"One year observational study of maternal and perinatal outcome in severe pre-eclampsia at tertiary care hospital, Belagavi"

By

REG. NO. BJ0120009

Dissertation

Submitted to the KAHER, Belagavi, Karnataka
In partial fulfilment

of the requirements for the degree of

MASTER OF SURGERY (M.S.)

In

OBSTETRICS AND GYNAECOLOGY

J. N. MEDICAL COLLEGE, NEHRU NAGAR

BELAGAVI-590010

JUNE / JULY - 2023

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Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"ONE YEAR CROSS SECTIONAL STUDY OF MATERNAL AND PERINATAL OUTCOME IN SEVERE PRE-ECLAMPSIA AT A TERTIARY CARE HOSPITAL"**, is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

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LIST OF ABBREVIATIONS

AKI	:	Acute kidney injury
ANP	:	Atrial natriuretic peptide
ARDS	:	Acute respiratory distress syndrome
ACOG	:	Amercian college of obstetrices and gynaecology
CTG	:	Cardiotocography
CDMR	:	Cesarean delivery at maternal request
CPD	:	Cephalopelvic disproportion
DM	:	Diabetes mellitus
DIC	:	Disemminated intravascular coagulation
DTA	:	Deep transverse arrest
ELBW	:	Extremely low birth weight
FGR	:	Fetal growth restriction
FHR	:	Fetal heart rate
HELLP	:	Hemolysis, elevated liver enzymes, and low platelets
HCG	:	Human chorionic gonadotropin
HLA	:	Human leukocyte antigen
ICU	:	Intensive care unit
IU/L	:	International units/litre
IUD	:	Intrauterine death
LSCS	:	Lower segment cesarean section
LFT	:	Liver function test
LDH	:	Lactate dehydrogenase
LBW	:	Low birth weight

MSL	:	Meconium stained liquor
MAS	:	Meconium aspiration syndrome
NICU	:	Neonatal intensive care unit
NST	:	Non stress test
NPL	:	Non-progress of labor
NK	:	Natural killer cell
PRES	:	Posterior reversible encephalopathy syndrome
PGI2	:	Prostaglandin I2
PPH	:	Postpartum hemorrhage
RFT	:	Renal function test
RCOG	:	Royal College of Obstetricians and Gynaecologists
SGOT	:	Serum glutamic-oxaloacetic transaminase
SGPT	:	Serum glutamate pyruvate transaminase
TXA2	:	Thromboxane A2
USG	:	Ultrasonography
VEGF	:	Vascular endothelial growth factor
VLBW	:	Very low birth weight

ABSTRACT

Background: Preeclampsia is a multisystem disorder involving placenta, kidney, liver, blood, cardiovascular and neurovascular system, occurring exclusively during pregnancy whose etiology is not known. It occurs in approximately 5-7% of pregnancies. It is an important cause of morbidity as well as mortality in both the mother and fetus. Features of severe preeclampsia include severe proteinuria, hypertension, symptoms of central nervous system dysfunction, hepatocellular injury thrombocytopenia, oliguria, pulmonary edema, cerebrovascular accident, and FGR. Women with severe preeclampsia must be hospitalized to try to stabilize the disease.

Objective: To study the fetomaternal outcome in severe pre-eclampsia.

Materials and methods: The present prospective observational study was carried out at Dr. Prabhakar Kore Hospital and MRC", Belagavi attached to KAHER's Jawaharlal Nehru medical College, Belagavi from 1st January 2021- 31st December 2021 . Pregnant women with severe preeclampsia satisfying inclusion and exclusion criteria admitted in labor room at tertiary care centre were included in the study.

Results: The maximum age observed was 41 years and the minimum age observed was 19 years. The mean gestational age at diagnosis was 35.32 weeks with standard deviation of 3.7 weeks. Among the study population, 73.4% women underwent LSCS in comparison to 22.4% women who delivered vaginally out if which 3 had instrumental delivery (ventouse). In our study, increased incidence of HELLP in 32.4% patients, abruption in 30%, PPH in 22%, Partial HELLP in 16.2% , pulmonary edema in 11% , eclampsia &DIC in 8.1%. The incidence of prematurity was 55 % and IUFD were 7% in the study.

Conclusion: Life threatening complications like eclampsia, abruption, HELLP, DIC, and pulmonary edema were more common in unbooked and severe pre-eclampsia

category. Good antenatal care, early diagnosis and prompt treatment can prevent severe pre eclampsia and eclampsia.

Keywords: Severe preeclampsia, HELLP syndrome, maternal outcome, perinatal outcome.

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INTRODUCTION

Preeclampsia is a multisystem disorder involving placenta, kidney, liver, blood, cardiovascular and neurovascular system, occurring exclusively during pregnancy [1]. It occurs in approximately 5-7% of pregnancies. It is an important cause of morbidity as well as mortality in both the mother and fetus[2].

Pre-eclampsia is defined as multisystem, multifactorial disease with blood Pressure (B.P) reading of ≥140/90 mm Hg on two occasions 4 hours apart and /or >0.3 g protein in 24hour urine specimen after 20 weeks of gestation in a previously normotensive woman. **Severe pre-eclampsia** is B.P reading of ≥160/110 mm Hg and with/without >5 g protein in 24hour urine specimen or symptoms of end organ damage like deranged LFT, thrombocytopenia, oliguria, visual disturbances, pulmonary oedema etc[2]. **Eclampsia** is defined as generalised tonic clonic seizures and /or unexplained coma in a woman with pre-eclampsia. Pre-eclampsia is considered early onset if elevation of blood pressure and proteinuria occur before 34 weeks of pregnancy [3].

The incidence of pre-eclampsia is reported as 7.6 % and severe pre-eclampsia as 3.3 %. Pre-eclampsia alone accounts for 12–18 % of maternal mortality. The highest maternal mortality rate due to pre-eclampsia reported in developing country is 0.4 %. Besides, it is associated with a fivefold increase in perinatal mortality[4].

It is estimated that the global maternal mortality by the year 2017 was more than 303,000, and low and middle-income countries accounted for approximately 99% of the global maternal deaths due to severe pre-eclampsia. The sub-Saharan Africa

countries alone accounts for 66% (201 0000 while the Southern Asia region accounted for 66,000 maternal mortality in 2017 [5].

Hypertension and its complications is ranked third as a leading cause of maternal mortality, responsible for over 17% of maternal deaths [6]. Even in developed countries, women still die from severe pre–eclampsia and eclampsia. There is an increased risk of acute renal failure, cardiovascular and cerebrovascular complications, abruption placenta, disseminated intravascular coagulation, and even maternal death in patients with severe pre-eclampsia .So, early diagnosis and close monitoring of preeclampsia plays a vital role in preventing its complications.

Up to 10% of women have elevated blood pressures during their pregnancies. Three to eight percent of these women in developed countries develop severe pre-eclampsia and up to 0.56 /1000 births are complicated by eclampsia [7] . Due to absence of epidemiological information in many low and middle income countries; lack of effort and capacity for data collection and reporting of vital statistics, the exact prevalence of pre-eclampsia and associated morbidity and mortality from low and middle income countries is unknown. The WHO reported an overall prevalence of 2.2% ranging from 1.4% in the middle east region to 3.9% in the African region [8] .

Pre-eclampsia and eclampsia are significant contributors to the global burden of maternal and perinatal mortality with severe pre-eclampsia being responsible for an estimated 10% of maternal deaths annually [9].

In the WHO Multicountry Survey study, women with severe pre-eclampsia or eclampsia had an odds ratio of perinatal death of 3.0(95% CI 2.7 -3.3) and 4.9 (95% CI 4.1-5.9) respectively [8].

It is found that 3-5% of first pregnancies are complicated by pre-eclampsia [10]. In a study conducted in Mumbai Maharashtra revealed that the percentage prevalence of severe pre-eclampsia among pregnant women is about 50% [11]. In a study done in a tertiary care hospital in Belgaum district showed that the incidence of severe pre-eclampsia among pregnant women is about 5% [10].

Eclampsia was the major cause of death and accounted for 51.1% of the hypertensive deaths [10]. There were 29.3% deaths from preeclampsia, 5% associated with chronic hypertension, 12.7% were due to HELLP syndrome and 1.3% related to rupture of the liver, while pulmonary oedema accounted for 27% of cases [6]. Cerebral causes were the final cause of death in 51.1% of maternal deaths [8].

