# "A STUDY ON THE ASSOCIATION OF ELEVATED D-DIMER LEVELS AND THE EXTENT OF SEVERITY OF PRE ECLAMPSIA"

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## CERTIFICATE BY THE INSTITUTION

This is to certify that the dissertation entitled **"A STUDY ON THE ASSOCIATION OF ELEVATED D-DIMER LEVELS AND THE EXTENT OF SEVERITY OF PRE ECLAMPSIA"** is a bonafide record of the work done by **Dr M.MONISHA** at Government R.S.R.M Lying in Hospital, Stanley Medical College, Chennai-1. This dissertation is submitted to Tamil Nadu Dr M.G.R Medical University in partial fulfilment of university rules and regulations for the award of M.S Degree in Obstetrics and Gynaecology.

**Dr. P. BALAJI MS, FRCS, FCLS, PHD**
Dean,
STANLEY MEDICAL COLLEGE,
Chennai-1

**Dr. V.RAJALAKSHMI MD (OG), DGO**
Superintendent,
Govt. R.S.R.M Lying in Hospital,
Stanley Medical College,
Chennai-13

## CERTIFICATE BY THE GUIDE

This is to certify that this dissertation entitled **"A STUDY ON THE ASSOCIATION OF ELEVATED D-DIMER LEVELS AND THE EXTENT OF SEVERITY OF PRE ECLAMPSIA"** submitted by **DR.M.MONISHA** appearing for M.S.,Obstetrics and Gynaecology(Branch II) Degree Examination in MAY 2022 is a bonafide record of work done by her, under my direct guidance and supervision as per the rules and regulations of the Tamilnadu Dr.M.G.R.Medical University, Chennai. I forward this dissertation to the Tamilnadu Dr.M.G.R Medical University, Chennai, Tamilnadu, India.

**Dr MANICKADEVI MD(OG),DCE.,**
ASSOCIATE PROFESSOR,
DEPARTMENT OF OBSTETRICS & GYNAECOLOGY,
GOVT. R.S.R.M LYING IN HOSPITAL,
STANLEY MEDICAL COLLEGE, Chennai - 13

## DECLARATION BY THE CANDIDATE

I, **Dr M.MONISHA**, solemnly declare that the dissertation **"A STUDY ON THE ASSOCIATION OF ELEVATED D-DIMER LEVELS AND THE EXTENT OF SEVERITY OF PRE ECLAMPSIA"** is a bonafide work done by me at Govt. R.S.R.M Lying in Hospital, Stanley Medical College, Chennai, under the supervision and guidance of Associate Professor **DR MANICKADEVI M.D(OG), DCE**, Department of Obstetrics and Gynaecology, Government RSRM Lying In Hospital, Stanley Medical College, Chennai. This thesis is submitted to The Tamil Nadu Dr M.G.R Medical University in partial fulfilment of the rules and regulations for the M.S Degree examinations in obstetrics and Gynaecology to be held in MAY 2022.

Place: Chennai

Date :

DR.M.MONISHA,
M.S Post Graduate in Obstetrics and Gynaecology,
Govt. R.S.R.M Lying in Hospital,
Stanley Medical College, Chennai-13.

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**CONTENTS**

# *INTRODUCTION*

# INTRODUCTION

In normal pregnancy, hemostatic changes occurs with increased levels of coagulation factors and suppression of fibrinolysis. Thus pregnancy is considered to be a state of hypercoagulability. This physiological change happens to ensure effective control of bleeding from placental site and to prevent intrapartum and postpartum hemorrhage(1). Hypertensive disorder constitutes 7-15% of all gestations and a quarter of all antenatal admissions. About 16% of maternal deaths are due to hypertensive disorders in developed countries(2). There is accentuation of hypercoagulable state of pregnancy during eclampsia and pre-eclampsia. Postmortem evidences in women who have died of eclampsia show fibrin deposition in the blood vessels of vital organs.

Pre-eclampsia, a pathology of pregnancy by nature, is a dynamic process of varying severity associated with high levels of maternal and fetal morbidity and mortality. Pre-eclampsia involves an increase in inflammatory mediators and pro-coagulation factors and decreased amount of fibrinolytics in response to cellular dysfunction attributable to the condition. The state of hypercoagulability rendered by pregnancy worsens and organ malfunction can occur.

D-dimer is a fibrin degradation product and the main component of a thrombus. Its presence suggests activation of the coagulation cascade and fibrinolysis. The clinical utility of measuring D-dimer lies in its high negative predictive value (98%–100%) and high sensitivity (95%–100%) for detection of thromboembolic events. For this reason, it is part of the diagnostic algorithm of deep vein thrombosis and pulmonary thromboembolism.

Nonetheless, several factors modify the operational characteristics of the test. During pregnancy, D-dimer levels increase progressively from the first trimester and still more during the postpartum period, which makes interpretation of D-dimer levels difficult, especially when standard cut-off points for a non-pregnant population are used. 14.7% of patients in the first trimester, 70.8% in the second trimester, and 95.9% in the third trimester had D-dimer levels above the reference value. Réger et al. quantified an average increase between weeks 16 and 26 of 133% and between weeks 26 and 36 of 156.0% with respect to levels prior to pregnancy(3).

Studies carried out on different populations10–18 have established cut-off points for different laboratory techniques in each trimester of pregnancy. Regarding pre-eclampsia, most researchers found increased D-dimer concentrations in patients with pre-eclampsia compared with pregnant patients with normal blood pressure, but some authors did not find significant differences.

It would be helpful in clinical practice to have a test that efficiently predicts the development of pre-eclampsia. Heilmann et al.(4) and Hale et al.(5) discovered that D-dimer levels had a significant relation in patients with early-onset pre-eclampsia compared with normotensive patients or those with late-onset pre-eclampsia. It has also been confirmed that D-dimer is significantly elevated in pregnant patients who have severe pre-eclampsia versus normotensive patients or those with mild pre-eclampsia.

D dimer is an excellent predictor of the severity of pre-eclampsia, with an area under the receiver operating characteristic (ROC) curve of 90%. Nonetheless, a meta-analysis concluded that although the results indicated that pre-eclampsia patients had increased levels of D-dimer, the severity of the disease was not mentioned, nor was the diagnostic or prognostic value of this diagnostic tool tested. It was found that patients with HELLP (hemolysis, elevated liver enzymes and low platelets) syndrome had D-dimer levels

significantly higher than patients with minor preeclampsia. D dimer levels are classed as elevated when they are greater than the 97.5 percentile(6).

**TABLE 1** D-dimer reference values in normal pregnancy using immunoturbidimetry adjusted for gestational age.

| Week of pregnancy | 2.5th P in mg FEU/L | 97.5 P in mg FEU/L |
|---|---|---|
| 13–20 | 0.2 | 1.4 |
| 21–28 | 0.3 | 1.7 |
| 29–34 | 0.3 | 3.0 |
| 35–42 | 0.3 | 3.1 |

Abbreviations: P, percentile; mg FEU/L, mg/L fibrinogen equivalent unit.

Imbalance in blood coagulation and fibrinolytic mechanisms cause diminished coagulation or excessive fibrinolysis which may lead to failure of hemostasis. In such cases, varying degrees of disseminated intravascular coagulation can contribute significantly to the maternal morbidity and mortality(7). Early detection of severity of pre-eclampsia is necessary to prevent complications. Hence this prospective study is undertaken to assess the severity of pre-eclampsia using D-dimer levels that will guide in the management and prevention of complications in pregnancy induced hypertension.

# *AIM AND*

# *OBJECTIVE OF*

# *THE STUDY*

## AIM OF THE STUDY

To study the association of elevated D-dimer levels and extent of severity of preeclampsia.

## OBJECTIVE OF THE STUDY

- To establish the association of increased D-Dimer values and severity of preeclampsia.
- To know whether it influences the maternal and foetal outcome

# *REVIEW OF LITERATURE*

# REVIEW OF LITERATURE

Pregnancy is characterized by a physiological rise in the strain exerted upon the endothelium. During pregnancy, the woman undergoes physiological changes in order to accommodate the developing foetus. It is essential to understand the normal physiological changes occurring in pregnancy as tis will help to differentiate from adaptations that are abnormal as well to determine and treat obstetric complications associated with hemostatic changes[6].

## HISTORY

The scientific contribution throughout history has influenced our current understanding about the etiology and pathogenesis of pre eclampsia. In ancient Greece, in the late 5$^{th}$ and early 4$^{th}$ century, the Hippocrates described the theory of four humors to describe the disease(8). They believed that the body is made up of four humors that included blood, phlegm, yellow bile and black bile.

The wet and dry theory was used to explain the vulnerability of female physiology to disease. Women's flesh was porous and soft and at risk of drawing too much moisture and overabundance of fluids and subsequent illness(9) and hence females are considered to be wet. Pre eclampsia was not formally classified as a disorder of pregnancy during ancient times.

Near the end of Renaissance, the classification of disease progressed. Gabelchoverus distinguished between four types of epilepsy which included epilepsy resulting from head, stomach, the pregnant uterus, and the chilled extremities. It wasn't until 1619 that the word eclampsia first appeared in Varandaeus treatise on Gynecology.

At the end of 18th century and through the 19th century, the classification of pre eclampsia-eclampsia continued to become more refined as the classic signs and symptoms of pre eclampsia-eclampsia became more readily recognized. In 1797, Demanet noted a relation between edematous women and eclampsia (Chelsey 1978) whereas John Lever discovered albumin in the urine of eclamptic women in 1843 (Thomas 1935). The connection between premonitory symptoms during the advanced gestation age of pregnancy and the development of puerperal convulsions was also recognized in 1843 by Dr. Robert Johns. Theses premonitory symptoms include headache, temporary loss of vision, severe pain in the stomach and edema of the hands, arms, neck and face (Johns, 1843). In 1897, Vaquez and Nobecourt were credited for the discovery of eclamptic hypertension (Chelsey, 1978). As a result of these contributions, the concept of pre eclamptic state was recognized(8).

The increase in blood volume is meant for

- Extra blood flow to the uterus and placenta
- Filling the expanded vascular system
- Protection against blood loss at delivery

The biggest discrepancy between plasma and red cell mass happens in the late second and early third trimester and physiological anemia is most common at 28-36 weeks.

## CLASSIFICATION OF HYPERTENSIVE DISORDERS OF PREGNANCY

Hypertensive disorders during pregnancy can be classified into four well defined groups(10):

- Gestational hypertension
- Pre eclampsia and eclampsia syndrome

- Chronic hypertension of any etiology
- Pre eclampsia superimposed on chronic hypertension

  Hypertension is diagnosed empirically when appropriately taken blood pressure exceeds 140mmHg systolic or 90mmHg diastolic. Korotkoff phase V is used to define diastolic pressure. A sudden rise in mean arterial pressure later in pregnancy also known as "delta hypertension" may also signify pre eclampsia even if blood pressure is less than 140/90mmHg.

**GESTATIONAL HYPERTENSION**

Gestational hypertension is defined as a systolic blood pressure of 140 mmHg or more or a diastolic blood pressure of 90 mmHg or more or both, on two occasions atleast 4 hours apart after 20 weeks of gestation, during labour, or in the first 24 hours postpartum, without proteinuria, or any systemic features of pre eclampsia in a previously normotensive non-proteinuric woman and the blood pressure returns to normal within 3 months postpartum.

Its prevalence is between 6-15% in nulliparous and 2-4% in multiparous(2).

**PRE ECLAMPSIA**

Pre-eclampsia is a multiorgan disease process of unknown aetiology characterized by de novo development of hypertension and proteinuria after 20weeks of gestation sometimes progressing into a multiorgan cluster of varying clinical features.

As per the new ACOG Practice Bulletin, although hypertension and proteinuria are the classical criteria for the diagnosis of pre-eclampsia, the presence of any of the following severe features in women with gestational hypertension without proteinuria should also be

classified as pre-eclampsia. The following features are included in the diagnostic criteria for pre-eclampsia(11).

- Thrombocytopenia (platelet count less than 1,00,000/$mm^3$)
- Impaired liver function as indicated by abnormally elevated blood concentrations of liver enzymes (to twice the upper limit of normal concentration)

Severe persistent right upper quadrant or epigastric pain and not accounted for by alternate diagnosis

- Renal insufficiency (serum creatinine concentration greater than 1.1mg/dL or a doubling of the serum creatinine concentration in the absence of other renal diseases)
- Pulmonary edema
- New-onset headache unresponsive to acetaminophen and not accounted for by alternative diagnosis or with visual disturbances.

Pre-eclampsia is classified into severe and non-severe pre-eclampsia(12).

**NONSEVERE PRE-ECLAMPSIA**

The non-severe category includes what was earlier called mild and moderate pre-eclampsia. Pre eclampsia is defined as Hypertension associated with proteinuria greater than 0.3g/L in a 24 hour urine collection or 1+ by qualitative urine examination, after 20 weeks of gestation.

**SEVERE PRE-ECLAMPSIA**

As per the most accepted guidelines, severe pre-eclampsia is pre-eclampsia with severe hypertension and/or with symptoms and/or biochemical and/or hematological impairment. Severe pre-eclampsia is confirmed by a diastolic BP≥110mmHg on two occasions, or a

systolic BP≥160mmHg on two occasions, together with significant proterinuria (atleast 1g/L)(13). Women with gestational hypertension without proteinuria with severe range BPs(systolic BP of 160mmHg or higher, or diastolic BP of 110mmHg or higher) should also be diagnosed as pre-eclampsia with severe features.

Clinical features of pre-eclampsia are(14):

- Severe headache
- Visual disturbances
- Epigastric pain and/or vomiting
- Signs of clonus
- Papilloedema
- Liver tenderness
- Platelet count below 1lakh/mm3
- Abnormal liver enzymes(ALT or AST rising to above 70 IU/L)
- HELLP syndrome

**ECLAMPSIA**

Eclampsia is the convulsive manifestation of the hypertensive disorders of pregnancy and is among severe manifestations of the disease. Eclampsia is defined by new onset tonic-clonic, focal or multifocal seizures in the absence of other causative conditions such as epilepsy, cerebral arterial ischemia and infarction, intracranial hemorrhage, or drug use. Some of the alternative diagnoses may be more likely if new onset seizures occur after 48-72 hours postpartum or when seizures occur during administration of magnesium sulphate.

## CHRONIC HYPERTENSION

Chronic hypertension is defined as hypertension present before 20$^{th}$ week of pregnancy or that is diagnosed preconceptionally. Hypertension should be documented on at least two occasions, measurea at least 4 hours apart. Blood pressure elevation that persists >12 weeks postpartum is also retrospectively considered as chronic hypertension.

- **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, endocrine disorders or coarctation of aorta(2).

### *Aetiology of Chronic Hypertension*

- Renal parenchymal disease (glomerulonephritis, reflux nephropathy, adult polycystic disease)
- Reno-vascular hypertension (renal artery stenosis)
- Diabetes with vascular involvement
- Thyrotoxicosis
- Pheochromocytoma
- Primary aldosteronism
- Cushing syndrome
- Systemic lupus erythematosus
- Scleroderma
- Aortic coarctation

## PRE ECLAMPSIA SUPERIMPOSED ON CHRONIC HYPERTENSION

It is diagnosed when one or more features of pre eclampsia (elevated liver enzymes, low platelets, proteinuria) develop for the first time during pregnancy after 20 weeks, in a woman with pre-existing chronic hypertension.

## HELLP SYNDROME

The clinical presentation of hemolysis, elevated liver enzymes and low platelet count (HELLP) syndrome is one of the more severe forms of pre eclampsia because it is associated with increased rates of maternal morbidity and mortality. Although different theories have been proposed, many clinicians use the following criteria to arrive at the diagnosis:

- Lactate dehydrogenase (LDH) elevated to 600 IU/L or more
- Aspartate aminotransferase (AST) and alanine aminotransferase (ALT) elevated more than twice the upper limit of normal
- Platelet count less than 100,000/mcL

Although HELLP syndrome is mostly a third trimester condition, in 30% of cases it is expressed or progresses postpartum. HELLP syndrome has an insidious and atypical onset, with upto 15% cases lacking either hypertension or proteinuria. In HELLP syndrome, the main presenting complaints are right upper quadrant pain and generalized malaise in upto 90% cases and nausea and vomiting in 50% of cases.

## PHYSIOLOGICAL CHANGES DURING PREGNANCY

### BLOOD VOLUME

Blood volume increases by 40-50%. It starts in the first trimester(6 weeks), reaches a peak by 32-34 weeks and plateaus thereafter. Plasma volume increases by 50% but the red cell volume increases by 30%. This gives rise to 'physiological hemodilution' or 'physiological anemia' of pregnancy, the increase in red cells is due to increase in erythropoietin levels, beginning at 10 weeks and continuing till term.

### COAGULATION SYSTEM

Pregnancy is a hypercoagulable state caused by an increase in coagulation factors and a decrease in inhibitors of coagulation and fibrinolytic activity.

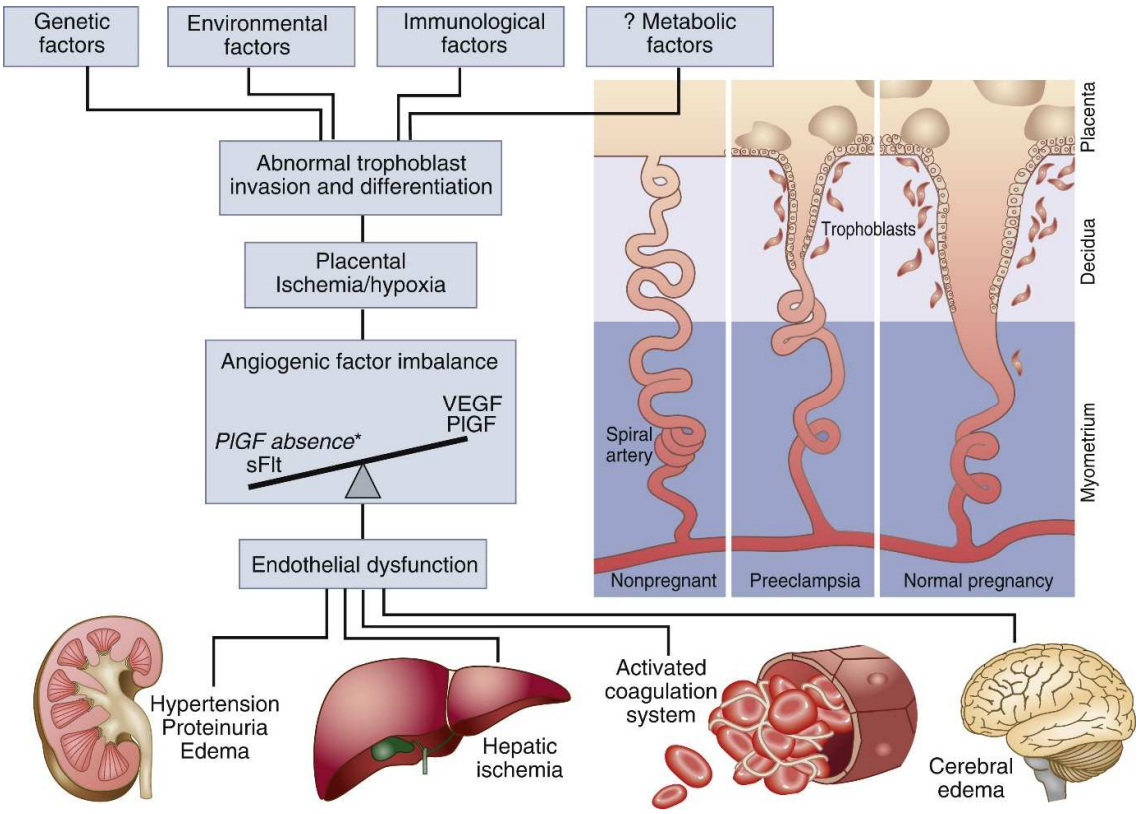
- Increase in all clotting factors except XI and XIII
- Decrease in fibrinolytic activity

  -decrease in plasminogen activator

  -increase in plasminogen activator inhibitor
- Decrease in antithrombin
- Decrease in Protein S

  The commonly used coagulation tests (bleeding time, clotting time, prothrombin time and activated partial thromboplastin time) are unaffected.

