2	0.25	9.8	26	normal	1	no	Ft emergency LSCS	fetal distress	none	nil	no,no	no	no	no	no	no	stable	live	Term	3.2kg	no		normal
172	10050708	26	primigravida	37 week 1 day	non severe PE	nil	22.7	142/90	107.3	1+	2.44	11.8	0.26	10.5	23.2	normal	1	no	Ftemergency LSCS	fetal macrosomia	none	nil	no,no	no	no	no	no	no	stable	live	Term	4.3kg	no		normal
173	10051338	25	primigravida	40 week	non severe PE	nil	23.2	140/90	106.6	1+	2.5	9.9	0.24	12.1	20.6	normal	1	no	FTVD		none	nil	no,no	no	no	no	no	no	stable	live	Term	2.9kg	no		normal
174	10031385	21	primigravida	40 week 4 day	non severe PE	nil	23.2	142/90	107.3	1+	3.73	9.5	0.35	9.3	30	normal	1	no	FTVD		none	nil	no,no	no	no	no	no	no	stable	live	Term	2.4kg	no		normal