In spite of advances in medicine, severe pre-eclampsia and eclampsia continue to remain leading causes of maternal and perinatal mortality and morbidity throughout the world [6]. Severe pre-eclampsia can lead to multiple life threatening complications like eclampsia, cerebral haemorrhage, cardiovascular complications, hepatic failure, acute renal failure, pulmonary oedema, ARDS, DIC ,HELLP syndrome, retinal detachment, cortical blindness, hypoxic cerebral damage and even maternal death .Fetal morbidities include preterm delivery, small for gestation , fetal growth restriction ,still birth , low birth weight babies are also fetal complications of severe pre-eclampsia

The incidence of severe pre-eclampsia can be reduced by better antenatal care, early recognition and prompt treatment to the women . The clinical management of severe pre-eclampsia has gone through many changes to reduce both maternal and perinatal mortality and achieve good results with the introduction of various treatment regimes. The triad of hemolysis elevated liver enzymes and low platelets poses a serious

threat to maternal and fetal outcome. Hence this study was undertaken to study the maternal and perinatal complications in severe pre-eclampsia.

In cases of severe pre-eclampsia or eclampsia most guidelines recommend expeditious delivery regardless of gestational age, in particular when pre-eclampsia is complicated by the HELLP syndrome . In recent years, as methods for monitoring maternal and fetal wellbeing improved, these guidelines have been challenged and attempts have been made to postpone delivery also in women with severe pre-eclampsia remote from term [12]. However, recent reports of small series of patients indicate that such an approach could be associated with increased maternal morbidity, attempts have been made to postpone delivery also in women with severe pre-eclampsia remote from term.

Access to perinatal care, early detection of the disorder, careful monitoring, and appropriate management are crucial elements in the prevention of severe pre-eclampsia related deaths.

The ultimate treatment for severe pre-eclampsia is to prevent potential maternal complications and to deliver the patient. However, delivery is not always in the best interest of the fetus. The rationale for delaying delivery in these pregnancies, is to reduce perinatal morbidity and mortality by delivery of a more mature fetus and to a lesser degree to achieve a more favorable cervix.

The main treatment option is to deliver the patient. The time interval from admission to delivery is a major factor influencing maternal and fetal outcomes.

Ours being a tertiary care center, that receives many complicated cases as an emergency from peripheral centre maternity clinics and nursing homes the present study is undertaken to find out maternal and perinatal outcomes in severe pre-eclampsia.

AIMS AND OBJECTIVES

- To study the maternal and fetal outcomes in severe pre-eclampsia in a teritary care centre.

REVIEW OF LITERATURE

Background:

The hypertensive disorders in pregnancy comprise several disease and Syndromes associated with high blood pressure. The disorders were called toxemias of Pregnancy, but that term is an ambiguous misnomer.

Hypertension may antedate conception or a rise during gestation, or in the early puerperium. It is often accompanied by edema and proteinuria separately or together and occasionally it culminates in convulsions and coma. The combined prevalence and incidence of the hypertensive disorders in pregnancy is probably about 6 percent, although wide variations have been alleged in relation to geographic, racial and socio economics factors.

The hypertensive disorders pose a serious threat to the fetus and to the new-born infant who is often delivered prematurely either following spontaneous labour or by therapeutic termination of pregnancy.

History of Pre-eclampsia:

The current hypothesis of pre-eclampsia is a disorder of maternal and fetal incompatibility. This is consistent with the knowledge that pre-eclampsia is more likely when there is an usually large trophoblast mass as with vesicular pregnancy, placental hydrops and multiple pregnancies and shown by **Chung et al., 1964 (Clinical Observations on some aspects of hydatiform mole journal of obstetrics and gynaecology British Commonwealth) Jeffcoate and Scott 1959 - some observation of placental factor in pregnancy toxemia American journal of obstetrics Gynaecology).** In these circumstances it is postulated that immune regulatory mechanism of normal pregnancy are overwhelmed by the size of antigenic

load of placenta, particularly as moles as androgenic in origin and this fact is reassured by Kojil and Ohamma 1977 [12].

CLASSIFICATION OF HYPERTENSIVE DISORDERS IN PREGNANCY:

GESTATIONAL HYPERTENSION

· Hypertension first time during pregnancy.

· No proteinuria

· BP returns normal twelve weeks postpartum.

PRE-ECLAMPSIA & ECLAMPSIA

· Hypertension diagnosed after 20 weeks of gestation

· Proteinuria

· Associated with other signs & symptoms of pre-eclampsia

· Eclampsia associated with seizures that cannot be attributed to other causes like space occupying lesions, seizure disorders, head injury and electrolyte imbalance.

PRE-ECLAMPSIA SUPERIMPOSED ON CHRONIC HYPERTENSION:

· New onset proteinuria in hypertensive women after twenty weeks gestation

CHRONIC HYPERTENSION:

· Hypertension before pregnancy

· Hypertension diagnosed before twenty weeks of pregnancy not attributable to trophoblastic disease or multiple pregnancy. Hypertension diagnosed 1st diagnosed after twenty weeks gestation &persistent beyond 12 weeks postpartum.

MINIMUM CRITERIA OF DEFINING SEVERE PREECLAMPSIA (ACOG) [26]

A diagnosis of severe pre-eclampsia should be entertained in women with new onset proteinuria hypertension and one or more of the following complications:

I. Severe elevations of blood pressure ≥ 160/110mmHg on 2 occasions at least 6 hours apart.

II. Proteinuria (>5g/24h)

III. Oliguria <500cc/24hrs

IV. Cerebral, visual symptoms like blurred vision, scotomata , altered mental status, severe headache.

V. Symptoms of liver capsule distention (right upper quadrant or epigastric pain)

VI. Pulmonary edema or cyanosis

VII. Thrombocytopenia (<100000 platelets / mm3)

VIII. Hepatocellular injury (serum transaminase level > 2 times normal)

IX. Fetal growth restriction

RISK FACTORS:

The most predictive risk factors of pre - eclampsia were mean arterial pressure and parity. The risk with mean arterial pressure was 8% when < 75mm Hg and 27% if > 85 mm Hg. Risk was 26% is nulliparous where as 17% in parous patients [12].

There is increased risk of pre - eclampsia in women with periodontal disease and systemic inflammation early in pregnancy with (CRP ≥ 75th percentile). Recent study demonstrated a significant association between maternal thrombophilia and severe pre-eclampsia. Maternal and paternal genetic factors increase the risk of pre - eclampsia.

Pregnancy associated:

- Chromosomal abnormalities

- Hydatidiform mole

- Hydrops fetalis

- Multiple gestations.

Maternal specific:

- Primiparity

- Age < 20 & > 35 Yrs.

- Previous pre-eclampsia

- BMI > 35

- Family history of pre-eclampsia.

- Gestational diabetes or pre existing diabetes.

- Chronic hypertension.

- Nephropathy.

- Thrombophilias.

Paternal Specific:

- First time father

- Previously fathered a pre-eclampsia pregnancy is another woman.

THEORIES ABOUT CAUSES OF PREECLAMPSIA :

1**. Immunological mechanism - BARDEQUEZ [14]**

There is immunological resistance to invading trophoblast by maternal immune system. Blocking antibodies and T helper cells, interleukins, interferon, growth factors play a major role. This results in inadequate trophoblast invasion of myometrial spiral arterioles.

2. Genetic predisposition - CHESLEY & COOPER 1986 [15]

Susceptibility is by both single gene and multifactorial inheritance.

a. Women with angiotensin gene variant T232 had increased incidence

b. There is higher incidence of factor V Leiden mutation in pre-eclamptic patients

3. Increased pressor response to angiotensinogen II - ABDUL KAREEM 1961 [16]

4.**Altered vasoactive factors : VOLHARDT 1918 [33]**

a. Endothelin - 1. A potent vasoconstrictor produced by endothelium is increased.

b. NITRIC OXIDE: A potent vasodilator is decreased. (Chang et al .)

c. Reversal of PGI2 to TXA2 and vit.E ratio

d. Vasoactive maternal factor (VMF) has been imposed to cause the endothelial changes involved in the pathophysiology of PIH.

5. Oxidants and Antioxidants

Hubal et al have confirmed that pre-eclampsia may have its origin in a disturbed oxidation mechanism. Under normal conditions equilibrium is maintained by antioxidants. With increase in severity of pre-eclampsia there is increase in lipid peroxidase and reduction in antioxidants. Vit E lipid peroxidase cause endothelial damage [34].