**FIGURE 1: RISK FACTORS AND DISEASE PATHOGENESIS**




**RISK FACTORS FOR PRE-ECLAMPSIA**

Factors associated with increased risk of pre-eclampsia are[5]:

- Extremes of age(<18 or >35yrs)
- Obesity(BMI>30) and insulin resistance/gestational diabetes
- Smoking
- Pregnancy after assisted reproductive technology
- Multifetal pregnancies
- Pre-eclampsia in previous pregnancy
- Maternal low birth weight
- Family history of pre-eclampsia
- Maternal immunological diseases/SLE

- Pre-existing thrombophilia
- Antiphospholipid antibody syndrome

**AETIOPATHOGENESIS**

Various mechanisms for the pathogenesis of pre-eclampsia have been proposed.

- Chronic utero-placental ischemia
- Immune maladaptation
- Very low density lipoprotein toxicity
- Genetic imprinting
- Increased trophoblastic apoptosis
- Exaggerated maternal inflammatory response to deported trophoblasts and imbalance of angiogenic factors or a combination of any of the above.

The abnormal trophoblastic invasion of spiral arteries, inappropriate endothelial cell activation and exaggerated inflammatory response are key features in the pathogenesis of pre-eclampsia. The pre-eclamptic syndrome has been hypothesized as a two-stage disorder.

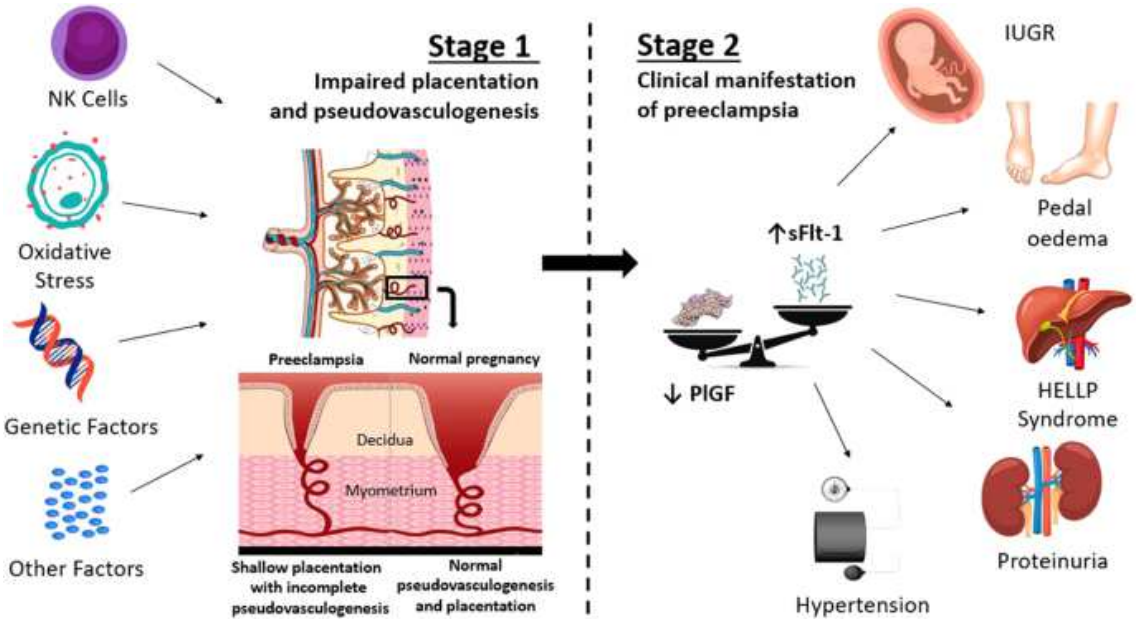
Primary stage involves abnormal placentation. In normal pregnancy, the wall of the spiral arteries is invaded by endovascular trophoblastic cells. This migration transforms the small, musculo-elastic spiral arteries into large, tortuous channels that carry a large amount of blood to the intervillous space are resistant to the effects of vasomotor agents. Although this starts in the first trimester, this invasion is completed in the second trimester after the second wave of trophoblastic invasion. These physiological changes are incomplete in patients with pre-eclampsia and the trophoblastic invasion affects only some of the spiral arteries and does not progress into the myometrial portion of the arteries (incomplete trophoblastic invasion).

This deficiency results in decreased uteroplacental perfusion. In addition to failure of demuscularisation, the arteries maintain their response to the vasomotor influences.

The second stage of pre-eclampsia involves the conversion of this earlier described uteroplacental maladaptation to the maternal systemic syndrome of pre-eclampsia. Stage 2 is amenable to a modification by the pre-existing maternal conditions such as cardiac, renal disease, diabetes, obesity or hereditary influences.

This secondary stage of systemic maternal disease is associated with an exaggerated endothelial cell activation and a generalized hyperinflammatory state. Episodes of placental hypoxia or reperfusion result in oxidative stress, subsequent apoptosis and necrotic disruption of syncytial architecture. The anatomic and physiological disruption of normal placentation is thought to lead to release placental debris from the intervillous space into the maternal circulation thereby inciting a systemic inflammatory response by stimulating the synthesis of inflammatory cytokines, products that affect angiogenesis and abnormal lipid peroxidation. These products will affect the endothelial system with the production of signs and symptoms of multi organ compromise[7].

**FIGURE 2: PRE-ECLAMSPIA - A TWO STAGE DISORDER HYPOTHESIS**




## HEMOSTASIS AND UTEROPLACENTAL CIRCULATION

The process of hemostasis is a dynamic equilibrium between the coagulation and fibrinolytic system. Pregnancy is normally associated with significant changes in all aspects of Virchow's classical triad of venous stasis, endothelial damage and increased coagulation. The primary initiator is tissue factor (TF). TF is a membrane bound non-enzymatic protein. TF is expressed on the surface of cells that are not in contact with blood plasma like fibroblasts and macrophages. When plasma gets exposed to these cells, coagulation cascade is activated outside the damaged endothelium of a blood vessel. Endothelial cells also express TF when stimulated by endotoxin, tumor necrosis factor (TNF), Interleukin-1 and thus may be involved in thrombus formation in pathological conditions.

## MATERNAL CHANGES IN PRE-ECLAMPSIA

### Hemodynamic changes

An increase in maternal cardiac output rather than increased peripheral vascular resistance is the most common hemodynamic feature in pre-eclampsia. However, once pre-eclampsia becomes severe, there is a switch to normal or decreased cardiac output and elevated peripheral vascular resistance as a manifestation of systemic vasoconstriction[8]. There is a crossover to low cardiac output along with increased PVR causing a rise in both systolic and diastolic blood pressure. This causes traumatic intravascular hemolysis (microangiopathic hemolytic anemia)(15).

### Hematological abnormalities

Overt hematological abnormalities exist in only a minority of patients with severe pre-eclampsia. Thrombocytopenia occurs due to increased platelet activation, aggregation and consumption and is a marker of disease severity. The most common is mild thrombocytopenia, which affects 20% of the cases. Overt thrombocytopenia (platelet count<1,00,000/μL) indicates severe disease. Subtle hematological changes similar to mild intravascular coagulation may occur in pre-eclampsia. The levels of factor VIII plasma clotting factor are mildly increased due to consumptive coagulopathy. Except for thrombocytopenia, coagulation aberrations are generally minimal and present only in association with abruption.

The most serious hematological complications of pre-eclampsia is HELLP syndrome that is a form of severe pre-eclampsia that presents with hemolytic anemia, thrombocytopenia and elevated liver enzymes. HELLP syndrome is a manifestation of microangiopathic hemolysis caused by endothelial disruption with platelet adherence and fibrin deposition and not a form

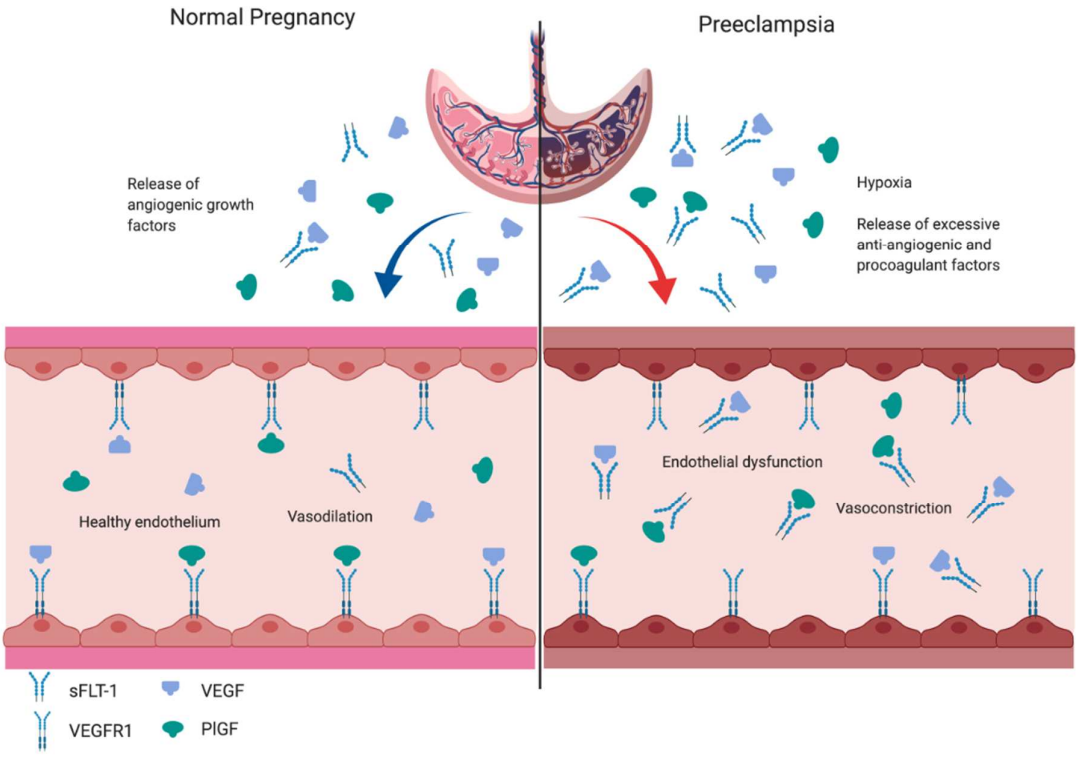
of DIC. Here microangiopathic hemolysis and thrombocytopenia precedes the appearance of consumptive coagulopathy and DIC. The association between thrombophilic factors (factor V Leidin mutation, prothrombin 20210 promoter mutation, methylenetetrahydrofolate reductase (MTHFR) mutation, protein S, plasminogen activator inhibitor (PAI) (4G/4G mutation and antiphospholipid antibodies) and pre-eclampsia has been a subject of multiple investigations resulting in conflicting information.

**Angiogenic and anti-angiogenic proteins**

Placental vasculogenesis is evident after 21 days of conception. Angiogenic imbalance describes excessive amounts of anti-angiogenic factors, which are thought to be stimulated by worsening hypoxia at the uteroplacental interface. Trophoblast of women describes to develop preeclampsia overproduces atleast two antiangiogenic peptides that enter the maternal circulation.

Solube-fms like tyrosine kinase 1 is a receptor for VEGF. Elevated sFlt-1 levels inactivate and reduce circulating free placenta growth factor (PIGF) and VEGF concentrations, leading to endothelial dysfunction. A second anti-angiogenic peptide, soluble endoglin (sEng), inhibits various transforming growth factor beta (TGF-β) isoforms from binding to endothelial receptors. Decreased binding to endoglin diminishes endothelial nitric oxide dependent vasodilation. S-Flt-1 and sEng levels begin to rise months before clinical pre-eclampsia develops(10).

**FIGURE 3: ANGIOGENIC AND ANTI-ANGIOGENIC PROTEINS IN PRE ECLAMPSIA**



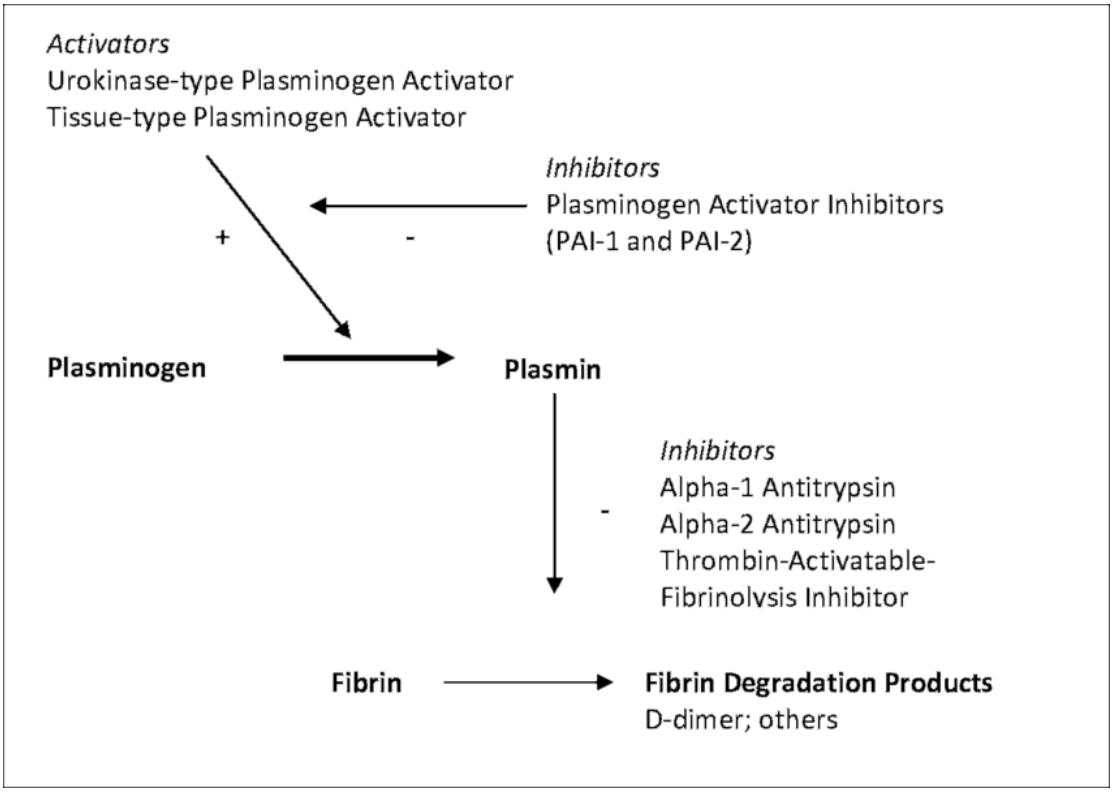

**Natural anticoagulation system**

The anticoagulation system is regulated by protein C and protein S, which forms the central component of this system(16). Tissue factor pathway inhibitor (TFPI), predominantly produced by endothelial cells is a key regulator of thrombin generation(17). TFPI can be short circuited by factor XIa, hence an effective feedback system is needed to regulate the activator of factor IXa and VIIIa. This is achieved by binding of thrombomodulin, a transmembrane glycoprotein of vascular endothelial cells, serves as a receptor for thrombin and form a complex with thrombin which in turn activates protein C to so-called activated protein C (APC). Protein S act as a catalyst for activate protein C. APC inactivates both factor VIIIa and factor Va. Antithrombin III (AT III) is a serine protease inhibitor synthesized in the liver and is a major physiological inhibitor of coagulation. It forms complex with

thrombin forming TAT complex (thrombin-antitrhombin complex) causing activation of blood coagulation system . AT III can also inactivate factors like factor Xa, IXa and VIIa.

**FIBRINOLYSIS IN PRE ECLAMPSIA**

**FIGURE 4: FIBRINOLYTIC PATHWAY**




Fibrinolytic pathway—products of fibrinolysis

- Fibrin Degradation Products (FDPs)
- D-dimer
- Plasmin
- PAP complex

Conflicting results have been obtained concerning the fibrinolytic system's role in PE. Several studies have shown that both t-PA and PAI-1 increases in PE whencompared to

normotensive pregnancy. Since t-PA and PAI-1 are synthesized from endothelial cells, their increase in level would reflect endothelial dysfunction. PAI-1is an acute phase reactant and their levels increases with the vascular synthesis of their proteins in abnormal condition(18). However, other studies have revealed that there is a significant reduction or no difference in the PAI-1 when comparing PE women and normotensive pregnant subjects.

Siti et al., in his review article stated that in PE, there is higher maternal plasma concentration of PAI-1 compared with a normal pregnancy. In contrast, PAI-2 levels are significantly lower in PE compared with normal pregnancy with the PAI-1 to PAI-2 ratio significantly increased(19). The decrease in PAI-2 in PE may reflect placental dysfunction. Increased PAI-1 level are detected pre-clinically in patients who show early evidence of placental dysfunction as detected by bilateral notching on uterine artery Doppler studies and in whom PE with fetal growth restriction occurs in the second trimester.

Thamrin et al proposed that in preterm PE the level of t-PA is significantly elevated compared to normal pregnancy. Significantly elevated t-PA antigen and reduced activity levels ($p<0.05$) was seen in preterm PE compared to term preeclamptic women in labor. The reduced PAI-2 level in PE would suggest a prognostic marker for reduced placental function(18).

Lothar Heilman proposed a study with the main objective to find an association between coagulation variables and the onset of symptoms in PE demonstrates an up-regulation of the intravascular coagulation system in early cases of severe PE. In contrast, the level of plasminogen inhibitor activity, a parameter of the fibrinolytic capacity in pre eclamptic patients, showed no increase compared with healthy pregnant women, and the increased level of D-Dimer indicates an increased fibrinolysis after the fibrin formation(19).

The increase of the D-dimer concentration reflected the severity of disease, the activation of platelets, and the consumption of clotting parameters and is indicative of a substantial increase in fibrinolytic system activation. According to Kobayashi et al, high levels of D-dimer together with a significant decrease of AT III had a strong association with the termination of pregnancy.

Certain studies haven't found any significant increase in D-Di in PE women compared to normal pregnancy subjects. A meta-analysis has evaluated some publications that assessed D-Di by enzyme linked immune-sorbent assay (ELISA) to define its diagnostic value in PE. However they highlighted the need for more comprehensive studies throughout pregnancy including the establishment of an appropriate cut-off, to establish the diagnostic/prognostic role in PE.

**CLINICAL FEATURES OF PRE ECLAMPSIA**

Clinical features of preeclampsia are(14):

- Severe headache
- Visual disturbances
- Epigastric pain and/or vomiting
- Signs of clonus
- Papilloedema
- Liver tenderness
- Platelet count below 100 x 106/l
- Abnormal liver enzyme (ALT or AST rising to above 70 IU/L
- HELLP syndrome

**Criteria for Severe Preeclampsia**(20)

1. Blood pressure of 160 mmHg systolic or 110 mmHg diastolic, recorded on at least 2 occasions at least 6 hours apart with the patient on bed rest

2. Proteinuria of 5g in 24 hours

3. Oliguria , 500 mL in 24 hours

4. Cerebral visual disturbances

5. Epigastric pain, nausea and vomiting

6. Pulmonary oedema

7. Impaired liver function of unclear aetiology

8. Thrombocytopenia

9. Convulsions (eclampsia)

10. Fetal growth restriction

**FOETAL CONSEQUENCES**

As a result of impaired utero-placental blood flow secondary to failure of physiologic transformation of the spiral arteries or placental vascular insults, or both, manifestations of preeclampsia also may be seen in the feto–placental unit. Abnormalities in the placental bed and subsequent failure of physiologic transformation of the spiral arteries in the first or early second trimester limit the blood flow to the utero-placental unit. Additional mechanisms for chronic utero-placental ischemia include placental vascular insults. Among women with preeclampsia, clinical manifestations that follow from this utero-placental ischemia include foetal growth restriction, oligohydramnios, placental abruption, and non-reassuring foetal

status demonstrated on antepartum surveillance. Consequently, foetuses of women with preeclampsia are at increased risk of spontaneous or indicated preterm delivery(11).

## MANAGEMENT OF PRE ECLAMPSIA

The clinical findings of preeclampsia can manifest as either maternal syndrome alone (Hypertension and proteinuria, 0.3 g/24 hour-urine with or without other multisystem dysfunction) or in association with fetal syndrome (fetal growth restriction, oligohydramnios). Appearance of proteinuria remains an important diagnostic criterion to differentiate from gestational hypertension. Proteinuria is defined as a 24-hour urinary protein excretion exceeding 300 mg, a urine protein:creatinine ratio of . 0.3, or persistent 30 mg/dL (11 on dipstick) protein in random urine samples.

## CLINICAL DIAGNOSIS

### Blood Pressure Elevation

Persistent elevation in blood pressure is the hallmark of preeclampsia and reflects the severity of the disease. One common error is taking the blood pressure in an obese patient with regular size cuff. This causes abnormally high readings. Another common error is not using the same maternal position in repeated measurements. This is an inappropriate technique because in the pregnant woman the lateral recumbent values are always lower than those in the sitting position, and to ignore the initial high blood pressure value will delay proper diagnosis and treatment. To avoid these errors, the blood pressure at each pregnant visit should be taken with the patient in the sitting position. A third error is the use of different end points to measure the diastolic blood pressure. The official recommendation of the NHBPEP and the ACOG is to use the Korotkoff V sound, the point of disappearance of the sound, as the marker of diastolic pressure.

**Proteinuria**

Proteinuria is a sign of preeclampsia which is defined as 300 mg of protein in a 24-hour urine collection. This usually correlates with 30 mg/dL or a 11 reading dipstick in a random urine specimen. Proteinuria is also valuable as a sign of severity and a value □ 5 g in 24 hours is one of the criteria to classify preeclampsia as severe. The 24-hour urine collection for protein is the gold standard in the diagnosis of preeclampsia. The dipstick that has a good, although not perfect, correlation with the protein concentration in the urine. Urine dipsticks can be affected by variable excretion, maternal dehydration and bacteriuria. The correlation of the dipstick with the 24 hours excretion of protein was studied by Meyer et al. A 11 dipstick has a 92% positive predictive value to predict 300 mg of protein. Approximate equivalence is 11 5 0.3g/l, 2 1 5 1g/l, 31 5 3g/l.

A second rapid method for evaluation of proteinuria is the protein/creatinine ratio that in the non-pregnant state, correlates well with the 24-hour collection. A protein/creatinine ratio of 0.3 has a positive predictive value of 85.5% and a sensitivity of 81.0% for significant proteinuria in the 24-hour collection. Unfortunately, a negative result has a negative predictive value of 47.5%, meaning that about half of the women with a negative result will have significant proteinuria in the 24-hour collection specimen.

The definitive test for diagnosing proteinuria is a quantitative measurement of total protein excretion in 24-hour urine sample. Significant proteinuria is diagnosed if the urinary protein:creatinine ratio is greater than 30 mg/mmol or a validated 24-hour urine collection result shows greater than 300 mg protein. Proteinuria in preeclampsia characteristically occurs in the absence of either a nephritic (red cells, red cells casts) or a nephrotic (birefringent lipids, wax casts) urinary sediment. The urinary sediment in preeclampsia is usually unrevealing and in most cases shows an abundance of fine and coarse granular casts.

**Excessive Weight Gain and Edema**

Excessive weight gain and edema are no longer considered a diagnostic criteria for preeclampsia as both are commonly seen in normal pregnancies with no increase in the incidence of preeclampsia. There is no evidence to indicate that measures limiting weight gain during pregnancy, such as the use of low salt diet or diuretics, prevent the development of preeclampsia.

## TREATMENT WITH ANTIHYPERTENSIVES

The objectives of treating severe hypertension are to prevent congestive heart failure, myocardial ischemia, renal injury or failure, and ischemic or hemorrhagic stroke. Antihypertensive treatment should be initiated expeditiously for acute-onset severe hypertension (systolic blood pressure of 160 mm Hg or more or diastolic blood pressure of 110 mm Hg or more, or both) that is confirmed as persistent (15 minutes or more). The available literature suggests that antihypertensive agents should be administered within 30–60 minutes. However, it is recommended to administer antihypertensive therapy as soon as reasonably possible after the criteria for acute-onset severe hypertension are met. Intravenous hydralazine or labetalol and oral nifedipine are the three agents most commonly used for this purpose. A recent Cochrane systematic review that involved 3,573 women found no significant differences regarding either efficacy or safety between hydralazine and labetalol or between hydralazine and calcium channel blockers(21). Thus, any of these agents can be used to treat acute severe hypertension in pregnancy. Although parenteral antihypertensive therapy may be needed initially for acute control of blood pressure, oral medications can be used as expectant management is continued. Oral labetalol and calcium channel blockers have been commonly used. One approach is to begin an initial regimen of labetalol at 200 mg orally every 12 hours and increase the dose up to 800 mg orally every 8–12 hours as needed

(maximum total 2,400 mg/d). If the maximum dose is inadequate to achieve the desired blood pressure goal, or the dosage is limited by adverse effect, then short-acting oral nifedipine can be added gradually.

***TABLE 2:ANTIHYPERTENSIVES USED FOR CONTROL OF BLOOD PRESSURE DURING PREGNANCY*** (11)

| Drug | Dose | Comments | Onset of Action |
|---|---|---|---|
| Labetalol | 10–20 mg IV, then 20–80 mg every 10–30 minutes to a maximum cumulative dosage of 300 mg; or constant infusion 1–2 mg/min IV | Tachycardia is less common with fewer adverse effects.<br><br>Avoid in women with asthma, preexisting myocardial disease, decompensated cardiac function, and heart block and bradycardia. | 1–2 minutes |
| Hydralazine | 5 mg IV or IM, then 5–10 mg IV every 20–40 minutes to a maximum cumulative dosage of 20 mg; or constant infusion of 0.5–10 mg/hr | Higher or frequent dosage associated with maternal hypotension, headaches, and abnormal fetal heart rate tracings; may be more common than other agents. | 10–20 minutes |
| Nifedipine (immediate release) | 10–20 mg orally, repeat in 20 minutes if needed; then 10–20 mg every 2–6 hours; maximum daily dose is 180 mg | May observe reflex tachycardia and headaches | 5–10 minutes |

Abbreviations: IM, intramuscularly; IV, intravenously.

**TABLE 3: NICE GUIDELINES 2020**(22)

| Degree of hypertension | Mild (140/90 mmHg to 149/99 mmHg) | Moderate (150/100 mmHg to 159/109 mmHg) | Severe (160/110 mmHg or higher) |
|---|---|---|---|
| Admit to hospital | Yes | Yes | Yes |
| Treat | No | With oral labetalol as first-line treatment | With oral labetalol as first-line treatment |
| Measure blood pressure | At least 4 times a day | At least 4 times a day | More than 4 times a day |
| Test for proteinuria | Do not repeat quantification of proteinuria | Do not repeat quantification of proteinuria | Do not repeat quantification of proteinuria |
| Blood tests | Monitor the following twice a week: kidney function, bilirubin, electrolytes, full blood count, transaminases | Monitor the following 3 times a week: kidney function, bilirubin, electrolytes, full blood count, transaminases | Monitor the following 3 times a week: kidney function, bilirubin, electrolytes, full blood count, transaminases |
| Fetal assessment | Offer fetal heart auscultation at every antenatal appointment<br>Carry out ultrasound assessment of the fetus at diagnosis and, if normal, repeat every 2 weeks<br>Carry out a CTG at diagnosis and then only if clinically indicated<br>(See section 1.6 for advice on fetal monitoring) | Offer fetal heart auscultation at every antenatal appointment<br>Carry out ultrasound assessment of the fetus at diagnosis and, if normal, repeat every 2 weeks<br>Carry out a CTG at diagnosis and then only if clinically indicated<br>(See section 1.6 for advice on fetal monitoring) | |

[a] Use an automated reagent-strip reading device for dipstick screening for proteinuria in a secondary care setting.

Abbreviations: BP, blood pressure; CTG, cardiotocography.

| Weeks of pregnancy | Timing of birth |
|---|---|
| Before 34 weeks | Continue surveillance unless there are indications (see recommendation 1.5.7) for planned early birth. Offer intravenous magnesium sulfate and a course of antenatal corticosteroids in line with the NICE guideline on preterm labour and birth. |
| From 34 to $36^{+6}$ weeks | Continue surveillance unless there are indications (see recommendation 1.5.7) for planned early birth.<br>When considering the option of planned early birth, take into account the woman's and baby's condition, risk factors (such as maternal comorbidities, multi-fetal pregnancy) and availability of neonatal unit beds. Consider a course of antenatal corticosteroids in line with the NICE guideline on preterm labour and birth. |
| 37 weeks onwards | Initiate birth within 24–48 hours. |

**TIMING OF DELIVERY**(22)

•Record maternal and fetal thresholds for planned early birth before 37 weeks in women with pre-eclampsia.

• Thresholds for considering planned early birth could include (but are not limited to) any of the following known features of severe pre-eclampsia:

•inability to control maternal blood pressure despite using 3 or more classes of antihypertensives in appropriate doses

•maternal pulse oximetry less than 90%

•progressive deterioration in liver function, renal function, hemolysis, or platelet count

• ongoing neurological features, such as severe intractable headache, repeated visual disturbances , eclampsia placental abruption, reversed end-diastolic flow, abnormal CTG or stillbirth.

•Involve a senior obstetrician in any decisions on timing of birth for women with pre-eclampsia.

•Discuss with the anaesthetic team if birth is planned in a woman with pre-eclampsia.

•Discuss with the neonatal team if birth is planned in a woman with pre-eclampsia, and neonatal complications are anticipated.

•Offer intravenous magnesium sulphate and a course of antenatal corticosteroids if indicated, if early birth is planned for women with preterm pre-eclampsia, in line with the NICE guideline on preterm labour and birth.

## INTRAPARTUM MANAGEMENT

The management of preclamptic women during labour is aimed at

1. Prevention of seizures
2. Control of hypertension

## SEIZURE PROPHYLAXIS

Magnesium sulphate is more effective than phenytoin, diazepam, or nimodipine (a calcium-channel blocker used in clinical neurology to reduce cerebral vasospasm) in reducing eclampsia and should be considered the drug of choice in the prevention of eclampsia in the intrapartum and postpartum periods(23). Benzodiazepines and phenytoin are justified only in the context of antiepileptic treatment or when magnesium sulphate is contraindicated or unavailable (myasthenia gravis, hypocalcemia, moderate-to-severe renal failure, cardiac ischemia, heart block, or myocarditis)(24).

GENERAL MANAGEMENT

➢ Patient NEVER LEFT ALONE

➢ Initially manage Airway, Breathing and Circulation

➢ Prevention of injuries (cot with railings )

➢ Maintain Cardiorespiratory function (oral airway tube )

➢ Lateral decubitus position

➢ Suction (vomit and oral secretion)

➢ Oxygen administration

➢ Vitals monitoring

➢ Catheter insertion

CONTROL OF CONVULSIONS

***PRITCHARD REGIMEN***

Magnesium sulphate

➢ Loading dose :

4g of 20% magnesium sulphate solution IV ( 15 minutes)

10g of 50% magnesium sulphate solution IM ( 5g each buttock)

If Recur - 2g of 20% magnesium sulphate solution IV ( 5 minutes)

➢ Maintenance dose:

Every 4 hours, 5g 50% magnesium sulphate solution IM (5g each buttock)

Continued for 24 hours after delivery or last convulsion.

OTHER REGIMENS:

***Continuous IV regimen :***

Loading dose : 6g MgSo4 in 100ml NS IV slowly over 15-20 minutes

Maintenance dose : 1-2g/hr in 100 ml IV

***Zuspan regimen***:

Loading dose : IV dose of 4g MgSo4 initially given slowly over 5 - 10 minutes

Maintenance dose : 1 g / hour MgSo4 continuous IV

***Sibai regimen :***

6g MgSo4 IV over 20 minutes followed by 2g MgSo4 IV

**POSTPARTUM MANAGEMENT**

•In the early post-partum period, women with pre-eclampsia should be considered at high risk for pre-eclamptic complications for at least 3 days and should have their clinical condition monitored at least every four hours while awake.

• Antihypertensives should be continued, and consideration should be given to treating any hypertension before D6 postpartum with antihypertensive therapy. Thereafter, antihypertensives may be withdrawn slowly over days, but not ceased abruptly.

•It is important to note that eclamptic seizures may develop for the first time in the early post-partum period.

•Avoid Non-steroidal anti-inflammatory drugs (NSAIDs) for postpartum analgesia in pre-eclampsia and AKI.

## MANAGEMENT OF HELLP SYNDROME

The clinical course of HELLP syndrome often is characterized by progressive and sometimes sudden deterioration in maternal and fetal condition. Considering the serious nature of this entity, with increased rates of maternal morbidity and mortality, many authors have concluded that women with HELLP syndrome should be delivered regardless of their gestational age. Because the management of patients with HELLP syndrome requires the availability of neonatal and obstetric intensive care units and personnel with special expertise, patients with HELLP syndrome who are remote from term should receive care at a tertiary care center.

It has been hypothesized that the anti-inflammatory and immunosuppressive effects of corticosteroids may modify some of the pro-inflammatory features of preeclampsia with severe features and favourably affects the clinical course. Several randomized controlled trials of high-dose corticosteroid treatment for antepartum or postpartum stabilization of HELLP syndrome have been conducted. The use of corticoids in the management of HELLP syndrome compared with placebo or no treatment was reviewed in a Cochrane Database Systematic Review, which included 11 randomized trials (550 women). There was no difference in the risk of maternal death, severe maternal morbidity, or perinatal or infant death. The only effect of treatment on individual outcomes was improved platelet count (standardized mean difference [SMD] 0.67; 95% CI, 0.24−1.10)(25). The authors concluded that the evidence is insufficient to support the use of corticosteroids for attenuation of the disease process in HELLP syndrome.

Very close monitoring is required in HELLP syndrome until delivery and in the postpartum period, with laboratory testing at least at 12-hour intervals. Aspartate aminotransferase levels more than 2,000 IU/L or LDH more than 3,000 IU/L suggest an

increased mortality risk. In the natural history of HELLP syndrome there is an inverse relationship between the trends in platelet values and liver enzymes level. During the aggravation slope in the disease evolution, platelet count usually decreases at an average rate of approximately 40% per day, whereas the liver enzymes values tend to increase. The lowest observed platelet count occurs at a mean of 23 hours after delivery. The disease may achieve peak intensity during the first 2 days after delivery, including a downward trend in hematocrit. If the platelet count continues to drop and liver enzymes to increase after 4 days postpartum, the validity of the initial diagnosis of HELLP syndrome should be reassessed. With supportive care alone, 90% of patients with HELLP syndrome will have platelet count more than 1 lakh/cu.mm and reversed trend (decrease) in liver enzymes values within 7 days after delivery(26). Women with HELLP syndrome are also at increased risk of pulmonary edema, acute respiratory distress syndrome and renal failure.

## MANAGEMENT OF DIC

Blood transfusion is recognised as one of the eight essential components of the Comprehensive Emergency Obstetric Care module, which has been designed to reduce maternal mortality rates. Key to the treatment of DIC is the specific and vigorous treatment of the underlying disorder. The blood component therapy should not be instituted on the basis of laboratory results alone, but is indicated in patients with active bleeding, in those requiring an invasive procedure and those who are otherwise at risk for bleeding complications.

***RCOG Guideline on obstetric emergency transfusion***(27)

- Blood transfusion is almost always required when the Hemoglobin is less than 60 g/l and it is rarely required when the Hemoglobin is greater than 100 g/l.
- Clinical evaluation of the patient in this situation is extremely important in bleeding patients since patients with acute hemorrhage can have normal Hemoglobin.
- Maintain PT and APTT ratios at less than 1.5 x normal.
- It recommends transfusion of FFP at a dose of 12–15 ml/kg should be administered for every 6 units of red cells during a major obstetric bleed**.**
- It is essential that regular full blood counts and coagulation screens (PT, APTT and fibrinogen) are performed during the bleeding episode.
- Cryoprecipitate at a standard dose of two 5-unit pools should be administered early in major obstetric hemorrhage. Subsequent cryoprecipitate transfusion should be guided by fibrinogen results, aiming to keep levels above 1.5 g/l.

# *MATERIALS AND METHODS*

# MATERIALS AND METHODS

## STUDY DESIGN

Prospective observational study

## STUDY PLACE

Department of Obstetrics and Gynecology, Government R.S.R.M Lying in Hospital, Stanley Medical College.

## STUDY PERIOD

October 2020 to September 2021 (1 year)

## STUDY SAMPLE

70

**INCLUSION CRITERIA**

- Age >18 years
- Patients who give consent for undergoing the blood investigation
- Gestational age > 20 weeks to 40+3 weeks
- Single pregnancy with a living foetus
- Diagnosis of pre-eclampsia at hospitalization according to ACOG criteria (2013).

**EXCLUSION CRITERIA**

- Twin pregnancy
- Use of anticoagulants during pregnancy
- Systemic lupus erythematosus
- Foetal death
- Smoking
- diseases affecting organs affected by preeclampsia
- Chronic renal failure
- Autoimmune hepatitis
- Intrahepatic cholestasis

# METHODOLOGY

The study group were women with pre eclampsia who are admitted at Government RSRM lying in hospital. They should meet one or more criterion of pre eclampsia at hospital admission or during hospitalization with BP more than 140/90mmHg plus proteinuria with or without organ damage. They are explained regarding the nature of the study and informed consent is obtained in their own language. Patients are selected according to the inclusion criteria.

A detailed history is taken and their routine investigation findings and ultrasound findings are noted. Complete information of the patients which include name, age, weight, blood pressure measurement, urine analysis, treatment history are obtained from outpatient and inpatient records.

The coagulation parameters that are taken into consideration are Prothrombin time (PT), activated Partial Thromboplastin time (aPTT), Thrombin time (TT), serum fibrinogen and D-Dimer along with complete blood count (CBC).

## SAMPLE COLLECTION

After getting written informed consent from the patients, 2.7ml blood sample was collected in a vacuum tube containing sodium citrate (0.3ml of 3.2% sodium citrate) in a (9:1) volume ratio. The tubes were mixed by inverting the tubes 3-4 times immediately after the blood drawn. The samples were sent to the laboratory and tests were performed in 4 hours of sample collection. The testing was done by immunogenic turbidimetry assay (800nm).

Levels of D dimer at the time of hospitalization or during the time of diagnosis of pre eclampsia are measured by immunoturbidimetry, taking as reference the levels for a normal pregnant population adjusted for gestational age. D dimer test is done only once during admission or during the first diagnosis of pre eclampsia. Samples for D dimer analysis are

collected in a standard tube for coagulation testing. These samples are not subjected to freezing process. It is immediately processed in a clinical laboratory and the results are obtained.