6.Endothelial dysfunction: HAYMAN & ASS 2000 [35]

Deficiency in trophoblastic invasion of placental blood spiral arteries leads to poorly perfused fetoplacental unit. This results in secretion of plasminogen activator inhibitor, into the maternal circulation leading to activation of endothelial cells to promote coagulation and increased sensitivity to vasopressor agents. Banker and Coll 1995 have shown that BEGF levels are increased in serum of PE which may activate endothelial cells and release of inflammatory substances [36].

7.Placental proteins

Corticotrophin releasing factor, HCG, Activin A, Inhibin A are said to play a role.

8. **Dietary deficiency: DAWSON, KELLY & Coauthors Mac Gillivray** viewed the evidence for a role of dietary deficiency in pathology of PE . It was concluded that when concentration of calcium is low in extra cellular fluid, amount of ionic calcium entering cell wall increases making vascular smooth muscle more sensitive to excitation.

9. Hyperhomocystinemia: COLLER ET AL [37]

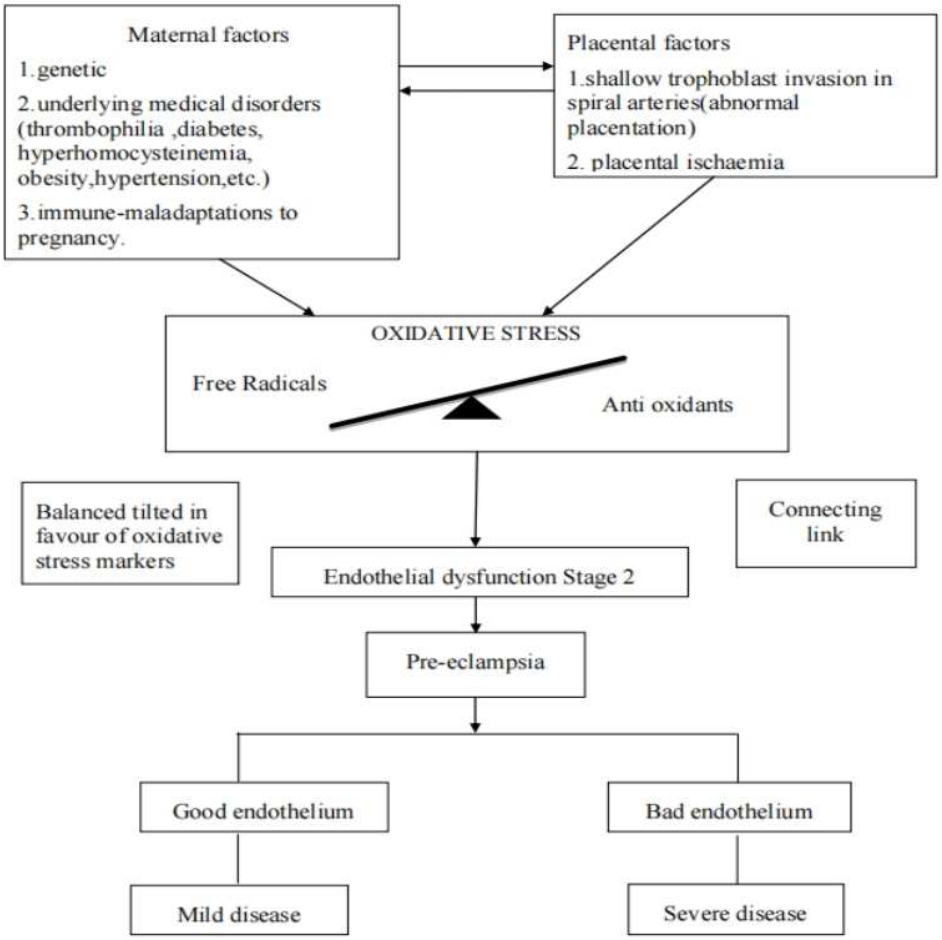
Presence of infarcts retroplacentally is said to be cause of elevated levels of circulatory homocysteine. This is due to atherosis formed at placental site.

Elevated levels damage endothelium by H202 generation depletes nitric oxide mediated detoxification of homocysteine. Elevated levels of factor V increase in prothrombin activation.

Figure 1: Risk factors in severe pre-eclampsia

Pregnancy related:	Maternal risk factors:	Couple related :
• Multiple fetal gestation • Hydropic degeneration of placenta • Hyadatidiform mole of placenta	• Extremes of age • Prior history of pre-eclampsia • Renal disease • Infection • Susceptible history	• Primiparity • Limited sperm exposure • Paternal factors

Figure 2: Genesis of Pre-eclampsia as a two stage disease



AETIOPATHOGENESIS:

The exact etiology of pre-eclampsia is unknown. Pre-eclampsia is a two-stage disorder. The theory was propounded by Redman and colleagues. According to this stage, one is preclinical and characterized by poor placentation or faulty endovascular trophoblastic remodeling of uterine arteries which causes placental hypoxia. Stage two is caused by oxidative stress which causes the release of placental factors into the maternal circulation. This in turn causes a systemic inflammatory response and endothelial activation results in clinical syndromes of pre-eclampsia and intrauterine growth restriction.

ABNORMAL TROPHOBLASTIC INVASION:

In normal pregnancy, spiral arteries of the placenta are invaded by cytotrophoblast and the elastic and muscular layers are replaced by fibrinoid. In the second trimester second wave of cytotrophoblastic invasion transforms the myometrial segments of spiral arteries into wide-mouthed vessels unresponsive to vasomotor stimuli. Blood supply is transformed from a high resistance low flow system to a low resistance high flow system to increase uteroplacental flow and meet the needs of the fetus. In pre-eclampsia primary wave of trophoblastic invasion is impaired and the secondary wave fails to occur.

ABNORMAL ANGIOGENESIS:

Angiogenesis and Antiangiogenesis factors involved in placental vascular development .There is imbalance in these factors. There excess antiangiogenic factors produced as a result of hypoxia. Trophoblast produces at least two antiangiogenic peptides in angiogenic factors like vascular endothelial growth factor [VEGF] [19].

Endothelial cell dysfunction and vasospasm:

Endothelial cell dysfunction is the most important factor in pre-eclampsia. Antiangiogenic and metabolic factors and other inflammatory factors provoke endothelial cell injury. Another theory is lipid peroxidation is stimulated by free oxygen radicals because of oxidative stress. Cytokines like tumor necrosis factor and interleukins also contribute to pre-eclampsia. It causes endothelial cell injury, modifies nitric oxide production, and interferes with prostaglandin balance. Increased capillary permeability manifests as edema and proteinuria.

Figure 3: Pathogenesis of severe pre-eclampsia:

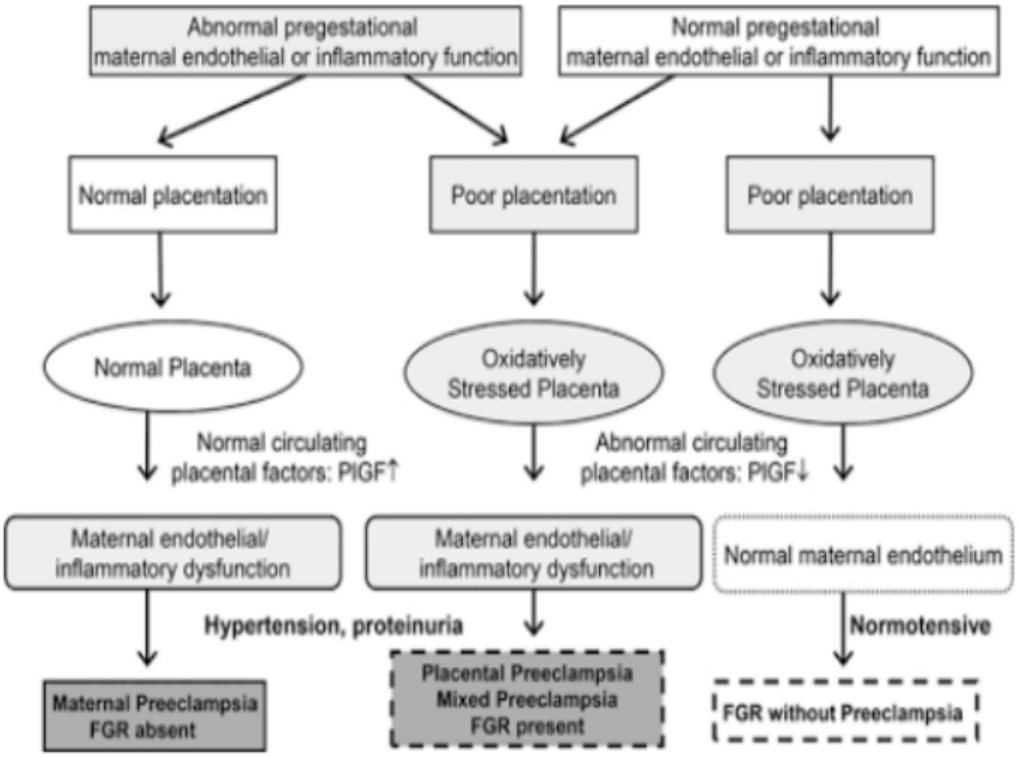


Figure 4: Trophoblastic invasion in severe pre-eclampsia

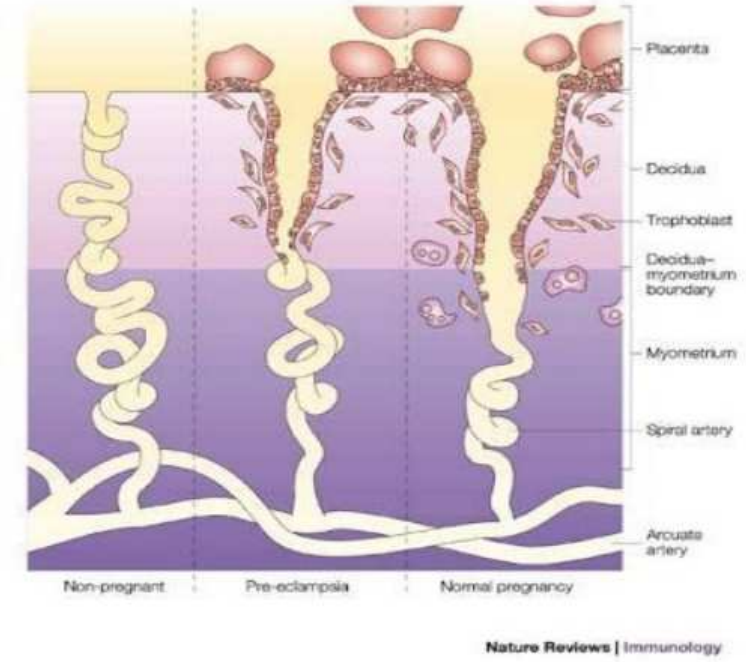
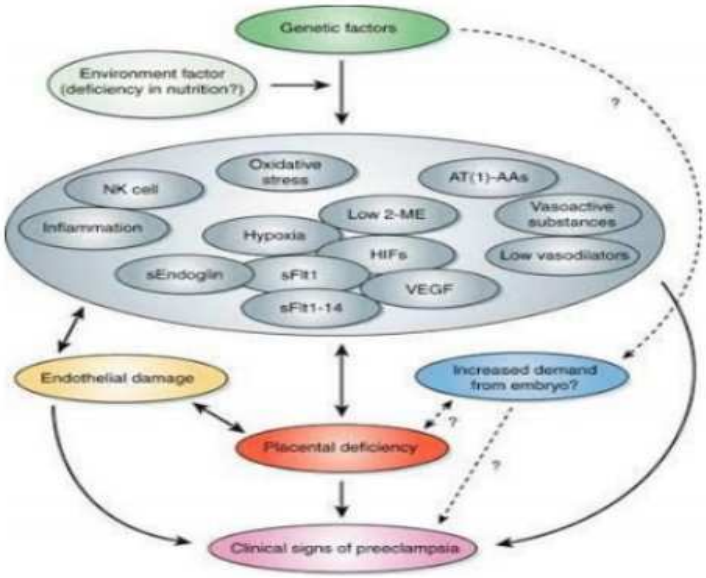


Figure 5: Factors contributing to clinical signs of severe pre-eclampsia



ALTERATION IN NITRIC OXIDE AND PROSTOGLANDINS:

Prostacyclin is a prostaglandin produced by the vascular endothelium. It is a powerful vasodilator and inhibitor of platelet aggregation. Nitric oxide is another potent vasodilator produced by the endothelium. Thromboxane is produced by platelets and causes vasoconstriction and platelet aggregation. In a normal pregnancy, there is an increased production of prostacyclin resulting in vasodilatation. Damaged endothelial cell lead to reduced production of nitric oxide. So in pre-eclampsia vasospasm and platelet activation and adhesion occurs and activation of the coagulation system also occurs [18] .

Figure 6: Immune factors and inflammatory response in severe pre-eclampsia

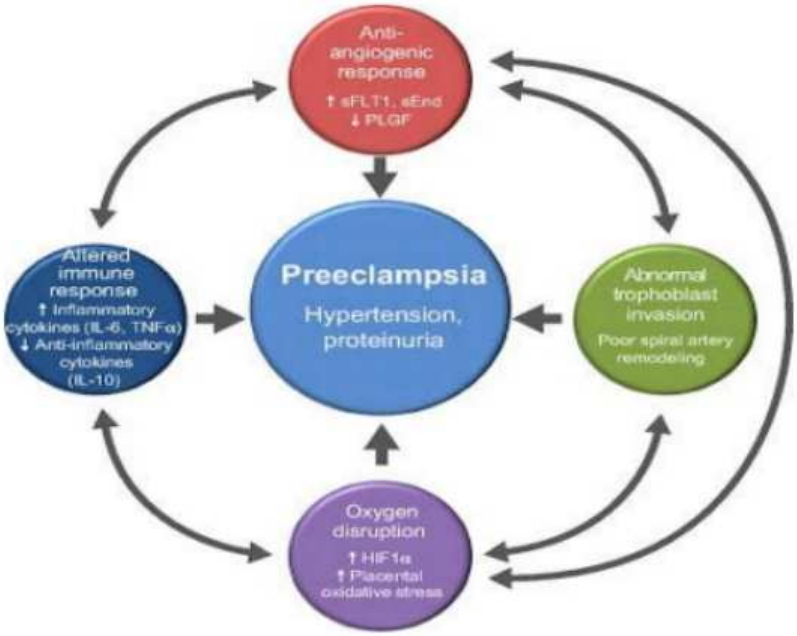
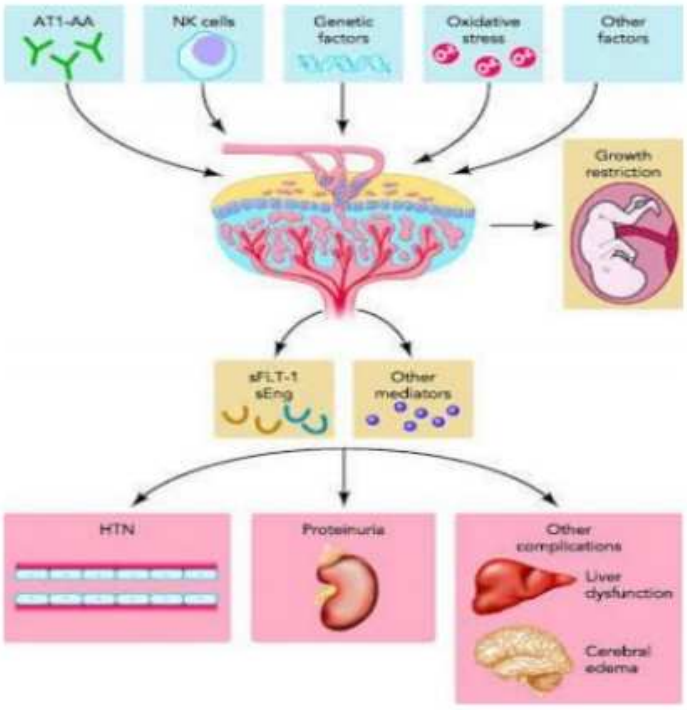


Figure 7: Factors involved in pre-eclampsia



METABOLIC FACTORS :

Central obesity and insulin resistance are risk factors for pre-eclampsia. In pre-eclampsia there is a dramatic increase in free fatty acids and triglycerides

GENOTYPE AND PHENOTYPE:

There is a definite inherited maternal component in pre-eclampsia. Phenotypes will differ among genotypes depending on interaction with environmental factors.