The severity of the disease is followed up until delivery or termination of pregnancy. During this Period of study, the factors for severity of preeclampsia followed up include:

- Severe hypertension systolic BP of 160mmHg o higher, or diastolic BP of 110 mmHg or higher
- Thrombocytopenia (platelet count less than 1,00,000/cu.mm
- Impaired liver function as indicated by abnormally elevated blood concentrations of liver enzymes (to twice the upper limit of normal concentration)
- Severe persistent right upper quadrant or epigastric pain and not accounted for by alternative diagnosis
- Renal insufficiency (serum creatinine concentration greater than 1.1 mg/dL or a doubling of the serum creatinine concentration in the absence of other renal disease)
- Pulmonary edema
- New onset headache unresponsive to acetaminophen and not accounted for by alternative diagnosis or with visual disturbances
- Eclampsia

# *RESULTS AND ANALYSIS*

## STATISTICAL ANALYSIS

A minimum of 70 Preeclampsia patients of gestational age more than 20 weeks admitted at Government RSRM lying in hospital are included in this study. After collecting, the Data will be compiled and entered in Microsoft Excel Sheet. Analysis will be done using Statistical software SPSS version 16.All Continuous variables will be expressed as Mean and Standard Deviation .

All Categorical variables will be expressed as Percentages and Proportions. The test will be considered Significant if $P<0.05$, at 95% Confidence Interval. Chi square test will be used as test of significance.

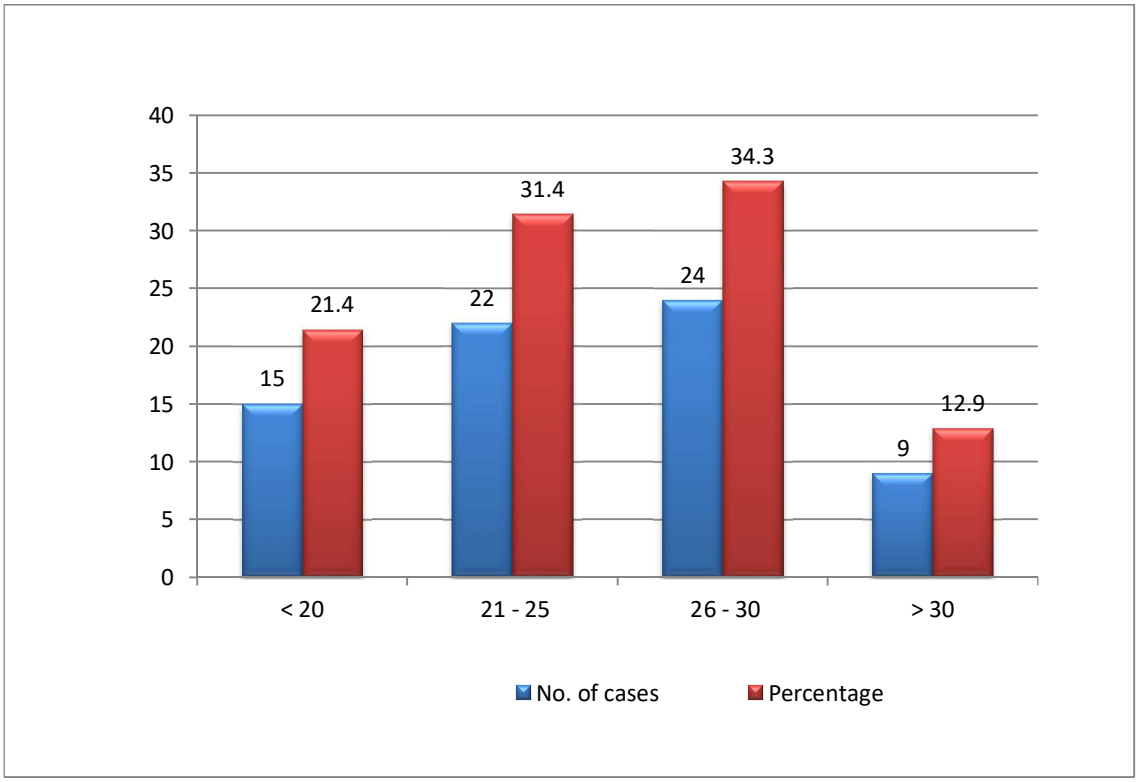
## RESULTS AND ANALYSIS

### TABLE NO.4: AGE DISTRIBUTION OF STUDY PARTICIPANTS

The age group of the subjects was between 18-35 years. The mean age and standard deviation in preeclamptic patients was 25.414±4.617 years.

| Age | No. of cases | Percentage |
|---|---|---|
| 18-20 | 15 | 21.4% |
| 21 – 25 | 22 | 31.4% |
| 26 – 30 | 24 | 34.3% |
| 31-35 | 9 | 12.9% |
| Total | 70 | 100.0% |
| Mean | 25.414 | |
| SD | 4.617 | |

**FIGURE 5: AGE DISTRIBUION IN STUDY PARTICIPANTS**




The findings show that the incidence of pre eclampsia is more among women in age group 26-30 years.

**FIGURE 6 : PARITY DISTRIBUTION**

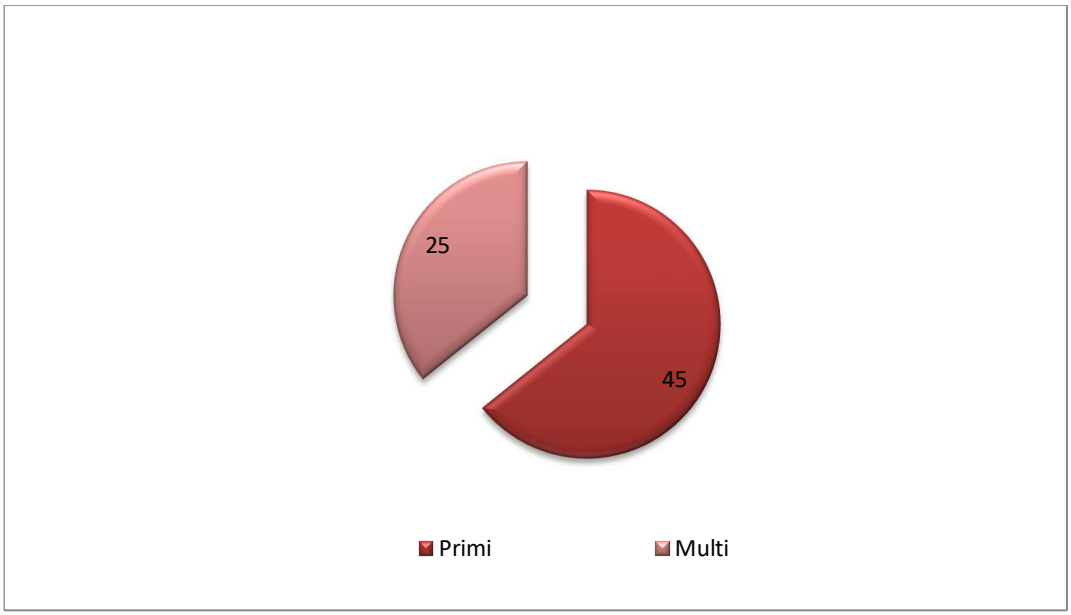



**TABLE 5 : PARITY DISTIBUTION AMONG STUDY GROUP**

| Gravida | No. of Case | Percentage |
|---|---|---|
| Primigravida | 45 | 64.3% |
| Multigravida | 25 | 35.7% |
| Total | 70 | 100.0% |

Among the preeclamptic cases the number of primigravida was 45 and multigravida was 25.

The findings show that the incidence of pre eclampsia is higher in primigravida when compared to multigravida.

**TABLE 6 : BMI STATUS OF THE STUDY GROUP**

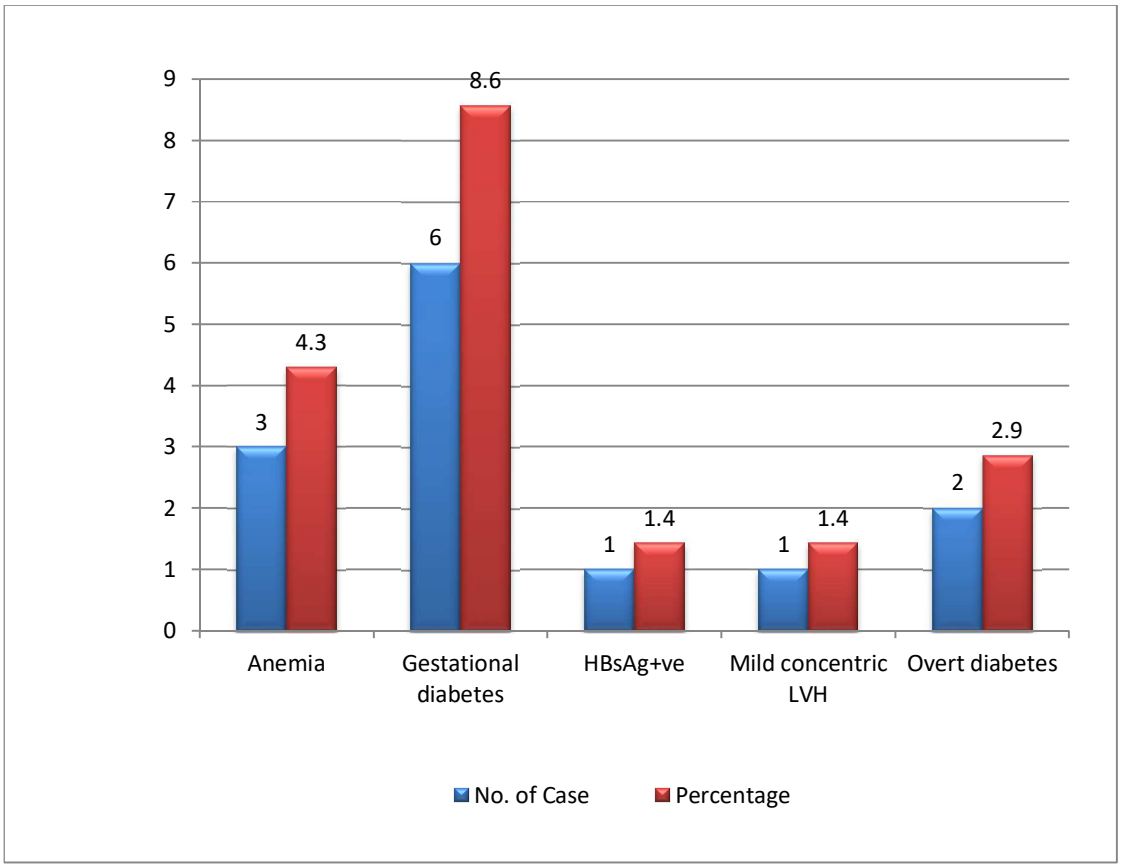
| BMI | No. of Case | Percentage |
|---|---|---|
| < 25 | 11 | 15.7 |
| 26 - 30 | 31 | 44.3 |
| > 30 | 28 | 40.0 |
| Total | 70 | 100.0 |
| Mean | 30.514 | |
| SD | 5.605 | |

The number of obese pre eclamptic women was 28(40%) whose Body mass index were more than 30. Overweight women whose body mass index is 26-30 among the study group were 31 (44.3%).

**TABLE 7: ASSOCIATED COMORBIDITIES AMONG STUDY GROUP**

| COMORBIDS | No. of Case | Percentage |
|---|---|---|
| Anemia | 3 | 4.3 |
| Gestational diabetes | 6 | 8.6 |
| HBsAg+ve | 1 | 1.4 |
| Mild concentric LVH | 1 | 1.4 |
| Overt diabetes | 2 | 2.9 |
| Nil | 57 | 81.4 |
| Total | 70 | 100.0 |

**FIGURE 7 : ASSOCIATED COMORBIDITIES AMONG STUDY GROUP**

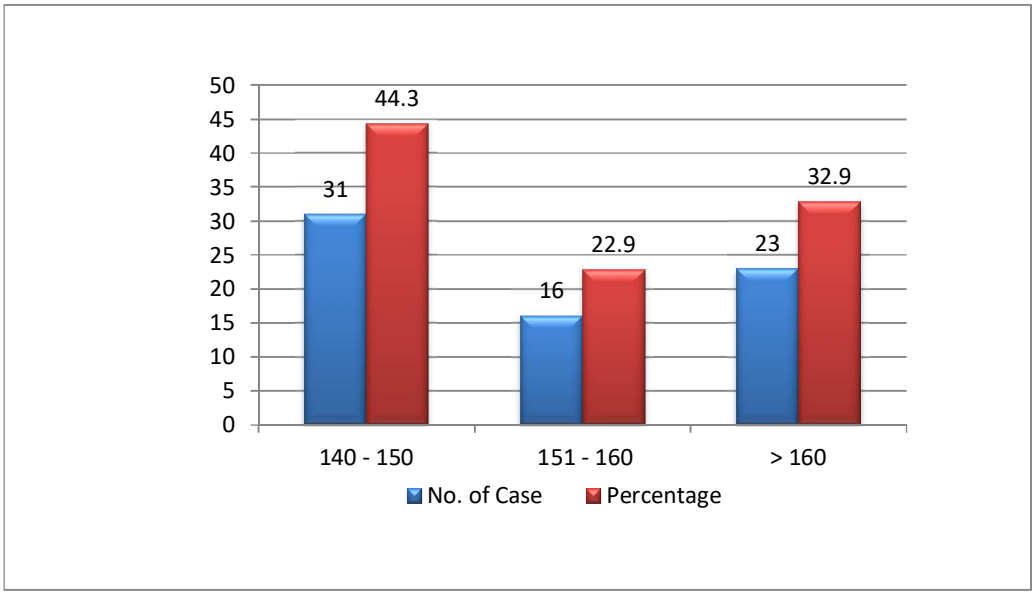



57 cases were with no added comorbid conditions. Among 13 cases, 6 had gestational diabetes. 3 had anemia.

**TABLE 8: SYSTOLIC BLOOD PRESSURE VARIATION AMONG PRE ECLAMPSIA CASES**

| SYSTOLIC BP | No. of Case | Percentage |
|---|---|---|
| 140 - 149 | 31 | 44.3 |
| 150 - 159 | 16 | 22.8 |
| > 160 | 23 | 32.9 |
| Total | 70 | 100.0 |
| Mean | 157.586 | |
| SD | 13.319 | |

**FIGURE 8: SYSTOLIC BLOOD PRESSURE VARIATION AMONG PRE ECLAMPSIA CASES**

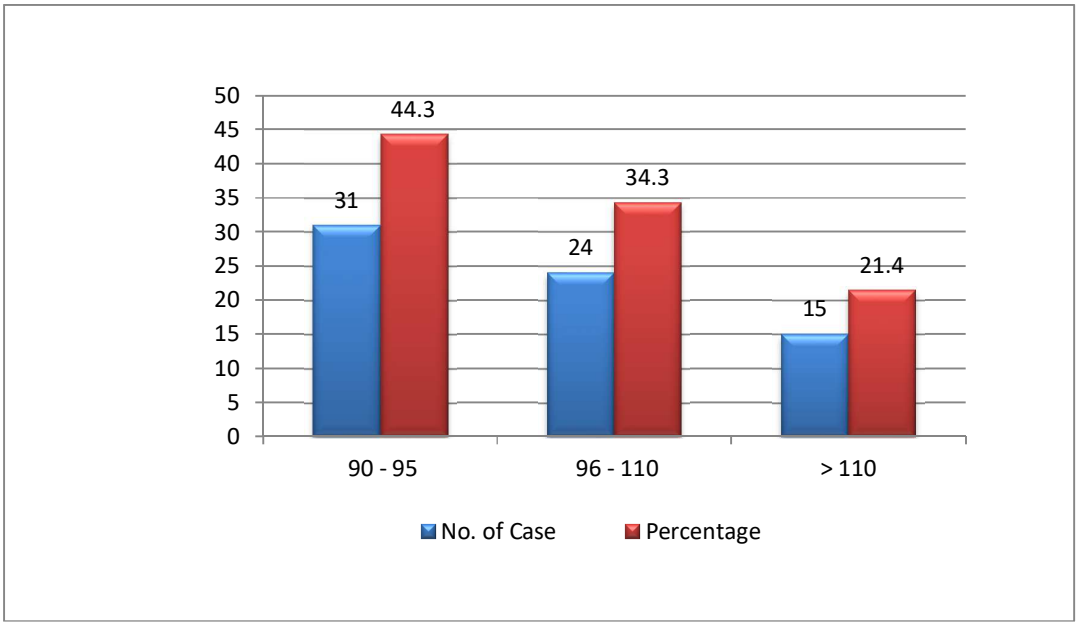



The mean systolic blood pressure among study group is 158±13. 44.3% cases had systolic blood pressure in the range of 140-149mmHg and 32.9% cases with blood pressure >160mmHg.

**TABLE 9: DIASTOLIC BLOOD PRESSURE VARIATION AMONG PRE ECLAMPSIA CASES**

| DIASTOLIC BP | No. of Case | Percentage |
|---|---|---|
| 90 – 95 | 31 | 44.3 |
| 96 – 110 | 24 | 34.3 |
| > 110 | 15 | 21.4 |
| Total | 70 | 100.0 |
| Mean | 101.114 | |
| SD | 10.995 | |

**FIGURE 9 :DIASTOLIC BLOOD PRESSURE VARIATION AMONG PRE ECLAMPSIA CASES**

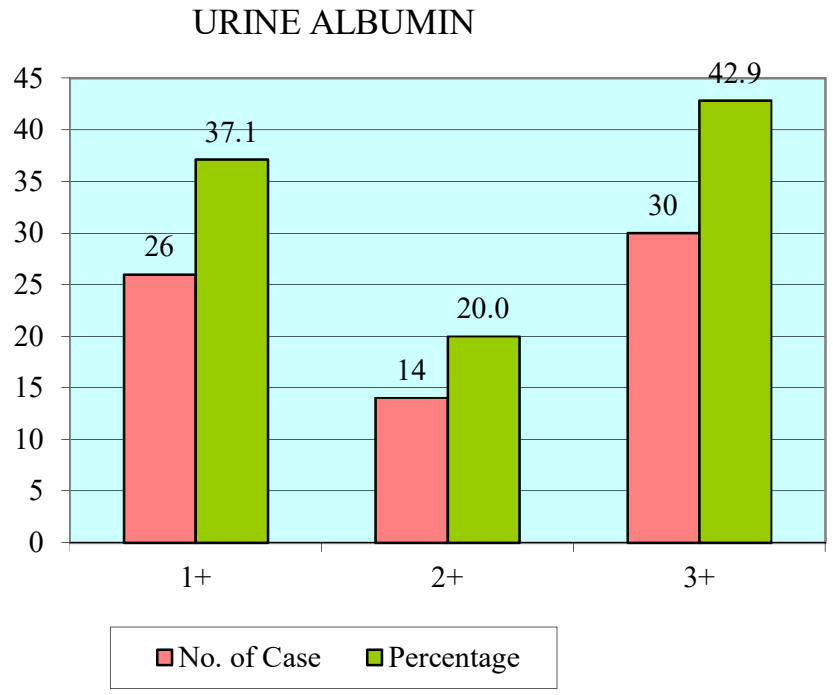



The mean diastolic blood pressure among the study group is 101±11 mmHg. 44% had diastolic blood pressure in the range of 90-95mmHg whereas 21.4% had diastolic blood pressure >110mmHg.

**TABLE 10 : URINE ALBUMIN ANALYSIS AMONG STUDY GROUP**

| URINE ALBUMIN | No. of Case | Percentage |
|---|---|---|
| 1+ | 26 | 37.1 |
| 2+ | 14 | 20.0 |
| 3+ | 30 | 42.9 |
| Total | 70 | 100.0 |

**FIGURE 10 :URINE ALBUMIN ANALYSIS AMONG STUDY GROUP**

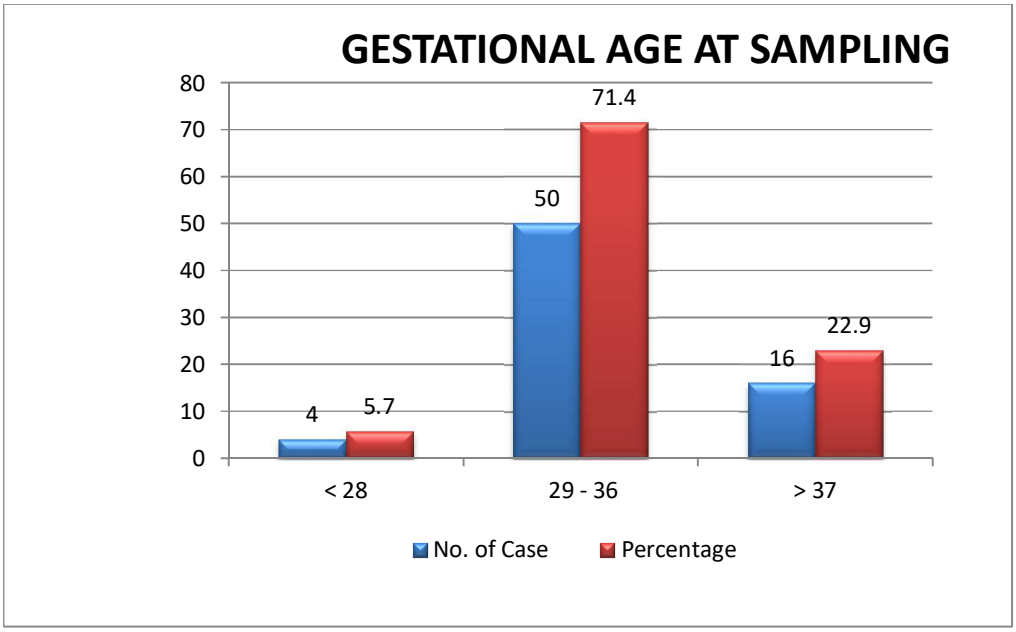



Proteinuria was observed in all cases. 37.1% had urine albumin 1+ which is 0.3mg/dL; 20% had urine albumin 2+ which is 1g/dL; 42.9% with urine albumin 3+ which is 3g/dL.

**TABLE 11:GESTATIONAL AGE AT WHICH SAMPLING DONE**

| GESTATIONAL AGE AT SAMPLING | No. of Case | Percentage |
|---|---|---|
| < 28 | 4 | 5.7 |
| 29 - 36 | 50 | 71.4 |
| > 37 | 16 | 22.9 |
| Total | 70 | 100.0 |
| Mean | 34.671 | 49.5 |
| SD | 3.713 | 5.3 |

**FIGURE 11:GESTATIONAL AGE AT WHICH SAMPLING DONE**

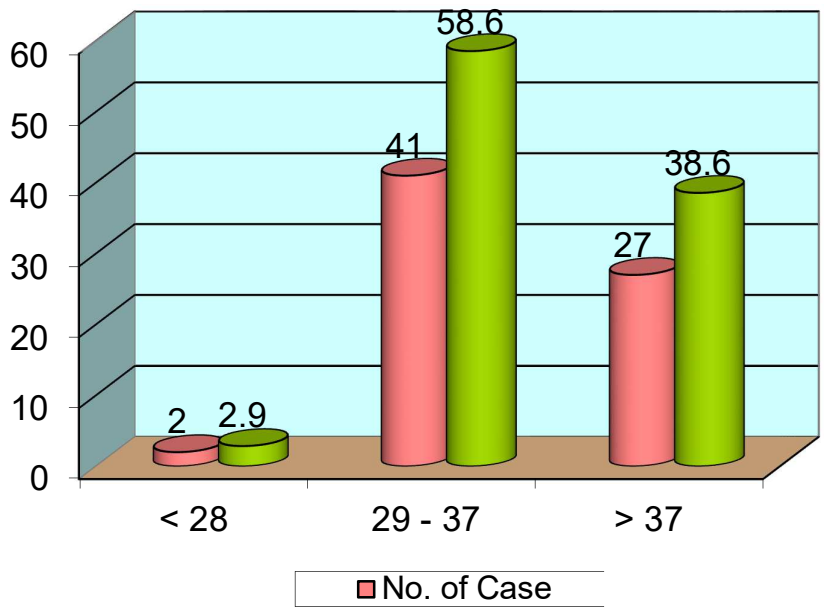



Among 70 cases, 4 cases (5.7%) were diagnosed to be pre eclampsia at less than 28 weeks of gestation and testing was done at this time. 50 cases (71.4%) were diagnosed and tested at 29-36 weeks of gestation. Only 16 cases (22.9%) were diagnosed as pre eclampsia at term(>37 weeks) and testing done at this time.

**TABLE 12:GESTATIONAL AGE AT DELIVERY**

| GESTATIONAL AGE AT DELIVERY | No. of Case | Percentage |
|---|---|---|
| < 28 | 2 | 2.9 |
| 29 - 37 | 41 | 58.6 |
| > 37 | 27 | 38.6 |
| Total | 70 | 100.0 |
| Mean | 36.257 | |
| SD | 3.278 | |

**FIGURE 12:GESTATIONAL AGE AT DELIVERY**

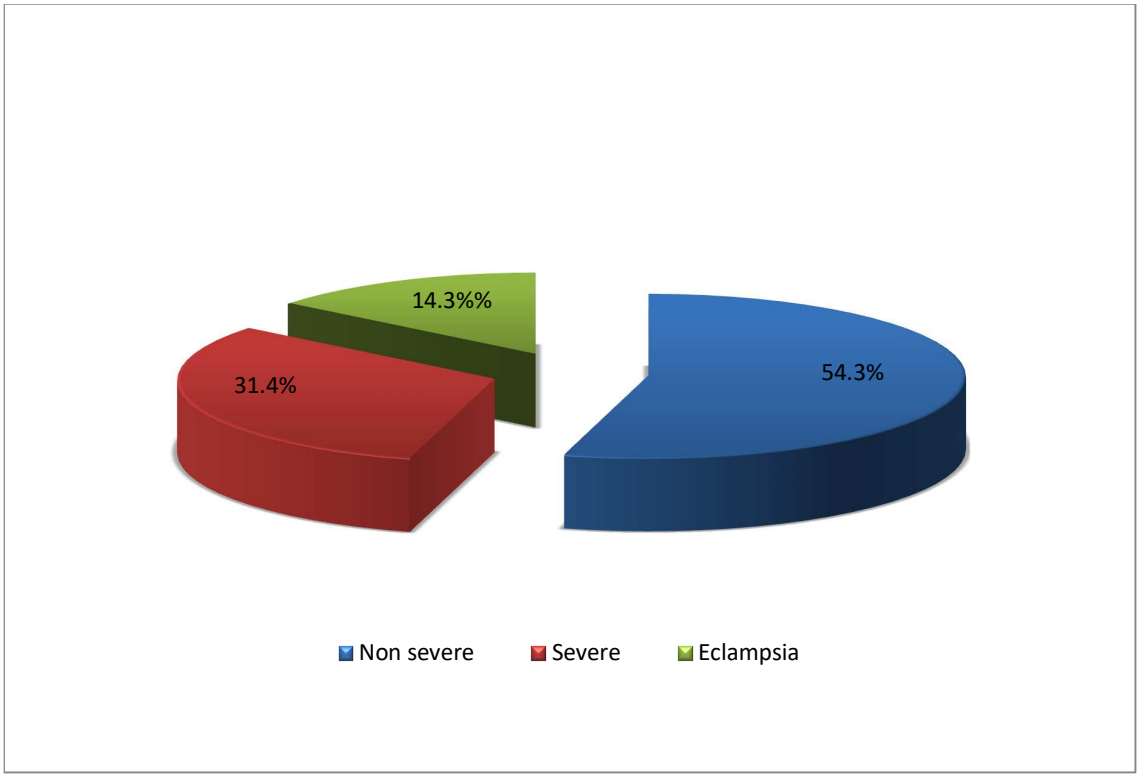



Out of 4 cases diagnosed as pre eclampsia less than 28 weeks, 2 cases delivered before 28 weeks. The mean gestational age of delivery of pre eclampsia patients among the study group is 36.257±3.278. 41 cases delivered between 29 to 37 weeks.

**TABLE 13:DISTRIBUTION OF NON SEVERE, SEVERE PRE ECLAMPSIA AND ECLAMPSIA CASES**

| SEVERE/NON SEVERE PRE ECLAMPSIA / ECLAMPSIA | No. of Case | Percentage |
|---|---|---|
| Non severe | 38 | 54.3 |
| Severe | 22 | 31.4 |
| Eclampsia | 10 | 14.3 |
| Total | 70 | 100.0 |

**FIGURE 13:DISTRIBUTION OF NON-SEVERE, SEVERE PRE ECLAMPSIA AND ECLAMPSIA CASES**

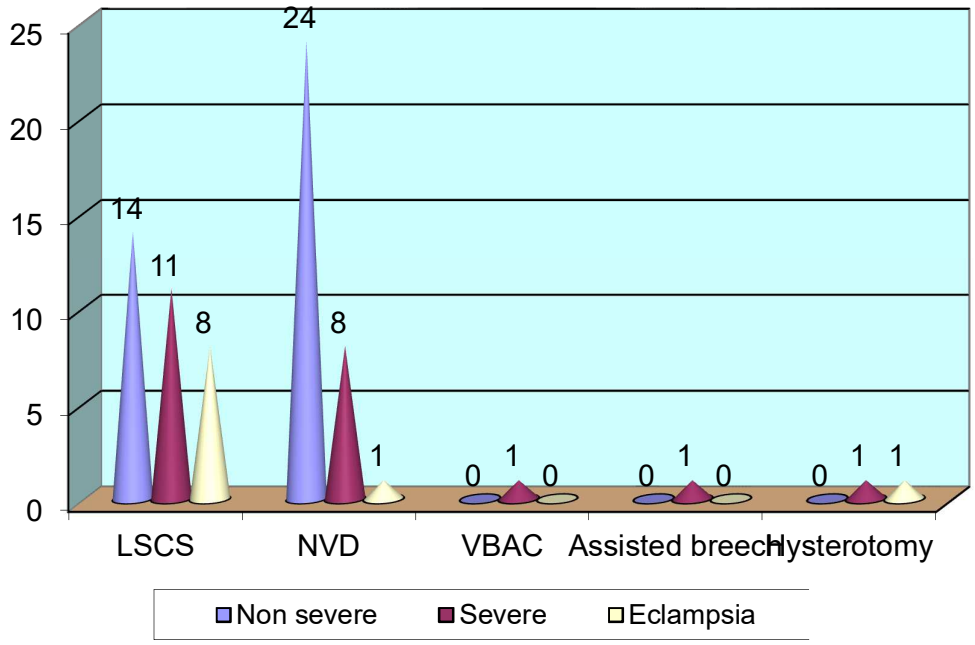



Out of 70 patients, 38 patients had non-severe pre eclampsia, 22 patients had severe eclampsia and 10 patients developed eclampsia (both antepartum and postpartum eclampsia.

**TABLE 14:DISTRIBUTION OF MODE OF DELIVERY**

| LSCS | NVD | VBAC | assisted breech | Hysterotomy | Total |
|---|---|---|---|---|---|
| 33 | 33 | 1 | 1 | 2 | 70 |
| 47.1% | 47.1% | 1.4% | 1.4% | 3.0% | 100% |

**FIGURE 14:DISTRIBUTION OF MODE OF DELIVERY**

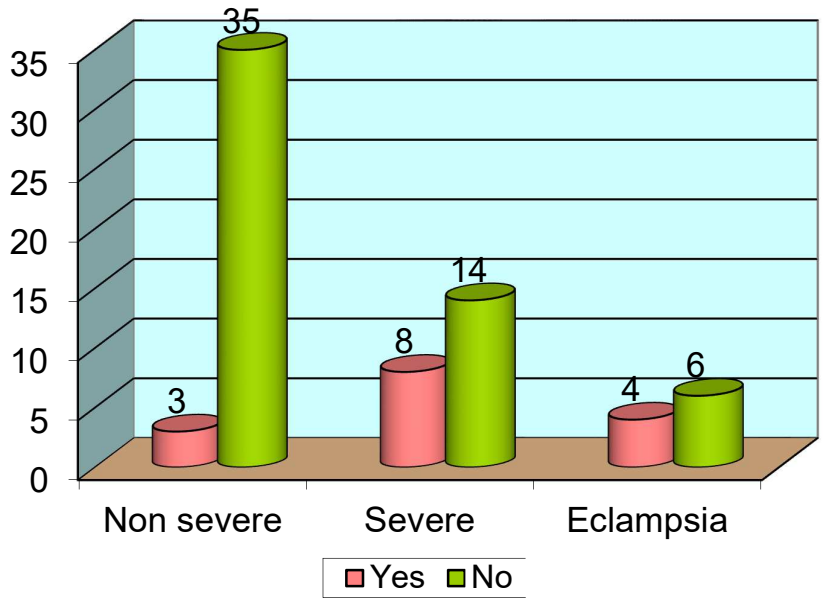



47.1% had delivery by cesarean section; 47.1% by normal vaginal delivery; 3% had hysterotomy; 1% by VBAC and 1% by assisted breech delivery.

**TABLE 15:DELIVERY OUTCOME IN PRE ECLAMPTIC MOTHERS**

| DELIVERY OUTCOME | NO. OF CASES | PERCENTAGE |
|---|---|---|
| PRETERM DELIVERIES | 15 | 21.4% |
| TERM DELIVERIES | 55 | 78.6% |
| TOTAL | 70 | 100% |

**FIGURE 15:DELIVERY OUTCOME IN PRE ECLAMPTIC MOTHERS**

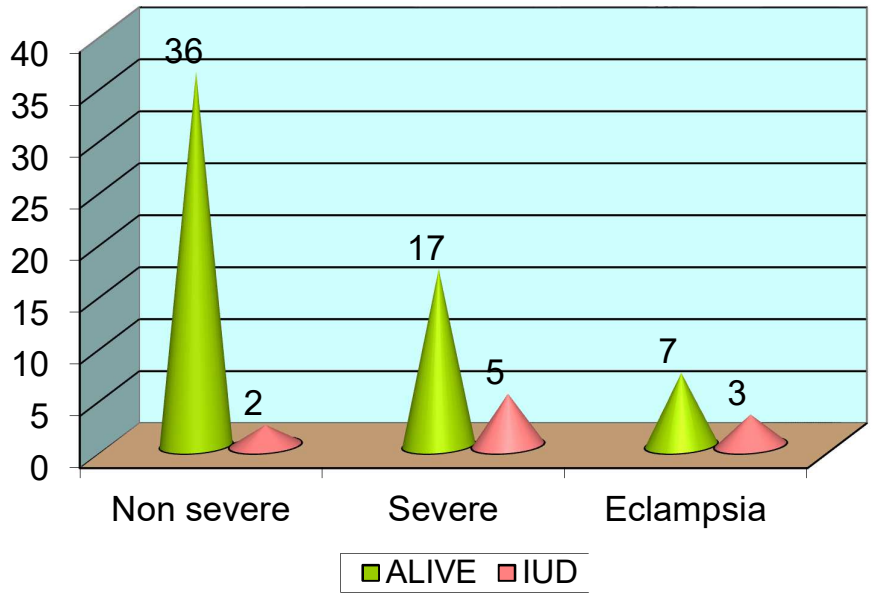



78.6%(55) had term deliveries and 21.4%(15) had preterm deliveries among pre eclamptic participants.

**TABLE 16:FOETAL OUTCOME IN STUDY PARTICIPANTS**

| OUTCOME | NO. OF CASES | FREQUENCY |
|---|---|---|
| ALIVE | 60 | 85.71% |
| IUD | 10 | 14.29% |
| TOTAL | 70 | 100% |

**FIGURE 16:FOETAL OUTCOME IN STUDY PARTICIPANTS**




Out of 70 cases, 60 babies were alive and 10 were dead born.

**FIGURE 17:FREQUENCY DISTRIBUTION OF PRE ECLAMPSIA CASES COMPARED WITH D-DIMER VALUES**

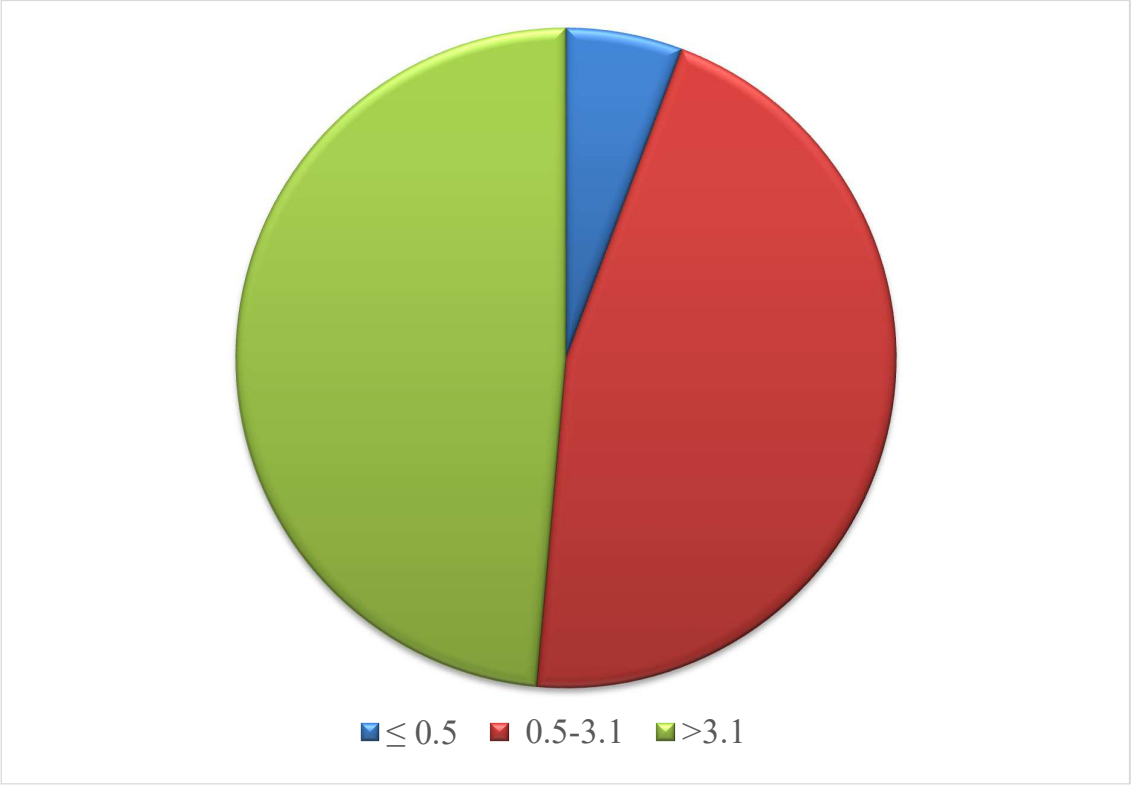


**TABLE 17:FREQUENCY DISTRIBUTION OF PRE ECLAMPSIA CASES COMPARED WITH D-DIMER VALUES**

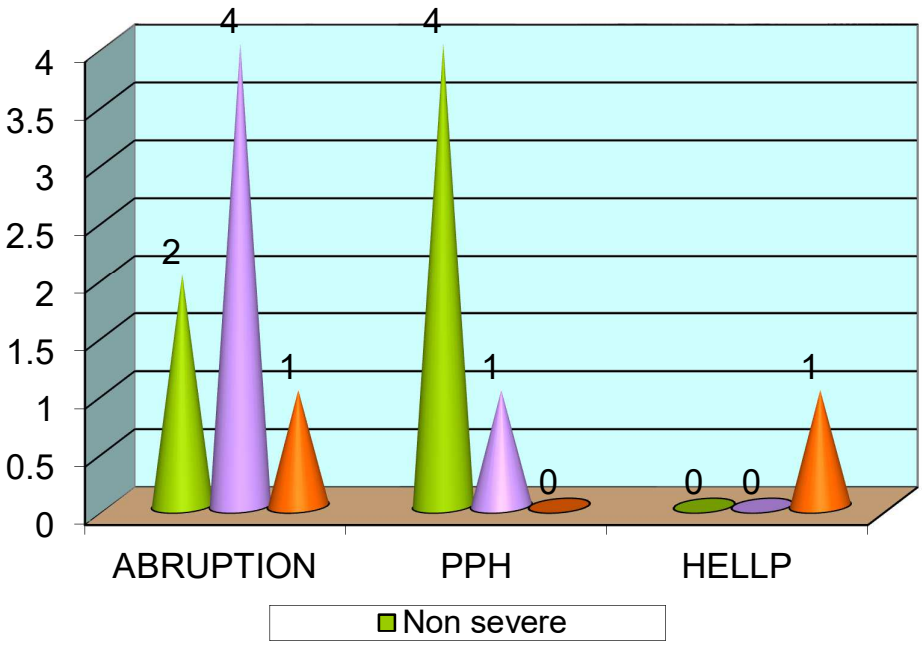
| D-dimer (µg FEU/mL) | No. of Case | Percentage |
|---|---|---|
| ≤ 0.5 | 4 | 5.7 |
| 0.5-3.1 | 32 | 45.7 |
| >3.1 | 34 | 48.6 |
| Total | 70 | 100.0 |