IMMUNOLOGICAL FACTORS:

Invasion of trophoblast into myometrium and decidua is controlled by immune mechanism. The decidua contains lymphoid tissue, predominantly natural killers .The NK cells express KIR receptors ,which recognise the LA class1 molecules. The NK cells VEGF, PLGF, and Angiotensin 2 which bring about maternal placental bed vascular changes .

MOFFET KING and colleagues studied the HLA C-NK cell receptor interaction and they stated that each pregnancy is unique because of the NK cell KIR –HLA C interaction .Mothers with absent or decreased KIRs which interact with HLA C group have increased propensity towards pre-eclampsia [17].

PATHOPHYSIOLOGY :

Changes because of vasospasm and endothelial dysfunction.

PLACENTA :

The typical vascular lesion is termed acute atherosis of the decidual arteries leads to fibrinoid necrosis, macrophages and mononuclear infiltration. Result in intra uterine growth restriction oligohydraminios , placental abruption and ultimately fetal demise.

KIDNEY :

Main pathology in kidney is glomerular and tubular dysfunction and glomerular endotheliosis and swollen endothelial cells which occurs due to fibrin deposition.Glomerular dysfunction lead to reduced glomerular filtration rate and creatinine clearance. Acute renal failure occurs usually due to acute tubular necrosis which is reversible. Rarely it lead to irreversible changes due to acute cortical necrosis. Tubular dysfunction manifest as hyperuricaemia . Proteinuria occurs due to increased capillary permeability.

LIVER :

Periportal thrombosis , fibrin deposition, hemorrhages and necrosis seen in liver. There is an increase in enzyme levels [SGOT,SGPT] and clinical jaundice can occur. Liver changes are responsible for nausea and vomiting. Small hemorrhages may coalesce to form a sub scapulars haematoma which causes stretching of glisson's capsule, and epigastric pain.Catastrophic rare complication is liver rupture. The

typical vascular lesion is termed acute atherosis of the decidual arteries leading to fibrinoid necrosis, macrophages and mononuclear infiltration resulting in intrauterine growth restriction, oligohydraminios, placental abruption and uiltimately fetal demise.

PATHOPHYSIOLOGY :

CARDIOVASCULAR SYSTEM:

Three major changes in the cardio vascular system

1. Increased cardiac afterload caused by hypertension .

2. Diminished cardiac preload due to the diminished hypervolaemia of pregnancy in pre-eclampsia .

3. Endothelial cell activation with increased capillary permeability which causes extravasation of fluid from the intravascular to extravascular space and into the lungs resulting in pulmonary edema. Hemoconcentration is the hallmark of pre-eclampsia so women with pre-eclampsia and eclampsia are sensitive to fluid therapy and easily can develop pulmonary edema .

BLOOD AND COAGULATION :

Endothelial dysfunction lead to activation of platelets and the coagulation system. By activation of tissue factor on the endothelium results in subclinical to frank DIC. Resulting in consumption coagulopathy which again result in thrombocytopaenia.This can be demonstrated by the presence of schistiocytes, Burr cells and fragmented red cells in peripheral blood and also by elevated lactate dehydrogenase levels.

BRAIN:

In the brain main pathology is cerebral vasospasm .Small cerebral hemorrhages , thrombosis and fibrinoid necrosis can occur. Cerebral edema also occurs . Massive cerebral haemorrhage may be the rare complication in severe hypertension .In CT imaging may show localized hyper intense lesions at the gray-white matter junction ,primarily in the occipital lobes. This is known as PRES .

EYES :

Retinal vasospasm is the most common finding .Haemorrhage and papilloedma are rarely seen in severe hypertension. Visual disturbances are common and are due to edema of the occipital lobe. Cortical blindness occurs rarely due to occipital edema . It is temporary. Blindness can also occur due to involvement of lateral geniculate nuclei and retina.In Retina ischaemia , infarction, or retinal detachment can occur. Prognosis is usually good and reversible following delivery.

CLINICAL MANIFESTATIONS :

Hypertension in pregnancy is generally asymptomatic and diagnosed during antenatal check up. Sudden onset, excessive weight gain, generalized edema affecting the face, hands and ankles, particularly non-dependent oedema, epigastric or right upper quadrant pain, headache and visual complaints like scotomata , blurred vision or rarely blindness in a woman with hypertension are features of severe pre-eclampsia. Symptoms of blurred vision and severe generalized or occipital headache are suggestive of accelerated hypertension and impending eclampsia. On physical examination, particular attention should be paid to the apex beat; the second sound at the aortic area may be accentuated . Ophthalmoscopic examination is an essential part

in the examination .In most women especially with mild pre-eclampsia, fundus is normal. Women with long standing preexisting hypertension ophthalmoscopic findings such as silver – wiring ,tortuosity of the arterioles and arteriovenous nipping is seen. The grave sign is the development of papilledema.

PREVENTION:

Low dose aspirin in women is at high risk for developing disease. Dietary supplements like magnesium, antioxidants, marine oils , and folic acid do not reduce the incidence of pre-eclampsia.

COCHRANE REVIEW (2012) which included over 15,000 women did not reveal any evidence of improvement of pregnancy. L-arginine calcium supplementation ,vitamin C, vitamin E β carotene was used in the prevention of severe pre-eclampsia . However the studies investigating so far are having conflicting results [31].

REST: COCHRANE REVIEW (2006) showed that there was a significant reduction in the relative risk of pre-eclampsia .

EXERCISE AND PHYSICAL ACTIVITY :

Prospective study failed to show the reduction in the relative risk of pre-eclampsia.

REDUCED DIETARY SALT :

Two trials conducted showed there was no correlation was observed.

ASPIRIN AND PLATELET AGENTS :

Aspirin is an anti-platelet aggregator so improves blood flow by preventing the formation of micro thrombi within the vessels .A large randomized control trial ,the Collaborative Low Dose Aspirin Study in Pregnancy (CLASP) .It showed a non

significant reduction of 12%in pre-eclampsia. Significant reduction of protein uric pre-eclampsia in a group of women who were at high risk of Developing early onset pre-eclampsia leading to preterm delivery, when aspirin was started early in the 2nd trimester. The study showed that low dose aspirin was generally safe for the fetus and neonate [32].

Maternal Outcome:

Women with severe pre-eclampsia are at increased risk for abruptio placenta, acute renal failure, disseminated intra vascular coagulation, cerebral haemorrhage, pulmonary edema, circulatory collapse .

Murphy and stirrat studied 71 pre-eclampsia women with gestational age less than 30 weeks and reported 21% had developed HELLP syndrome, 15% had abruptio placenta, 13% had renal failure and 1.4% eclampsia but no maternal mortality was observed [29].

Al –Mulhim et al reported that the commonest complication to be abruptio placenta [30].

Perinatal Outcome:

Perinatal outcome is usually dependent on one or more of the following:

1. Gestational age at the time of delivery.

2. Gestational age at the onset of pre eclampsia.

3. Presence of multiple gestation.

4. Severity of the disease.

5. Presence of other medical disorders.

Pre-eclampsia accounts for more than 40% of pre-mature deliveries and there is a substantial increase in the risk of low birth weight and SGA babies .

In a study performed by **odegard et al** compared 307 live singleton born to pre-eclampsia women to 619 controls, pre-eclampsia and severe pre-eclampsia were associated with a 5% and 12% reduction in birth weight respectively, and birth weight was 23% lower than expected [27].

Magee et al in a multi centric retrospective study found out that 16% of pre-eclampsia pregnancies being complicated by birth weight less than third percentile [28].

Very low birth weight (VLBW less than 1500 grams) and extremely low birth weight (ELBW, less than 1000 grams) babies often require re-admission to hospital in the first two years for respiratory infections .

EXPECTANT MANAGEMENT:

There have been 15 non randomized non controlled trials to assess the benefits of expectant management in pregnancy between 24 to 34 weeks and showed an average of 10 – 14 days prolongation of pregnancy in cases of severe pre-eclampsia without increase in maternal morbidity but these trials are not reliable as they lack randomization.

However, since 1990 only one maternal death has been reported in literature among 1677 women who underwent expectant management.