Considering the normal value of d-dimer to be less than 0.5µg FEU/mL, only 4 cases (5.7%) had d-dimer values less than the cut-off. 34 cases out of 70 (48.6%) had values more than the $97.5^{th}$ percentile (i.e.,3.1 µg FEU/mL)

**TABLE 18: DISTRIBUTION OF COMPLICATIONS**

| COMPLICATIONS | ABRUPTION | FREQUENCY |
|---|---|---|
| ABRUPTION | 7 | 10% |
| PPH | 5 | 7.14% |
| HELLP SYNDROME | 1 | 1.42% |
| NIL | 57 | 81.42% |

**FIGURE 19: DISTRIBUTION OF COMPLICATIONS**

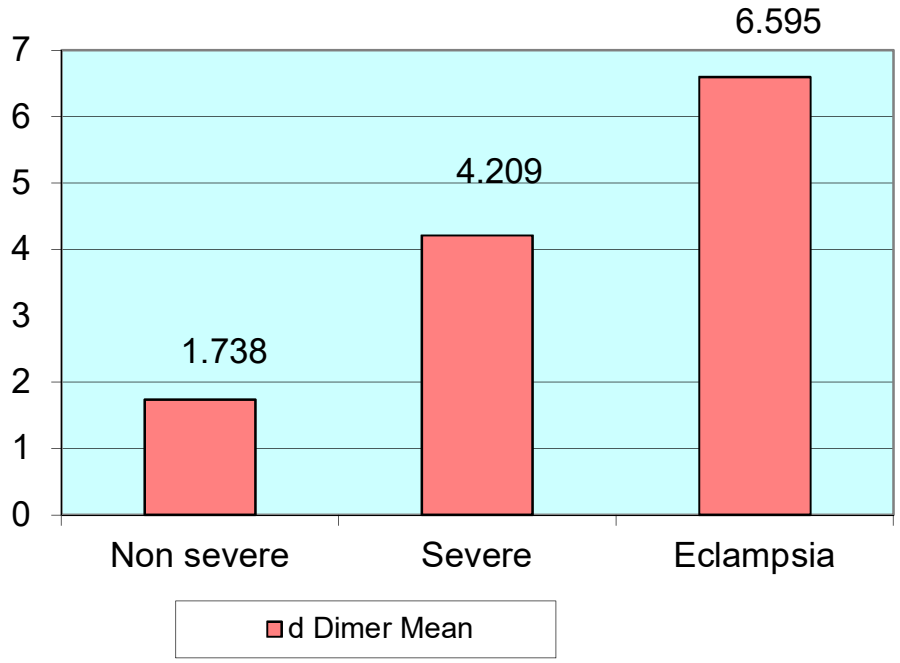



10% of pre eclamptic mothers had abruption; 7.14% developed PPH; 1.42% had HELLP syndrome.

**TABLE 20: DIAGNOSTIC EFFICACY OF D-DIMER IN PREDICTING THE SEVERITY OF PRE-ECLAMPSIA**

| | d Dimer Mean (µg FEU/mL) | SD | P'value |
|---|---|---|---|
| Non severe | 1.738 | 1.156 | <0.001 |
| Severe | 4.209 | 1.976 | |
| Eclampsia | 6.595 | 4.203 | |

**FIGURE 20:DIAGNOSTIC EFFICACY OF D-DIMER IN PREDICTING THE SEVERITY OF PRE-ECLAMPSIA**

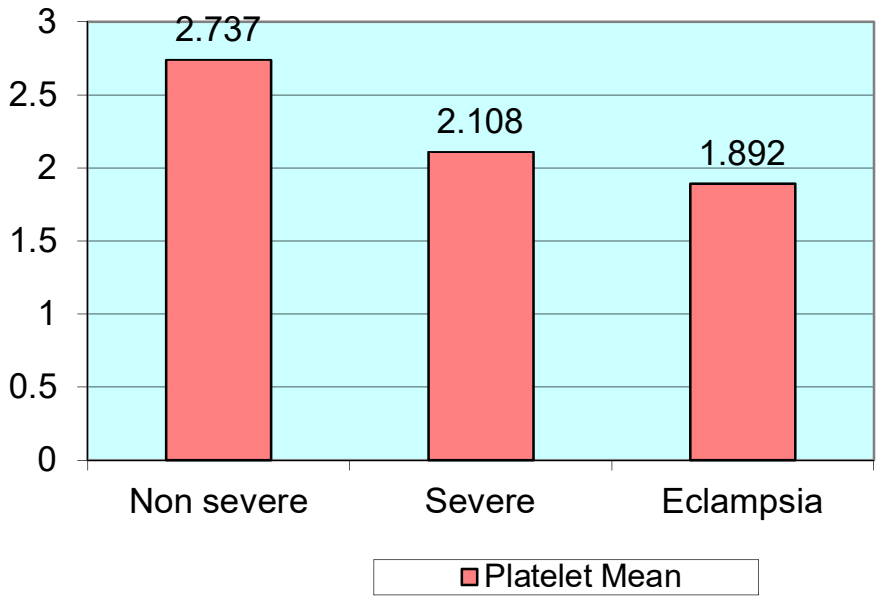



The mean value of d-dimer is compared to the severity of pre eclampsia. It indicates p<0.001 and considered statistically significant.

**TABLE 21:COMPARISON OF D-DIMER TO PLATELET COUNT**

| D-dimer | Mean | SD | P'value |
|---|---|---|---|
| < 0.5(5) | 3.22 | 0.235 | 0.089 |
| 0.5 - 3.1(34) | 2.531 | 0.837 | |
| > 3.1(31) | 2.295 | 0.974 | |

**FIGURE 21:COMPARISON OF D-DIMER TO PLATELET COUNT**

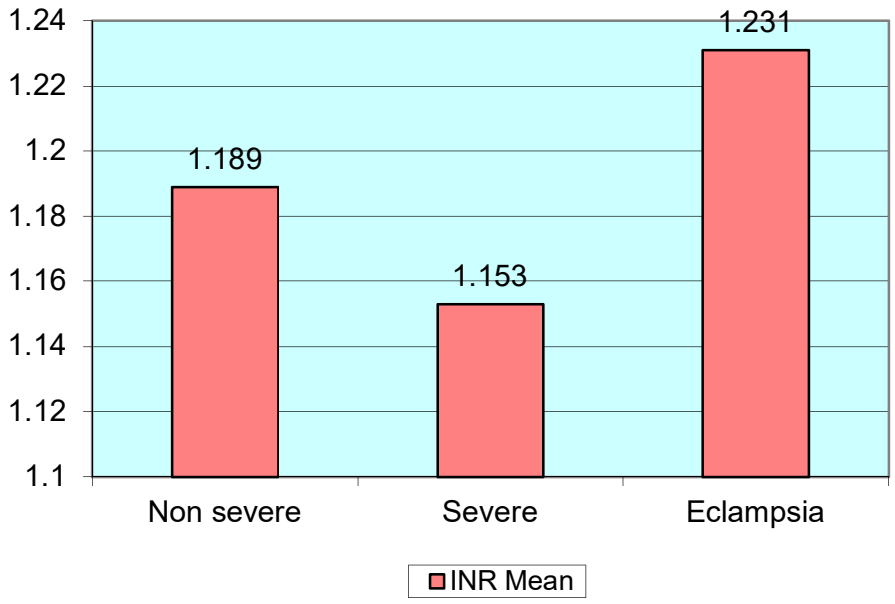



The mean platelet count compared to d-dimer value is not found to be satistically significant.

**TABLE 22:COMPARISON OF D-DIMER TO INR**

| D-dimer | Mean | SD | P'value |
|---|---|---|---|
| < 0.5(5) | 1.12 | 0.156 | 0.859 |
| 0.5 - 3.1(34) | 1.194 | 0.259 | |
| > 3.1(31) | 1.182 | 0.315 | |

**FIGURE 22:COMPARISON OF D-DIMER TO INR**




There was no significant association between INR to d-dimer values

**TABLE 23:COMPARISON OF D-DIMER VALUES TO THE MODE OF DELIVERY IN PRE ECLAMPTIC MOTHERS**

| D-dimer | LSCS | NVD | VBAC | assisted breech | Hysterotomy | Total |
|---|---|---|---|---|---|---|
| < 0.5(5) | 2 | 3 | 0 | 0 | 0 | 5 |
| 0.5 - 3.1(34) | 15 | 18 | 1 | 0 | 0 | 34 |
| > 3.1(31) | 16 | 12 | 0 | 1 | 2 | 31 |
| Total | 33 | 33 | 1 | 1 | 2 | 70 |
| P value | 0.019 significant | | | | | |

There was a significant association between mode of delivery and d-dimer values with p value=0.019

**TABLE 24:COMPARISON OF D-DIMER TO FOETAL OUTCOME**

| D-dimer | ALIVE | IUD | Total |
|---|---|---|---|
| < 0.5(5) | 5 | 0 | 5 |
| 0.5 - 3.1(34) | 31 | 3 | 34 |
| > 3.1(31) | 24 | 7 | 31 |
| Total | 60 | 10 | 70 |
| P value | 0.003 significant | | |

In our study, when comparing the d-dimer and foetal outcome there was a significant rise in d-dimer values in cases of intrauterine deaths.

**TABLE 25: COMPARISON OF D-DIMER TO PRETERM BIRTH**

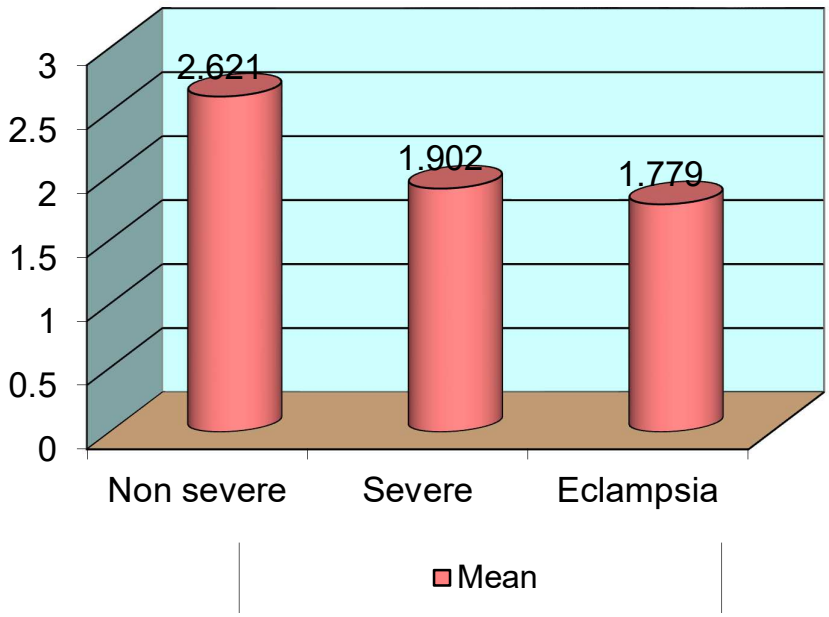
| D-dimer | Yes | No | Total |
|---|---|---|---|
| < 0.5(5) | 0 | 5 | 5 |
| 0.5 - 3.1(34) | 5 | 29 | 34 |
| > 3.1(31) | 10 | 21 | 31 |
| Total | 15 | 55 | 70 |
| P value | 0.406 not significant | | |

The d-dimer values and preterm births are not statistically significant since the p value is 0.406.

**TABLE 26:COMPARISON OF D-DIMER TO BIRTH WEIGHT OF BABIES**

| D-dimer | Mean | SD | P'value |
|---|---|---|---|
| < 0.5(5) | 2.68 | 0.494 | 0.005 |
| 0.5 - 3.1(34) | 2.499 | 0.627 | |
| > 3.1(31) | 1.964 | 0.789 | |

**FIGURE 23:MEAN BIRTH WEIGHT OF BABIES BORN TO PRE ECLAMPTIC WOMEN**




The mean birth weight of babies born to per-eclamptic mothers is significantly affected in cases with higher d-dimer values. P value is <0.005.

**FIGURE 24:COMPARISON OF D-DIMER AND NICU ADMISSION**

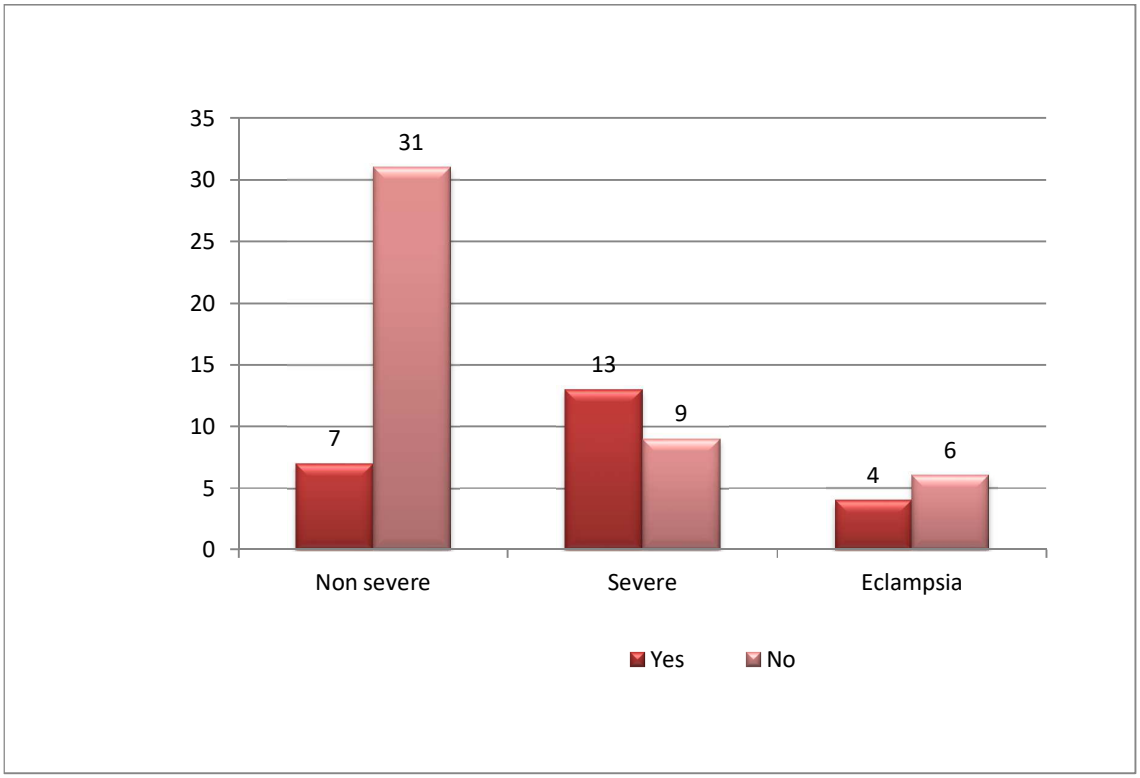



**TABLE 27:COMPARISON OF D-DIMER AND NICU ADMISSION**

| D-dimer | Yes | No | Total |
|---|---|---|---|
| < 0.5(5) | 1 | 4 | 5 |
| 0.5 - 3.1(34) | 8 | 26 | 34 |
| > 3.1(31) | 15 | 16 | 31 |
| Total | 24 | 46 | 70 |
| P value | 0.002 | | |

Number of babies admitted in NICU and elevated D-dimer levels were statistically significant.

# *DISCUSSION*

## DISCUSSION

The current study was conducted in 70 study participants. The age distribution of the study group was 18-35 years. The peak incidence of pre eclampsia in our study was between 26 to 30 years (34.3%) followed by 21 to 25 years (31.4%). It was correlated with the study done by Chaware et al., in India where the incidence of pre eclampsia was found to be 57.5% in 20-24 years and 33.3% in 25-29 years whereas in another study study in India by Vijayalakshmi et al., the peak incidence was 45% in the age group of 25-29 years. In a study by Lei Han et al the peak incidence of pre eclampsia is between 25-29 years(28). The mean age of development od severe pre eclampsia in a study done by Lothar Heilmann et al was 33±7. In contrary, the Indian population has a much earlier onset of pre eclampsia probably due to younger age at marriage and childbirth. Pre eclampsia is common in extremes of ages. In our study, 15 women (21.4%) were 18-25 years of age and 9 women(12.9%) were more than 30 years of age. The mean age of incidence of pre eclamptic patients was 25.414±4.617 years.

Among the pre eclamptic cases the number of primigravida were 45 (64.3%) and multigravida were 25 (35.7%). In our study, the incidence of pre eclampsia is higher in primigravida when compared to multigravida. The distribution of primigravida and multigravida in various Indian studies were 64% and 36%, 62.5% and 37.5%. The distribution is parity is comparable to other studies.

Obesity is considered as a risk factor for pre eclampsia. The number of obese pre eclamptic women was 28(40%) whose Body mass index were more than 30. Overweight women whose body mass index is 26-30 among the study group were 31 (44.3%). Robert et al in his review article has quoted that there is a moderate risk of pre eclampsia in women

with BMI 35kg/$m^2$ or more. Kim et al in his study mentioned the average BMI of patients with non-severe pre eclampsia to be 22.6kg/$m^2$ and for severe pre eclampsia 23.0kg/$m^2$(29).

Out of 70 cases, 57 cases were with no added comorbid conditions. Among 13 cases, 6 had gestational diabetes. **Michelle Ogunwole et al studied** the association between preeclampsia in the subsequent pregnancy (defined as incident or recurrent preeclampsia) and cardio-metabolic risk factors (i.e, obesity, hypertension, diabetes mellitus, preterm birth, low birth weight, and gestational diabetes mellitus) diagnosed before and during the index pregnancy, and between index and subsequent pregnancies(30).

At the subsequent pregnancy, 7% (36/540) had incident preeclampsia and 42% (33/78) had recurrent preeclampsia. Compared with women without obesity, women with obesity had greater risk of incident preeclampsia (unadjusted risk ratio [RR], 2.2 [95% CI, 1.1–4.5]) and recurrent preeclampsia (unadjusted RR, 3.1 [95% CI, 1.5–6.7]). Pre-index pregnancy chronic hypertension and diabetes mellitus were associated with incident, but not recurrent, preeclampsia (hypertension unadjusted RR, 7.9 [95% CI, 4.1–15.3]; diabetes mellitus unadjusted RR, 5.2 [95% CI, 2.5–11.1].

The mean systolic blood pressure among study group is 158±13. 44.3% cases had systolic blood pressure in the range of 140-149mmHg and 32.9% cases with blood pressure >160mmHg.

The mean diastolic blood pressure among the study group is 101±11 mmHg. 44% had diastolic blood pressure in the range of 90-95mmHg whereas 21.4% had diastolic blood pressure >110mmHg.

Proteinuria was observed in all cases. 37.1% (26 cases) had urine albumin 1+ which is 0.3mg/dL; 20% (14 cases) had urine albumin 2+ which is 1g/dL; 42.9% (30) with urine

albumin 3+ which is 3g/dL. Out of 30 cases, 8 were eclampsia. Other 2 eclamptic patients had urine albumin of 2+. Howie et al (1971) found proteinuria of 0.19 gm/dl (Nil-0.35) in mild pre-eclampsia and 5.15 gm/dl (1.5-10) in severe pre-eclampsia(31). Lopez-Lleraet al (1976) found proteinuria of 4.15 gm/L in severe pre-eclampsia(32). Proteinuria in women in a study conducted in Andhra Pradesh, India showed in antepartum Eclampsia group, out of 18 patient's majority of about 51 (83.3%) had 3+ proteinuria. In severe pre eclampsia group out of 32 patients 23 (71.9%) had 2+protienuria. None of the normotensive patients had proteinuria. The result of chi-square test shows that there is high association between urine albumin and clinical diagnosis.

Among 70 cases, 4 cases (5.7%) were diagnosed to be pre eclampsia at less than 28 weeks of gestation and testing was done at this time. 50 cases (71.4%) were diagnosed and tested at 29-36 weeks of gestation. Only 16 cases (22.9%) were diagnosed as pre eclampsia at term(>37 weeks) and testing done at this time.

Out of 70 patients, 38 patients had non-severe pre eclampsia, 22 patients had severe eclampsia and 10 patients developed eclampsia (both antepartum and postpartum eclampsia). Preeclampsia affects an estimated 4.6% of pregnancies globally. In India, the incidence of preeclampsia is reported to be 8-10% among the pregnant women. According to a study, the prevalence of hypertensive disorders of pregnancy was 7.8% with preeclampsia in 5.4% of the study population in India.

78.6%(55) had term deliveries and 21.4%(15) had preterm deliveries among pre eclamptic participants. The d-dimer values and preterm births are not statistically significant in our study. In a study done at Columbia, Preterm birth in pre eclampsia was reported in 55.3% of the patients and 34.8% suffered from early-onset pre-eclampsia. Kim et al found 80% of patients with the highest $10^{th}$ percentile concentrations of d-dimer had severe

gestational hypertensive disorder and delivered at preterm(29). However, there were a few cases with non-severe gestational hypertensive disorder and full-term delivery even though their markedly elevated levels of d-dimer.

Out of 70 cases, 60 babies were alive and 10 were dead born. In our study, when comparing the d-dimer and foetal outcome there was a significant rise in d-dimer values in cases of intrauterine deaths. In a Norwegian Mother and Child cohort study from 1999 to 2008 preeclampsia was recorded in 3.8% (n=21,020) of all pregnancies. Risk of stillbirth was 3.6 per 1,000 overall and 5.2 per 1,000 among pregnancies with preeclampsia.(33).

10% of pre eclamptic mothers had abruption; 7.14% developed PPH; 1.42% had HELLP syndrome. kumar et al found 41% had PPH and HELLP syndrome in severe eclampsia which explains that derangement in coagulation profile would have significantly associated with fetal morbidity. Abruptio placentae were associated with all hypertensive disorders of pregnancy. Audibert et al. reported that the rate of placental abruption was 5% in women with severe PE. Sibai et al reported the incidence of abruption placentae as 5.6%. Extensive placental abruption might results in immediate and frequently profound DIC

The mean platelet count compared to d-dimer value is not found to be satistically significant. Jaremo P. et al., 2000 mentioned in their study [10]. Srivastava. et al., (1995) reported mean platelet count of 1.94 lakh/cumm in normal pregnant control, 1.79 lakh/cumm in mild preeclampsia, & significantly low platelet count in severe preeclampsia i.e. 1.64 lakh/cumm and in eclampsia i.e. 1.52 lakh/cumm.

In our study, there was no significant association INR to d-dimer values. The prolongation of APTT in severe PE cases was concluded in a study by Osmanagaoglu et al but they didn't found any significant decrease in S.Fibrinogen levels in severe PE group at late pregnancy.

There was a significant association between mode of delivery and d-dimer values with in our study. In a study done by Pacher J et al considering the delivery mode, significant differences were found in favour of the elective Cesarean section. There were no differences in the rate of NICU admission between the groups.

The mean birth weight of babies born to per-eclamptic mothers is significantly affected in cases with higher d-dimer values in our study. The mean new born weight in severe PE in a study done by Lothar Heilman et al was $2.06 \pm 0.690$

Considering the normal value of d-dimer to be less than 0.5µg FEU/mL, only 4 cases (5.7%) had d-dimer values less than the cut-off. 34 cases out of 70 (48.6%) had values more than the $97.5^{th}$ percentile (i.e.,3.1 µg FEU/mL).

The mean value of d-dimer is compared to the severity of pre eclampsia. It indicates $p<0.001$ and considered statistically significant. In a meta-analysis by M.B. Pinheiro et al, they concluded that the measurement of D-Dimer in PE patients was a prognostic tool in assessing the pregnancy outcome. In accordance to our study maternal concentrations of d-dimer were significantly elevated in PE patients with severe features than those without severe features in a study done Se Jeong Kim et al.

Dusse et al.19 and Kim et al.21 found a significant elevation of D-dimer in patients with severe pre-eclampsia versus patients without criteria for severity(34)(29). One benefit of the D-dimer assay is the high negative predictive value close to 99% that is used as a diagnostic tool to rule out thromboembolic events in patients with low pre-test probability.

D-dimer assays are being used to predict the risk of recurrence of thromboembolic events, to diagnose and monitor disseminated intravascular coagulation, to guide anticoagulant therapy, and as a prognostic factor for thrombotic complications in patients

with malignant neoplasms, serious infections, pre-eclampsia, migraine, or placental abruption.

However, its clinical usefulness is limited by some clinical conditions and individual heterogeneity, which raises the importance of cut-off points adjusted for population groups. Models that combine D-dimer with other biomarkers could improve its diagnostic accuracy.

# *CONCLUSION*

# CONCLUSION

In our study, there was a strong association between severity of pre-eclampsia and elevated D-dimer levels (>97.5 percentile for gestational age), reinforcing the evidence that one of the condition's physiopathologic bases lies in the activation of the coagulation system and fibrinolysis. The clinical application shall be focused on identifying patients susceptible to an adverse prognosis, especially HELLP syndrome, or who will require monitoring in the ICU.

The exact time during pregnancy in which the mechanisms that unchain the pathology and allow the D-dimer to be used as a predictor marker have not been defined. Increased levels of D-dimer should be interpreted as evidence of activation of the physiopathologic mechanisms that contribute to placental ischemia and multiple organ dysfunction in pre-eclampsia.

Further, the D-dimer levels if continuously monitored can be helpful in early intervention for better pregnancy outcome.

However, serial monitoring of D-dimer in larger study population could reiterate the findings observed for better patient management.

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**PROFORMA**

**"A study on the association of elevated d-dimer levels and the extent of severity of pre eclampsia"**

Name:

age:

Ip no:

Height:

Weight:

BMI:

Address:

Contact no:

Socioeconomic status: class i/ ii/ iii/ iv/ v

Educational status:

Obstetric code

LMP: EDD: GESTATIONAL AGE:

TRIMESTER:

GESTATIONAL AGE AT THE TIME OF DIAGNOSIS OF PRE ECLAMPSIA:

EALRY ONSET PRE ECLAMPSIA (<34weeks):yes/no

LATE ONSET PRE ECLAMPSIA(>34weeks):yes/no

Chief Complaints :

Imminent symptoms :

Menstrual history :

Obstetric history :

Past history :

any other comorbidities(including coagulation disorders):

previous history of gestational hypertension/pre eclampsia/eclampsia:

use of any anticoagulants:

Family history :

Personal history :

**General examination:**

Pallor :

Icterus. :

Skin discolouration/petechiae. :

Pedal Edema :

Calf muscle tenderness :

Temperature :

Pulse rate :

BP :

CVS :

RS :

**On examination:**

P/A examination:

P/V examination:

Baby details:

Alive /Dead born

Term/preterm

Baby weight

APGAR

**<u>INESTIGATIONS</u>**

HEMOGLOBIN :

PLATELET COUNT :

UREA :

CREATININE :

URINE ALBUMIN. :

Urine spot PCR. :

SGOT :

SGPT :

TOTAL BILIRUBIN/DIRECT BILIRUBIN :

PROTHROMBIN TIME :

ACTIVATED PARTIAL THROMBOPLASTIN TIME:

INR :

PERIPHERAL SMEAR STUDY :

**D-dimer :**

**ULTRASOUND EXAMINATION**

Live/IUD

Presentation

Placental position

BPD

FL

AC

HC

AFI

Gestational age

Estimated foetal weight

Doppler study

Lower limb doppler study

## LIST OF ABBREVIATIONS

**PE - Preeclampsia**

**ACOG - American college of Obstetrics and Gynaecology**

**PLT - Platelet count**

**HELLP - Hemolysis, Elevated liver enzymes, Low platelet count**

**DIC - Disseminated Intravascular Coagulation**

**Hct - Hematocrit**

**MCV - Mean Corpuscular Volume**

**PGE2 - Prostaglandin E2**

**ITP - Idiopathic thrombocytopenic purpura**

**ADP - Adenosine diphosphate**

**PDW - Platelet Distribution Width**

**MPV - Mean Platelet Volume**

**TF - Tissue Factor**

**TNF - Tumour Necrosis Factor**

**TFPI - Tissue Factor Pathway Inhibitor**

**APTT - Activated Partial Thromboplastin Time**

**PT - Prothrombin time**

**TT - Thrombin time**

**APC - Activated Protein C**

**AT III - Antithrombin III**

**TAT complex - Thrombin-Antithrombin Complex**

**PLG - Plasminogen**

**t-PA - Tissue Type Plasminogen Activator**

**u-PA - Urokinase Type Plasminogen Activator**

**TAFI - Thrombin-Activatable Fibrinolysis Inhibitor**

**PAI-1 - Plasminogen Activator Inhibitor-1**

**PAI-2 - Plasminogen activator inhibitor-2**

**α2-PI - α2-Plasmin Inhibitor**

**FDP - Fibrin Degradation Products**

**PAP complex - Plasmin-α2-antiplasmin complex**

**BP - Blood Pressure**

**HLA - Human Leucocyte Antigen**

**Flt1 - fms-like tyrosine kinase-1**

**sFlt-1 - soluble fms-like tyrosine kinase-1**

**VEFGR - Vascular Endothelial Growth Factor Receptor**

**VEGF - Vascular Endothelial Growth Factor**

**PlGF - Placental Growth Factor**

**sEng - Soluble Endoglin**

**DNA - Deoxyribonucleic Acid**

**mRNA - Messenger Ribonucleic Acid**

**USG - Ultrasonogram**

**AFI - Amniotic fluid index**

**NST - Non stress test**

**BPP - Biophysical Profile**

**IUGR - Intrauterine growth retardation**

**NICE - National institute for health and clinical excellence**

**RCOG - Royal College of Obstetrics and Gynaecology**

**FFP - Fresh frozen plasma**

**GOVERNMENT STANLEY MEDICAL COLLEGE & HOSPITAL,CHENNAI_-01**

**INSTITUTIONAL ETHICS COMMITTEE**

| | |
|---|---|
| TITLE OF THE WORK | : "A STUDY ON THE ASSOCIATION OF ELEVATED D-DIMER LEVELS AND THE EXTENT OF SEVERITY OF PRE ECLAMPSIA" |
| PRINCIPAL INVESTIGATOR | : DR.M.MONISHA, |
| DESIGNATION | : PG IN OBSTETRICS AND GYNAECOLOGY , |
| DEPARTMENT | : DEPARTMENT OF OBSTETRICS AND GYNAECOLOGY, GOVT. STANLEY MEDICAL COLLEGE. |

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 03.11.2020 at the Council Hall, Stanley Medical College, Chennai-1 at 10am.

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

**MEMBER SECRETARY,**
**IEC, SMC, CHENNAI**

## CONSENT FORM

**PATIENT NAME:**

**IP NO.:**

**STUDY TITLE: "A STUDY ON THE ASSOCIATION OF ELEVATED D-DIMER LEVELS AND THE EXTENT OF SEVERITY OF PRE ECLAMPSIA"**

I agree to participate in the study entitled and have been informed about the details of the study in my own language.

I have completely understood the details of the study.

I am aware of the possible risks and benefits, while taking part in the study.

I understand that I can withdraw from the study at any point of time and even then, I can receive the medical treatment as usual.

I understand that I will not get any money for taking part in the study.

I will not object if the results of this study are getting published in any medical journal, provided my personal identity is not revealed.

I know what I am supposed to do by taking part in this study and I assure that I would extend my full cooperation for this study

Name of the participant :

Signature / Left thumb print:

Date :

Name of the investigator: Dr.M.Monisha

Signature of investigator:

Date:

## PATIENT INFORMATION SHEET

**Confidentiality:**

Utmost priority will be given to protect the privacy and confidentiality of your personal information. The collected information will not be shared with anyone not involved in the study and reporting will be done in aggregate form only

**Voluntary participation:**

Your participation in this study is voluntary and you have the right to withdraw your participation at any time during the interview without any explanation. Refusal to participate will not involve any penalty or loss of benefits to which you are otherwise entitled. There might be certain questions which you may not wish to answer. You can choose to decline answering these questions

## ஆராய்ச்சிஒப்புதல்படிவம்

உயர்த்தப்பட்ட டி டைமர் மற்றும் கர்ப்பகால உயர் இரத்த அழுத்தம் ஆகியவற்றின் தொடர்பு குறித்த ஆய்வு

ஆராய்ச்சியாளர்பெயர் : மருத்துவர்.

பங்கேற்பாளர்பெயர்:

பங்கேற்பாளர்எண் :

மேலேகுறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது .என்னுடைய சந்தேகங்களைகேட்கவும், அதற்கானதகுந்த விளக்கங்களை பெறவும்வாய்ப்பளிக்கப்பட்டது. நான் இவ்வாய்வில் தன்னிச்சையாகத்தான் பங்கேற்கிறேன் .எந்த காரணத்தினாலோ எந்த கட்டத்திலும்எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகிகொள்ளலாம் என்றும் அறிந்து கொண்டேன்.

இந்தஆய்வு சம்மந்தமாகவோஇதை சார்ந்த மேலும் ஆய்வுமேற்கொள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளைபார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன் .நான் ஆய்வில் இருந்து விலகிகொண்டாலும் இது பொருந்தும் என அறிகிறேன்.

இந்தஆய்வின் மூலம் கிடைக்கும் தகவல்களையும், பரிசோதனை முடிவுகளையும் மற்றும் சிகிச்சை தொடர்பானதகவல்களையும் மருத்துவர் மேற்கொள்ளும் ஆய்வில் பயப்படுத்திக்கொள்ளவும் அதை பிரசுரிக்கவும்என் முழுமனதுடன் சம்மதிக்கின்றேன்

இந்தஆய்வில் பங்கு கொள்ள ஒப்புக்கொள்கிறேன்.எனக்கு கொடுக்கப்பட்ட அறிவுரைகளின்படி நடந்துகொள்வதுடன் இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்று உறுதியளிக்கிறேன்.

ஆராய்ச்சியாளர் கையொப்பம் பங்கேற்பாளர் கையொப்பம்

தேதி

## தகவல்நகல்

உயர்த்தப்பட்ட டி டைமர் மற்றும் கர்ப்பகால உயர் இரத்த அழுத்தம் ஆகியவற்றின் தொடர்பு குறித்த ஆய்வு

இந்த ஆராய்ச்சியில் உங்களிடம் கேட்கப்படும் கேள்விகளுக்கு உங்கள் முழுமனதுடன் பதிலளிக்கவேண்டும்.

இந்த ஆராய்ச்சியில் உங்களுக்கு எந்த பின்விளைவும் ஏற்படாது என்பதை நான் உறுதியளிக்கிறேன்.

உங்களுக்கு பணம் எதுவும் அளிக்கப்படாது என்பதை இதன் மூலம் தெரிவிக்கிறேன்.

இந்த ஆய்வில் உங்களுக்கு எந்த நேரடி பயன்எதுவும் இல்லை. நீங்கள் அளிக்கும் தகவல் மூலம் புதுயுக்திகள் வகுக்கப்படலாம்.

அதன் மூலம் வருங்காலத்தில் உங்களுக்கோ அல்லது உங்களை போன்ற மக்களுக்கு பயன்படலாம்.

நீங்கள் எப்போது வேண்டுமானாலும் இந்த ஆய்விலிருந்து விலகிக்கொள்ளலாம் .அதனால் உங்களின் விபரங்கள் எதுவும் தெரிவிக்கப்படாது என்பதை உறுதியளிக்கிறேன்.

இந்த ஆராய்ச்சியின் முடிவுகள் பத்திரிக்கைகளில் பிரசுரிக்கப்படலாம். ஆனால் உங்களின் அடையாளம் எதுவும் தெரிவிக்கப்படாது.