RCOG AND ACOG RECOMMENDATIONS:

AIM:-

To prolong pregnancy in severe pre-eclampsia women remote from term in order to improve perinatal outcome without increasing maternal morbidity or mortality.

PLACE:-

Management is best accomplished in a tertiary care setting with senior obstetric consultants, obstetrician gynecologists trained in management of high risk pregnancy, experienced staff. Candidates depend on a number of factors like maternal and fetal conditions as well as gestational age. There is no place for expectant management in gestational age <26 weeks as there is increase in perinatal and maternal morbidity and mortality .

Expectant Management in FGR: - studies have showed that in women with severe pre-eclampsia at 24 – 33 weeks FGR is associated with high risk of fetal mortality but does not cause maternal complications and may benefit from prolongation of pregnancy beyond 48 hours that is required for the action of steroids [26].

MANAGEMENT OF PRE-ECLAMPSIA:

Natural course of pre-eclampsia is blocked at the secondary and tertiary level of prevention. Early detection and treatment according to severity reduces the complications ,thereby reduce the morbidity and mortality, which results in better maternal and neonatal outcome. Evidence based practice and setting a protocol in the management of acute onset severe hypertension in pre-eclampsia and eclampsia improves an immense outcome.

NICE guidelines state that intravenous or oral labetalol, oral nifedipine and intravenous hydralazine may be the 1st line of management of severe pre-eclampsia [25]. Magnesium sulphate regimen to be considered in case of eclampsia and imminent eclampsia . In case of severe pre-eclampsia after 34 weeks of gestation induction of

labour should be considered. The patient is delivered by induction or cesarean section depending on the obstetric and fetal indications and the benefits of termination is weighed against potential risk of continuation of pregnancy.

If pregnancy is less than 34 weeks betamethasone 12mg 2 doses 24hours apart for the benefit of baby is given . It will accelerate lung maturity , reduce the incidence of intra ventricular haemorrhage and necrotising enterocolitis. Likewise patients with gestation of ≤34weeks with imminent symptoms , signs of multiorgan failure, non reassuring fetus, and eclampsia are delivered, similarly .

A recent review states expectant management in a patients with pre-eclampsia at a gestational age between 24 and 33 weeks is a safe and a better practice and is said to bring prolongation of pregnancy for 7 to 10 days.

Criteria for termination of pregnancy for patients on expectant management are as follows:

Ø Uncontrolled blood pressure

Ø Imminent signs and symptoms of pre –eclampsia

Ø Nonreassuring fetal cardiac status

Ø Oligohydraminios

Ø Elevated liver enzymes

Ø Oliguria

Ø Elevated liver parameters especially serum creatinine concentration

Ø Elevated liver enzymes

Ø Development of Hellp syndrome

Ø Pulmonary edema

Pre-eclampsia is an unpredictable disorder, only definite cure is termination of pregnancy. Management depends upon the severity of disease and period of gestation. If the pregnancy is 37 weeks are more elective induction of labour may be performed when particularly if association of proteinuria. Time of delivery depends upon the gestational age ,fetal lung maturity,and most importantly severity of disease.

MATERNAL SURVEILLANCE

Blood pressure should be checked at least 4 times a day. Urine albumin once in two days. Biochemical parameters including full blood count, kidney function test ,electrolytes, liver enzymes,and serum bilirubin should be checked once in two days . Ophthalmic examination to be done on admission can be repeated if required.

FETAL SURVEILLANCE:

Fetal well being can be monitored by NST and BIOPHYSICAL PHYSICAL (BPP) .NST is performed usually twice a week .In severe cases twice daily can be done weekly. Fetal growth must be monitored by ultrasonagraphy Amniotic fluid volume can be assessed periodically. Doppler studies are useful,in case of intrauterine growth restriction .It helps in deciding the frequency of monitoring and optimal time of delivery. Doppler is velocimetery method started at 28 and 30 weeks .Repeated at 2 to 4 weeks intervals.

MODE OF DELIVERY:

Preferred mode of delivery for pre-eclampsia is vaginal. Cesarean section may be indicated in cases of fetal distress , malpresentation, placental abruption or placenta previa . In case of severe pre-eclampsia remote from term caesarean section may be advisable due to the chances of prolonged and unsuccessful induction and fetal compromise .

INTRA PARTUM MANAGEMENT.

Blood pressure should be measured every two hours. Aim is to maintain the diastolic BP below 110mmhg and systolic BP below 160mmhg .Urine output and signs of impending Eclampsia to be monitored carefully.Eclampsia prophylaxis to be given in case of severe pre-eclampsia and impending pre eclampsia. Continuous fetal monitoring should be done.Adequate pain relief by Epidural anaesthesia avoids the risk of aspiration and difficult intubation due to edema of the airway.Ergometrine to be avoided because it will cause intense vasoconstriction may lead to hypertensive crisis.

MATERNAL COMPLICATIONS :

Ø Eclampsia

Ø Cerebrovascular accident

Ø Hemiplegia , dysphasia

Ø Visual disturbances

Ø Placental abruption

Ø HELLP syndrome

Ø Pulmonary edema with or without left ventricular failure

Ø Acute renal failure

Ø Microangiopathic haemolytic anaemia

Ø Side effects of drug therapy.

FETAL COMPLICATIONS

Ø Intrauterine growth restriction related to duration of hypertension

Ø Prematurity

Ø Ante partum and Intrapartum asphyxia

Ø Intrauterine death

Ø Fetal side effects of antihypertensive drugs.

HELLP SYNDROME

Hellp syndrome is an acronym which was coined by Louis Weinstein in 1982 .It includes hemolysis ,elevated liver enzymes, and low platelets.Well recognised complication of severe pre eclampsia, can occur in the absence of hypertension and proteinuria.It occurs in about 0.2 to 0.6 % of all pregnancies and in 10 to 20 % of cases with severe pre eclampsia [33] .About 2/3 of patients present antepartum period and the rest in the postpartum period, usually within 48 hours of delivery.

DIAGNOSIS.

The syndrome generally presents in the third trimester. When it occurs earlier, particularly in association with early onset pre-eclampsia, antiphospholipid antibody syndrome symptoms it may present with vague symptoms like malaise, nausea,

vomiting, epigastric pain and headache. and it may be missed until laboratory investigations are performed. The diagnosis of HELLP requires the presence of elevated liver enzymes (ALT and AST) and low platelet count. Haemolysis can be documented by examination of a peripheral blood smear (schistocytes, schistocytes, and burr cells). Elevated indirect bilirubin, Low serum haptoglobin level, a low haematocrit, and serum LDH greater than 600IU/L. The coagulation profile is usually normal unless DIC supervenes. Positive D –dimer test, which indicates subclinical coagulopathy. The differential diagnosis for HELLP syndrome

1)Acute fatty liver during pregnancy

2)Thrombotic thrombocytopenic purpura

3)Haemolytic uremic syndrome.

CLASSIFICATION

Classified into three categories based on the platelet counts.

Class 1 :<50,000/mm3.

Class2 :50,000 to <1,00,000/mm3.

Class 3 :more than 1,00,000.

Another system of classification based on number of abnormalities present i.e hemolysis, elevated liver enzymes and low platelets.when 2 or 3 present this known as Partial HELLP ,when all are present this is known as complete or full Hellp syndrome [20].

MANAGEMENT.

Maternal mortality and morbidity increase with increasing disease severity and worsening laboratory parameters. Perinatal mortality and morbidity depends on the gestational age associated complications like intra uterine growth restriction or placental abruption rather than the severity of Hellp syndrome.LDH and platelet count will be the best marker to follow the HELLP syndrome affected patients and disease progression.Anti hypertensive treatment and anticonvulsant trearment will be administered as indicated.High dose corticosteroid treatment has to be proposed to improve maternal prognosis of HELLP Syndrome [48]. COCHRANE review, ELEVEN TRIALS were included comparing the corticosteroids with placebo or no treatment [31]. There was no difference in the risk of maternal death , maternal morbidity or perinatal morbidity. Termination of pregnancy is planned according to the gestational age, the favorability of cervix and severity of condition.

When gestational age 34 weeks or more , prompt delivery as soon as the maternal condition is stabilised .