Curiginal

## Document Information

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| **Analyzed document** | d dimer thesis review of literature.docx (D123517429) |
| **Submitted** | 2021-12-22T17:45:00.0000000 |
| **Submitted by** | Monisha M |
| **Submitter email** | monishamohan.3694@gmail.com |
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| **Analysis address** | monishamohan.3694.mgrmu@analysis.urkund.com |

## Sources included in the report

| | | |
|---|---|---|
| W | URL: https://www.medpulse.in/Pathology/Article/Volume10Issue3/Pathology_10_3_9.pdf<br>Fetched: 2021-12-22T17:45:46.4270000 | 2 |
| SA | **GARIMA ALL IN ONE THESIS.docx**<br>Document GARIMA ALL IN ONE THESIS.docx (D42379778) | 1 |
| SA | **Tamil Nadu Dr. M.G.R. Medical University / QUANTITATIVE ANALYSIS OF MITOCHONDRIAL DNA IN PLACENTA IN SEVERE GESTATIONAL HYPERTENSION.pdf**<br>Document QUANTITATIVE ANALYSIS OF MITOCHONDRIAL DNA IN PLACENTA IN SEVERE GESTATIONAL HYPERTENSION.pdf (D123212593)<br>Submitted by: surya.alagunathan@gmail.com<br>Receiver: surya.alagunathan.mgrmu@analysis.urkund.com | 1 |
| W | URL: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6046127/<br>Fetched: 2019-09-26T07:55:05.7670000 | 1 |
| W | URL: https://www.aafp.org/afp/1999/0901/p829.html<br>Fetched: 2019-12-27T20:47:56.9000000 | 1 |
| SA | **Tamil Nadu Dr. M.G.R. Medical University / PREDICTION OF PREECLAMPSIA BASED ON THE LATERAL LOCATION OF PLACENTA ON ULTRASONOGRA1(1).docx**<br>Document PREDICTION OF PREECLAMPSIA BASED ON THE LATERAL LOCATION OF PLACENTA ON ULTRASONOGRA1(1).docx (D42454851)<br>Submitted by: ahsadreams@gmail.com<br>Receiver: ahsadreams.mgrmu@analysis.urkund.com | 1 |
| W | URL: https://www.gponline.com/haematology-pre-eclampsia-hellp/haematology/article/1101448<br>Fetched: 2019-10-10T16:24:57.7630000 | 1 |
| W | URL: https://www.guideline.or.kr/func/download_comm.php?path=L2hvbWUvdmlydHVhbC9ndWlkZS9uZXcvL3VwbG9hZC91c2VyX2NvbW11bml0eS8xNDA0Mjg2MDMzXzcuIE5JQ0UgaHlwZXJ0ZW5zaW9uIGluIHByZWduYW5jeSgyMDExKS5wZGY=&filename=MTQwNDI4NjAzM183LiBOSUNFIGh5cGVydGVuc2lvbiBpbiBwcmVnbmFuY3koMjAxMSkucGRm&gubun=file_etc7&number=84<br>Fetched: 2021-12-14T15:58:46.2770000 | 3 |
| W | URL: https://knowledgecommons.popcouncil.org/cgi/viewcontent.cgi?article=1998&context=departments_sbsr-rh<br>Fetched: 2021-11-23T05:50:17.8600000 | 1 |

## PLAGIARSM CERTIFICATE

This is to certify that this dissertation work titled **"A STUDY ON THE ASSOCIATION OF ELEVATED D-DIMER LEVELS AND THE EXTENT OF SEVERITY OF PREECLAMPSIA"**of the candidate Dr. M. MONISHA with Reg. no. **221916061** for the award of M.S in the branch of OBSTETRICS AND GYNAECOLOGY. I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion pages and the result shows twenty percentage of plagiarism in the dissertation (D123517429)

Signature and Seal of the guide

Associate Prof. Dr. MANICKADEVI MD(OG), DCE

Govt. R.S.R.M Lying in Hospital,

Stanley Medical College,

Chennai

# MASTER CHART

| SL.NO | NAME | AGE | HEIGHT(m) | WEIGHT(kg) | BMI | GRAVIDA | COMORBS | GEST AGE AT SAMPLING | GEST AGE AT DELIVERY | SYS BP | DIA BP | URINE ALB | SEVERE/NON SEVERE PE/ECLAMPSIA | PLT | INR | D-dimer | FGR | ALIVE/IUD | PRETERM BIRTH | B. WEIGHT | NICU ADM | NVD/LSCS | COMPLICATIONS |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | barathi | 27 | 1.49 | 66 | 29 | primi | | 28 | 28 | 160 | 90 | 3+ | severe | 1.01 | 1.199 | 3.24 | | IUD | yes | 980gms | yes | NVD | |
| 2 | jayalakshmi | 23 | 1.5 | 67 | 29 | G2P1L1 | | 32 | 32 | 180 | 110 | 3+ | **eclampsia** | 1.2 | 1.19 | 10.7 | | alive | yes | 1.2kg | yes | LSCS | |
| 3 | durga | 22 | 1.43 | 59 | 28 | PRIMI | | 33 | 38 | 142 | 92 | 1+ | non severe | 2.5 | 1.87 | 3.4 | | alive | | 2.5kg | | NVD | |
| 4 | sudha | 31 | 1.51 | 63 | 27 | G4P1L1A2 | OVERT DM | 35 | 37 | 150 | 100 | 2+ | non severe | 3.12 | 1.24 | 1.3 | | alive | | 2.4kg | | LSCS | |
| 5 | elakkiya | 28 | 1.56 | 65 | 26 | G2P1L1 | | 30 | 30 | 170 | 110 | 3+ | severe | 3.83 | 0.975 | 6.1 | | IUD | yes | 1.25kg | | LSCS | ABRUPTION |
| 6 | lurdh mary | 29 | 1.5 | 69 | 30 | G3P1L1A1 | | 39 | 39 | 146 | 94 | 1+ | non severe | 3.4 | 1.12 | 0.3 | yes | alive | | 3.01kg | | LSCS | PPH |
| 7 | chellammal | 28 | 1.49 | 70 | 31 | G2P1L1 | | 28 | 38 | 148 | 92 | 1+ | non severe | 3.01 | 1.34 | 0.5 | | alive | | 2.89kg | | NVD | |
| 8 | abirami | 25 | 1.42 | 67 | 33 | PRIMI | | 30 | 38 | 150 | 90 | 1+ | non severe | 2.87 | 0.98 | 1.7 | | alive | | 2.7kg | | NVD | |
| 9 | zarina | 23 | 1.5 | 65 | 28 | G2A1 | | 32 | 37 | 164 | 90 | 2+ | non severe | 2.58 | 1.98 | 2.2 | | alive | | 2.68kg | | NVD | |
| 10 | karthika | 24 | 1.51 | 69 | 30 | PRIMI | | 37 | 37 | 170 | 110 | 3+ | **eclampsia** | 3.07 | 1.244 | 4.29 | | alive | | 2.5kg | | LSCS | |
| 11 | nandhini | 19 | 1.57 | 72 | 29 | PRIMI | | 38 | 38 | 150 | 90 | 2+ | non severe | 3.02 | 0.89 | 2.4 | | alive | | 2.67kg | | LSCS | |
| 12 | gowri | 20 | 1.4 | 78 | 39 | PRIMI | | 34 | 38 | 140 | 90 | 1+ | non severe | 1.5 | 1.34 | 1.2 | | alive | | 2.86kg | | NVD | |
| 13 | monisha | 33 | 1.41 | 74 | 37 | G3P2L2 | GDM | 37 | 37 | 146 | 94 | 1+ | non severe | 3.06 | 1.12 | 0.5 | yes | alive | | 1.85kg | yes | NVD | |
| 14 | kanchana | 25 | 1.48 | 60 | 27 | PRIMI | | 32 | 37 | 150 | 98 | 2+ | non severe | 2.17 | 0.98 | 2.3 | | alive | | 3.12kg | | LSCS | PPH |
| 15 | reshma | 24 | 1.45 | 64 | 30 | PRIMI | | 37 | 37 | 160 | 120 | 3+ | severe | 3.75 | 1.094 | 7.4 | | alive | | 2.85kg | YES | LSCS | ABRUPTION |
| 16 | sathya | 32 | 1.46 | 69 | 32 | G2P1L1 | | 39 | 39 | 154 | 94 | 1+ | non severe | 3.09 | 1.12 | 0.3 | | alive | | 3.04kg | | LSCS | |
| 17 | nagamani | 29 | 1.56 | 58 | 23 | PRIMI | GDM | 38 | 38 | 170 | 110 | 3+ | severe | 2.56 | 1.56 | 4.3 | | alive | | 3.1kg | | NVD | PPH |
| 18 | samundeeswari | 26 | 1.52 | 70 | 30 | PRIMI | | 36 | 38 | 160 | 90 | 2+ | non severe | 2.12 | 1.45 | 3.2 | | alive | | 2.89kg | | NVD | |
| 19 | sharmila | 31 | 1.5 | 68 | 30 | PRIMI | | 37 | 37 | 200 | 120 | 3+ | **eclampsia** | 2.27 | 0.99 | 7.3 | | alive | | 2.680kg | | LSCS | |
| 20 | swathi | 29 | 1.48 | 102 | 46 | PRIMI | | 30 | 30 | 166 | 110 | 3+ | severe | 3.74 | 1.064 | 4.98 | | alive | yes | 1.25kg | yes | NVD | |
| 21 | sathya | 35 | 1.5 | 90 | 40 | G2P1L1 | | 35 | 37 | 140 | 90 | 1+ | **PP eclampsia** | 3.12 | 1.244 | 3.25 | | alive | | 2.89kg | | LSCS | HELLP SYND |
| 22 | noorjahan | 33 | 1.51 | 85 | 37 | G4P1L1A2 | | 36 | 36 | 162 | 126 | 3+ | severe | 1.89 | 1.098 | 4.3 | | alive | | 2.5kg | | NVD | |
| 23 | kavitha | 20 | 1.56 | 75 | 30 | PRIMI | | 37 | 38 | 140 | 94 | 1+ | non severe | 2.14 | 1.133 | 2.1 | | alive | | 2.2kg | | NVD | |
| 24 | subhulakshmi | 25 | 1.48 | 64 | 29 | G3P1L1A1 | | 38 | 38 | 150 | 90 | 1+ | non severe | 2.02 | 1.12 | 1.23 | | alive | | 2.3kg | | LSCS | |
| 25 | syed sulfia | 26 | 1.44 | 69 | 33 | G3P2L2 | | 32 | 37 | 160 | 120 | 3+ | severe | 1.8 | 1.092 | 5.09 | | alive | yes | 1.15kg | yes | Ass breech | |
| 26 | pradeepa | 22 | 1.47 | 73 | 33 | PRIMI | | 34 | 34 | 150 | 100 | 3+ | **eclampsia** | 1.34 | 1.395 | 16.51 | | IUD | | 1.9kg | | LSCS | |
| 27 | narmadha | 22 | 1.57 | 82 | 33 | PRIMI | mild conc LVH | 36 | 38 | 168 | 112 | 3+ | severe | 2.1 | 1.064 | 4.48 | | alive | | 2.255kg | yes | LSCS | |
| 28 | parvathy | 23 | 1.48 | 80 | 36 | G2A1 | anemia | 37 | 37 | 140 | 90 | 1+ | non severe | 3.87 | 1.09 | 1.12 | | alive | | 2.8kg | | NVD | |
| 29 | arockiya mary | 27 | 1.49 | 70 | 31 | G2P1L1 | | 38 | 38 | 140 | 90 | 1+ | non severe | 3.54 | 0.9 | 0.45 | | alive | | 2.9kg | | NVD | |
| 30 | sunitha | 29 | 1.46 | 60 | 28 | PRIMI | HBsAg+ve | 38 | 38 | 160 | 114 | 3+ | severe | 1.52 | 1.035 | 1.05 | | alive | | 2.05kg | yes | LSCS | abruption |
| 31 | tamilselvi | 30 | 1.6 | 59 | 23 | G2P1L1 | | 36 | 37 | 154 | 90 | 2+ | non severe | 2.34 | 1.012 | 1.08 | yes | alive | yes | 1.6kg | yes | LSCS | |
| 32 | ammulu | 19 | 1.5 | 58 | 25 | PRIMI | | 32 | 32 | 148 | 90 | 1+ | non severe | 1.77 | 1.079 | 5.25 | yes | IUD | | 1.04kg | yes | LSCS | abruption |
| 33 | muthammal | 23 | 1.58 | 56 | 22 | G6P3L2A2 | | 37 | 37 | 170 | 110 | 3+ | severe | 36,000 | 1.2 | 8.09 | | alive | | 2.5kg | | NVD | |
| 34 | sasikala | 26 | 1.57 | 66 | 26 | PRIMI | | 38 | 38 | 142 | 90 | 1+ | non severe | 1.4 | 0.98 | 3.2 | | alive | | 2.75kg | | LSCS | |
| 35 | jerina bee | 20 | 1.59 | 64 | 25 | PRIMI | anemia | 30 | 36 | 164 | 114 | 3+ | severe | 2.6 | 1.34 | 1.2 | | alive | | 2.86kg | | LSCS | |
| 36 | manjula | 27 | 1.56 | 68 | 27 | G2P1L1 | | 32 | 37 | 160 | 90 | 3+ | non severe | 3.7 | 1.95 | 0.98 | | alive | | 2.97kg | | LSCS | |
| 37 | manimegalai | 18 | 1.48 | 90 | 41 | PRIMI | | 35 | 38 | 150 | 90 | 2+ | non severe | 4.8 | 1.02 | 0.85 | | alive | | 3.12kg | | NVD | |
| 38 | selvi | 32 | 1.5 | 82 | 36 | PRIMI | GDM | 36 | 36 | 166 | 112 | 3+ | severe | 1.01 | 1.98 | 6.6 | yes | alive | | 1.45kg | yes | LSCS | |
| 39 | kasturi | 26 | 1.51 | 76 | 33 | PRIMI | | 28 | 37 | 164 | 110 | 3+ | severe | 2.58 | 1.092 | 5.4 | | alive | | 2.8kg | yes | LSCS | |
| 40 | thilagavathy | 27 | 1.52 | 95 | 41 | PRIMI | | 35 | 35 | 190 | 110 | 3+ | **eclampsia** | 3.06 | 1.49 | 4.42 | | alive | yes | 2.060kg | yes | LSCS | |
| 41 | kirubha | 30 | 1.52 | 77 | 33 | G3P1L1A1 | | 36 | 36 | 154 | 96 | 2+ | non severe | 2.45 | 1.43 | 1.34 | | alive | yes | 2.1kg | yes | NVD | |
| 42 | sulthana | 27 | 1.5 | 80 | 35 | G2A! | GDM | 37 | 37 | 160 | 116 | 3+ | severe | 2.16 | 1.08 | 4.2 | yes | alive | | 1.610kg | yes | LSCS | |
| 43 | lakshmi | 39 | 1.58 | 79 | 31 | G3P2L2 | | 35 | 35 | 164 | 114 | 3+ | severe | 50,000 | 1.454 | 1.48 | | IUD | | 2.4kg | | LSCS | abruption |
| 44 | kathija bee | 26 | 1.49 | 83 | 37 | PRIMI | overt DM | 37 | 37 | 140 | 98 | 1+ | non severe | 2.1 | 1.65 | 1.65 | | alive | | 2.65kg | | NVD | |
| 45 | ramya | 24 | 1.47 | 73 | 33 | PRIMI | | 36 | 37 | 150 | 90 | 1+ | non severe | 2.2 | 1.05 | 2.13 | | alive | | 2.2kg | | LSCS | |
| 46 | meena | 23 | 1.48 | 70 | 31 | PRIMI | | 32 | 36 | 162 | 92 | 3+ | severe | 1.79 | 1.4 | 1.05 | yes | alive | yes | 1.4kg | yes | NVD | |
| 47 | manjula | 18 | 1.5 | 65 | 28 | PRIMI | | 34 | 34 | 180 | 110 | 3+ | **eclampsia** | 1.12 | 1.086 | 2.25 | yes | alive | yes | 1.180kg | yes | LSCS | abruption |
| 48 | meenatchi | 19 | 1.51 | 62 | 27 | PRIMI | | 40 | 40 | 176 | 118 | 3+ | severe | 3.03 | 1.25 | 2.52 | | alive | | 3.110kg | | LSCS | |
| 49 | pradeepa | 19 | 1.56 | 69 | 28 | PRIMI | | 38 | 38 | 150 | 92 | 1+ | non severe | 4.14 | 1.011 | 4.3 | | alive | | 2.89kg | | NVD | |
| 50 | archana | 20 | 1.57 | 59 | 23 | PRIMI | | 35 | 37 | 140 | 94 | 1+ | non severe | 3.08 | 1.07 | 2.1 | | alive | | 2.56kg | | NVD | |
| 51 | leena | 25 | 1.59 | 50 | 19 | PRIMI | | 30 | 38 | 170 | 114 | 3+ | severe | 2.14 | 1.09 | 3.56 | yes | alive | yes | 1.28kg | yes | NVD | |
| 52 | pavithra | 29 | 1.6 | 98 | 38 | PRIMI | anemia | 36 | 37 | 152 | 90 | 1+ | non severe | 3.23 | 1.09 | 2.32 | | alive | | 3.4kg | | LSCS | PPH |
| 53 | chitra | 26 | 1.58 | 80 | 32 | G2P1L1 | | 37 | 37 | 156 | 98 | 2+ | non severe | 1.54 | 1.199 | 2.75 | | IUD | | 3.02kg | | LSCS | abruption |
| 54 | jeyaseela | 35 | 1.57 | 75 | 30 | G4P3L1 | | 32 | 32 | 160 | 120 | 3+ | severe | 1.98 | 0.997 | 3.08 | | IUD | | 1.02kg | | VBAC | |
| 55 | soundari | 28 | 1.47 | 65 | 30 | G3P1L1A1 | | 35 | 37 | 146 | 98 | 1+ | non severe | 2.45 | 1.21 | 1.09 | | alive | | 2.54kg | | NVD | |
| 56 | meenatchi | 19 | 1.46 | 48 | 22 | PRIMI | GDM | 38 | 38 | 150 | 90 | 1+ | non severe | 3.12 | 1.02 | 0.65 | | alive | | 2.2kg | | NVD | |
| 57 | saranya | 24 | 1.48 | 105 | 47 | PRIMI | | 30 | 39 | 168 | 110 | 3+ | severe | 1.35 | 0.098 | 4.56 | yes | alive | yes | 1.85kg | yes | LSCS | |
| 58 | aruna prabha | 19 | 1.5 | 66 | 29 | PRIMI | | 32 | 35 | 154 | 96 | 1+ | non severe | 3.12 | 1.24 | 2.34 | yes | alive | yes | 1.65kg | yes | NVD | |
| 59 | usha | 20 | 1.4 | 54 | 27 | PRIMI | | 36 | 36 | 168 | 120 | 3+ | severe | 2.89 | 1.12 | 4.12 | | alive | yes | 2.12kg | yes | NVD | |
| 60 | rosy | 19 | 1.45 | 88 | 41 | PRIMI | | 30 | 30 | 200 | 120 | 3+ | **eclampsia** | 4.19 | 1.184 | 5.36 | | IUD | | 980gms | | LSCS | |
| 61 | mohana | 25 | 1.55 | 79 | 32 | PRIMI | | 41 | 41 | 148 | 104 | 1+ | non severe | 3.18 | 1.24 | 2.38 | | alive | | 3.85kg | yes | NVD | PPH |
| 62 | vasanthi | 25 | 1.58 | 65 | 26 | PRIMI | | 40 | 40 | 150 | 94 | 2+ | non severe | 2.47 | 1.14 | 3.21 | | alive | | 3.2kg | | NVD | |
| 63 | priya | 25 | 1.5 | 67 | 29 | PRIMI | | 20 | 20 | 150 | 100 | 2+ | **eclampsia** | 1.75 | 1.432 | 6.59 | yes | IUD | | 500gms | | hysteroromy | |
| 64 | selvi | 20 | 1.47 | 66 | 30 | PRIMI | | 35 | 38 | 146 | 96 | 2+ | non severe | 2.23 | 1.09 | 1.02 | | alive | | 2.9kg | | NVD | |
| 65 | priya | 27 | 1.44 | 60 | 28 | G2P1L1 | | 39 | 39 | 150 | 90 | 1+ | non severe | 3.1 | 1.01 | 0.58 | | alive | | 2.75kg | | NVD | |
| 66 | sarasu | 29 | 1.6 | 58 | 22 | PRIMI | | 32 | 32 | 160 | 102 | 2+ | **eclampsia** | 1.8 | 1.05 | 5.28 | | alive | yes | 1.9kg | yes | NVD | |
| 67 | nagalakshmi | 30 | 1.51 | 56 | 24 | G2P1L1 | | 34 | 37 | 148 | 96 | 1+ | non severe | 2.17 | 1.12 | 1.05 | | alive | | 2.12kg | yes | LSCS | |
| 68 | majitha kani | 24 | 1.57 | 70 | 28 | PRIMI | GDM | 30 | 30 | 175 | 110 | 3+ | severe | 1.78 | 1.08 | 5.8 | yes | IUD | | 700gms | | Hysterotomy | |
| 69 | jothi | 22 | 1.48 | 65 | 29 | PRIMI | | 38 | 38 | 160 | 92 | 2+ | non severe | 3.17 | 0.995 | 1.01 | | alive | | 3.1kg | | NVD | |
| 70 | deepa | 25 | 1.51 | 55 | 24 | PRIMI | | 40 | 40 | 150 | 92 | 1+ | non severe | 2.23 | 0.889 | 0.56 | | alive | | 2.96kg | | NVD | |