At 27 to 34 weeks , corticosteroid should be given to promote the fetal lung maturity prior to delivery. Expectant management before 34 weeks seems to be rational approach to increase fetal maturity and survival .There is no clinical trials to compare with conservative management and immediate delivery.The potential benefits have to be outweighed against the risks of expectant management, which include abruption placenta, acute renal failure, pulmonary edema , DIC, perinatal and maternal morbidity and mortality.Patients more than 34 weeks of gestational age may be induced unless there is no other contraindication.Women have past history of

HELLP syndrome carry on increased risk of at least 20% developing some form of gestational hypertension in the future pregnancy.

The following studies were reviewed and results were discussed.

- According to the study done by Neha Saxena et al proved that liver functions were deranged in 24% of our cases and renal functions were deranged in another 59%.15% of patients had low hemoglobin which was due to complications like abruption placentae, DIC and HELLP. 3% of patients also had elevated bilirubin and low platelet counts. PT INR was deranged in 12% of patients who were managed by giving FFP transfusions. Maternal complications included PPH in 38 cases followed by DIC in 18, renal dysfunction in 2, abruption in 10, HELLP in 5, pulmonary edema in 4, pulmonary embolism in 2, and maternal deaths in 4 cases. Perinatal mortality was seen in 45 cases (30%) which included IUD in 21, FSB in 9, and neonatal deaths in 15 [10].The major strength of the study was that it was a prospective study and all parameters were analyzed in detail and limitation of the study was that it was one year study and less number of patients were recruited in the study .
- A study done by Lakshmi Narayana Kota et al reported 45.8% of the perinatal mortality rate and 61.6% of the maternal mortality rate [21].The majar limitation of the study was that only maternal and perinatal mortality were studied in detail .
- A cross-sectional study done by Pandya jay et al showed that 312(5%) pregnant women developed severe preeclampsia and 33 women developed HELLP syndrome (10.57%). The mean age was 25.18 years. The most common indication for LSCS was HELLP 81.82%. The mean birth weight was 2.27 kg. The requirement of NICU admission was noted in 18.18% and the most common cause of NICU admission was Meconium aspiration syndrome with fetal

distress(50%). The perinatal mortality rate was 16.67% [11].The major limitations of the study were that only a smaller subset of the proportion of women with HELLP Syndrome which limited them to determining the significant risk factors and it was a single-center study design hence results were not generalized to entire population.

- A retrospective study done by Shobana .S.Pillai showed that 42% of the cases were in the age group of 26-30 years, nearly 61% were primigravidae and the majority (64) were referred from peripheral hospitals. Liver function tests were deranged in 19% of the patients and 17% had an abnormal renal function. Nifedipine was the most commonly used antihypertensive and magnesium sulfate was the anticonvulsant used in all the cases. Lower segment cesarean section was the mode of delivery in 64.5% of the cases. The commonest maternal complication was atonic PPH. There was no maternal mortality but there were 3 maternal near-miss cases due to DIC. 65% of the cases had a preterm delivery and 39% of the babies needed NICU admission. There were 10 neonatal deaths [22].The limitation of the study was, it was a retrospective study where there are more chances for inappropriate and wrong details of the study population, which may invariably affect the result of the study.

- In one of the prospective studies done at Mansoura university which included 204 patients with mean age, BMI, and gestational age at delivery of 28±7 years, 36±7 kg/m2, and 34±4 weeks respectively, while the median gravidity and parity were 2 and 1 respectively. Regarding maternal outcomes, the percentages of eclampsia, placental abruption, acute renal failure, ICU admission, DIC, HELLP syndrome, pulmonary edema, and postpartum hemorrhage (PPH) were 19.6%, 7.8%, 4.9%,

15.7%, 0.5%, 8.3%, and 3.4% respectively. Concerning fetal outcomes, the percentages of FGR and IUD were 13% each. In addition, neonatal outcomes revealed that the percentages of neonatal need for oxygen after delivery and NICU admission were 67% and 62% respectively. 69% of live-born babies were delivered preterm [23]. The strength of the study was that all maternal and perinatal outcomes were studied and analyzed in detail. The limitation of the study was the study design of the study.

- A prospective study was done at Andra medical college by U. Sujatha Patnaik et al, which included 120 antenatal women with severe pre-eclampsia. Most of them (70%) were between the ages of 20-30 years. 10% were in the age group of >30%. The majority (79%) of our patients were primigravida. In severe pre-eclampsia, 81.66% delivered at gestational age <37 weeks Prematurity was seen in 70% of women with severe pre-eclampsia. Fetal growth restriction was seen in 20% of cases, low birth weight was seen in 80% of cases and IUFD was seen in 5% of cases.The common complication seen in severe pre-eclampsia patients were eclampsia (9.1%), liver dysfunction (5%) post-partum hemorrhage (5.8%) and HELLP syndrome with DIC (0.83%). Maternal deaths 2% of severe preeclampsia were due to pulmonary edema (ARDS) [24] .The major limitation of the study was that the maternal charateristics of the study

  population was not analyzed in detail .

MATERIALS AND METHODS

Study setting:

"Dr.Prabhakar Kore Hospital and MRC", Belagavi attached to KAHER's Jawaharlal Nehru medical College, Belagavi

Study design:-

Prospective observational study

Study Period :-

1st January 2021- 31st December 2021

Duration Of Data Collection:-

1 Year.

Source of data :

Pregnant Women with severe pre-eclampsia i.e with blood pressure of more than or equal to 160/110 mmhg with or without proteinuria with or without imminent signs satisfying inclusion and exclusion criteria admitted in labor room at tertiary care centre were included in the study .

Selection criteria:

Inclusion criteria:

- Singleton Pregnancy
- Pregnant women between 20-42 Weeks of Gestation admitted in labor room
- Primigravida and multigravida with blood Pressure ≥160 Mm Hg systolic And or ≥110 mmhg diastolic with or without proteinuria and with or without imminent signs like headache, oliguria , visual disturbances , persistent epigastric pain , altered liver function and thrombocytopenia (<1,00,000 lakh)

Exclusion Criteria:

- Known Case of Essential Hypertension
- Known Case of Renal Disease
- Gestational Hypertension
- Diabetes Mellitus
- Cardiac Disease in pregnancy
- Medical disorders like epilepsy , cardiac disease , thyroid disease etc

Ethical clearance:

Approved by "Ethical and Research committee, KAHER's Jawaharlal Nehru Medical College" Belagavi, prior to its commencement.

WAIVER OF CONSENT :

Waiver of consent was sought and obtained as all data was procured throught the patient record , before the commencement of study .

Sample size:

Sample size calculation based on prevalence is

$$\frac{z_{\alpha} P(1-P)}{d^2}$$

where P is the percentage of prevalence and d is the percentage likely difference in the prevalence.

zα is linked with the level of significance.

For 5% level of the significance $z\alpha = 1.96$.

With P = 69.39% and d = 10% of P, the sample size is 75

To make the study more confirmatory, the sample size has been raised to 116

Since the present study is an observational study, there will be a single group with the above sample size

Method of data collection:

All antenatal women diagnosed with severe pre-eclampsia, as per inclusion criteria and institutional protocol for diagnosis of severe pre-eclampsia were included in the study.

On admission, detailed demographic, personal, medical, obstetric and family history was taken from the patient or her attended. General physical examination and systemic, abdominal and pelvic examinations were done. Data regarding blood pressure monitoring were collected . Date regarding the investigations like complete blood count , platelet count ,liver function ,renal function test and DIC profile were also collected. After assessment and stabilization of the women, the fetal condition was evaluated with the help of FHR recording, CTG tracing, and ultrasound. The decision regarding timing and mode of delivery were individualized. Corticosteroids were administered if the gestational age was less than 34 weeks gestation. Magnesium sulfate was given in eclamptic patients and patients with severe pre-eclampsia with the imminent sign of eclampsia. Obstetric management was done according to maternal and fetal conditions. The decision to deliver the patient vaginally (either spontaneous or induced) or by cesarean section was individualized and decided as per the hospital protocol and emergency medical team. The patient with uncontrolled hypertension was managed with the help of a physician and anesthetist. The outcome

of each pregnancy was obtained by examining the patient in the labor ward and the neonates in neonatal intensive care unit with the help of paediatrician.

Maternal outcomes were assessed in terms of complications like DIC, eclampsia , acute liver failure , acute kidney injury , pleural effusion ,ascites , abruption , retinal detachment ,pulmonary edema , ICU admissions and maternal death.

Perinatal outcomes were studied in terms of apgar score (score less than 7 was considered as low apgar score) , intrauterine fetal death , birth weight , prematurity , FGR ,NICU admission and perinatal death (from time of delivery upto 7 days of life) were studied.

At the end of the study all the above mentioned date was compiled and analysed .

Statistical analysis:

Since the study is of observational study the plan of analysis will be as follows.

For the continuous quantitative variables mean and standard deviation will be calculated. For the purpose of comparison if the data is divided into two groups with respect to certain qualitative characteristic, the continuous variables will be compared using suitable tools of statistics like student's unpaired t test. The pre and post treatment measures will be compared using student's paired t test

Discrete variables will be represented by median.

The categorical data will be expressed in terms of rates, ratios and percentages. The association between the outcome, clinical and demographic characteristics will be tested using Chi-square test, test of proportion or Fisher's exact test.

For discrete variables non parametric tests will be used.

Apart from the above suitable tools like ANOVA, correlation, regression will be used according to the need.

Suitable graphs will be used to depict the comparison.

In total of 116 women with severe pre-eclampsia were analyzed.

RESULTS

A prospective observational study was carried out on women admitted in labor room and diagnosed as severe pre-eclampsia at Dr.Prabhakar Kore hospital &MRC attached to KAHER'S JNMC during the period from January 2021 to December 2021.

Figure 8. Strobe diagram

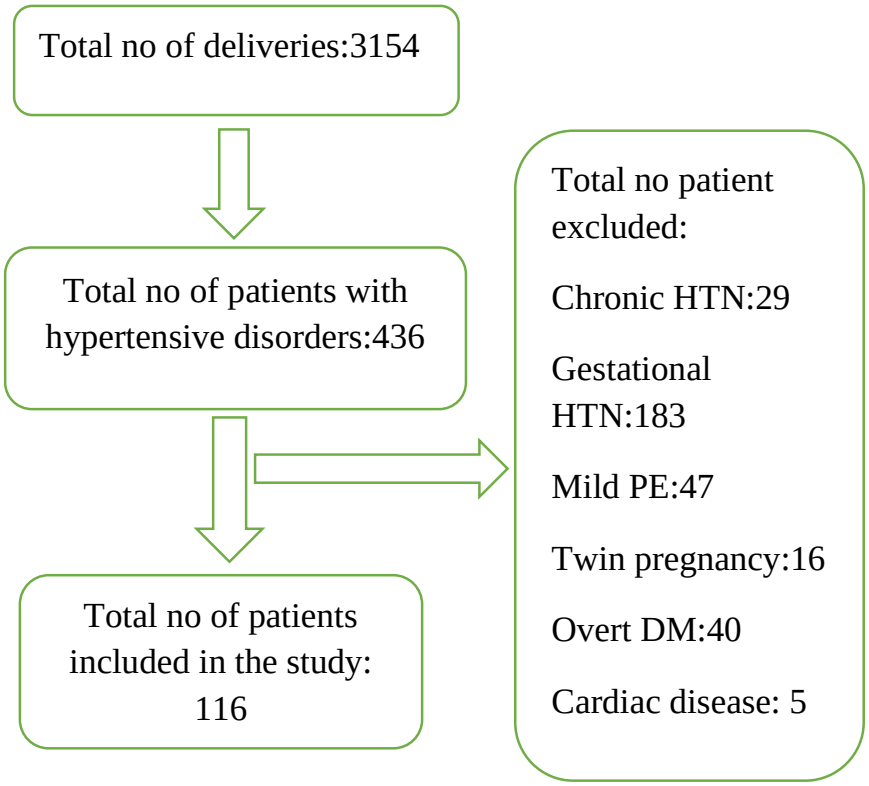


Table 1.Prevalence of severe pre-eclampsia in the current setup

Category	N=3154	%
Non severe pre-eclampsia	2977	94.3%
Severe pre-eclampsia	177	5.7%

In the current setup total number deliveries in one year was 3154 , in which 177 patients were severe pre-eclampsia . The prevalence of severe pre-eclampsia in the current setup accounts to be 5.7%

Figure 9.Prevalence of severe pre-eclampsia in the current setup

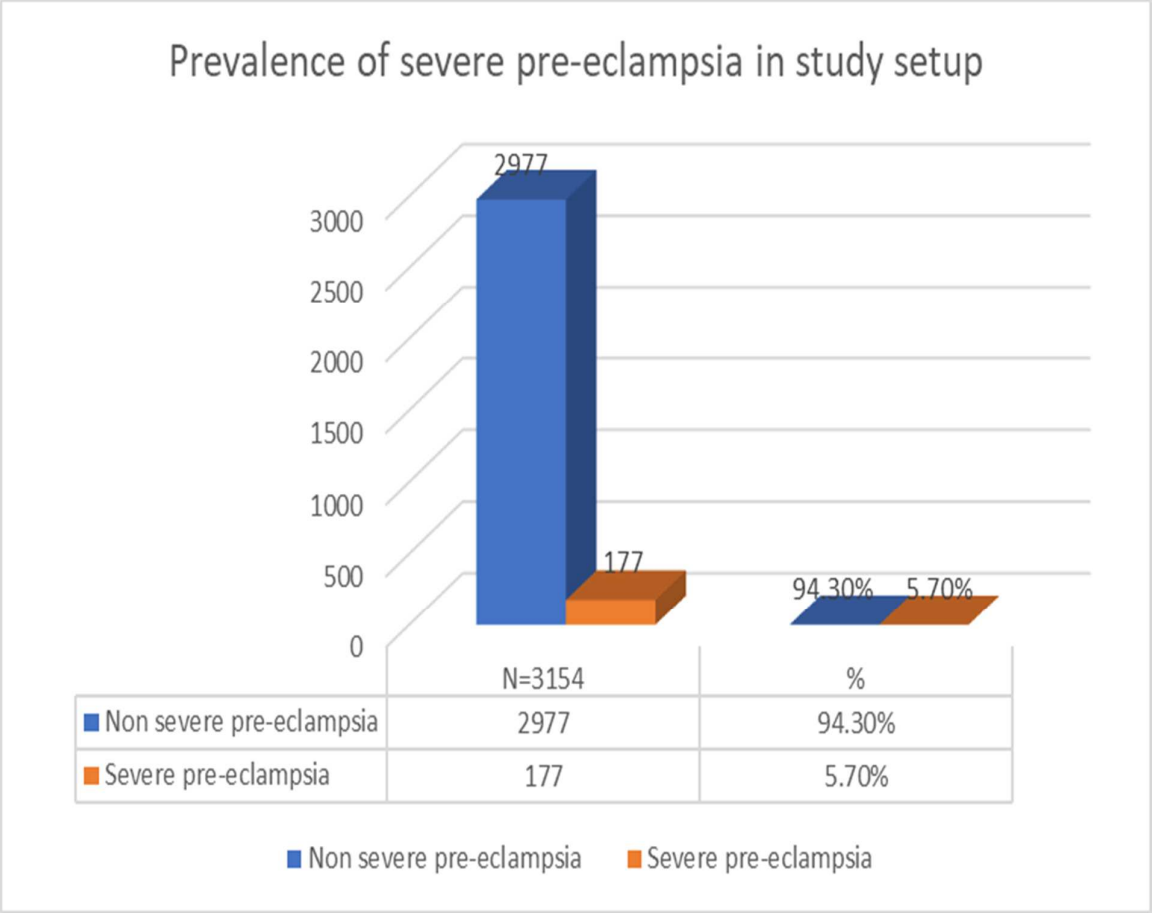


Table 2. Prevalence of severe pre-eclampsia in hypertensive disorders:

In the present study, there were a total of 3154 deliveries in which 436 women were screened. In which 29 patients were chronic hypertensive, 183 patients had gestational hypertension, 47 patients were mild pre-eclampsia and 177 patients were severe pre-eclampsia

Hypertensive disorders	N= 436	%
Chronic hypertension	29	6.6%
Gestational hypertension	183	41.9%
Mild pre-eclampsia	47	11%
Severe pre-eclampsia	177	40.5%

The prevalence of severe pre-eclampsia in the current setup was found to be 40.5%. In the total no of severe pre-eclampsia patients screened, 16 patients were twin pregnancy, 40 patients were overt DM and 5 patients had cardiac pregnancy and were excluded from the study. Hence a total 116 patients were included in the study and data were studied.

Figure 10. Prevalence of severe pre-eclampsia in hypertensive disorders

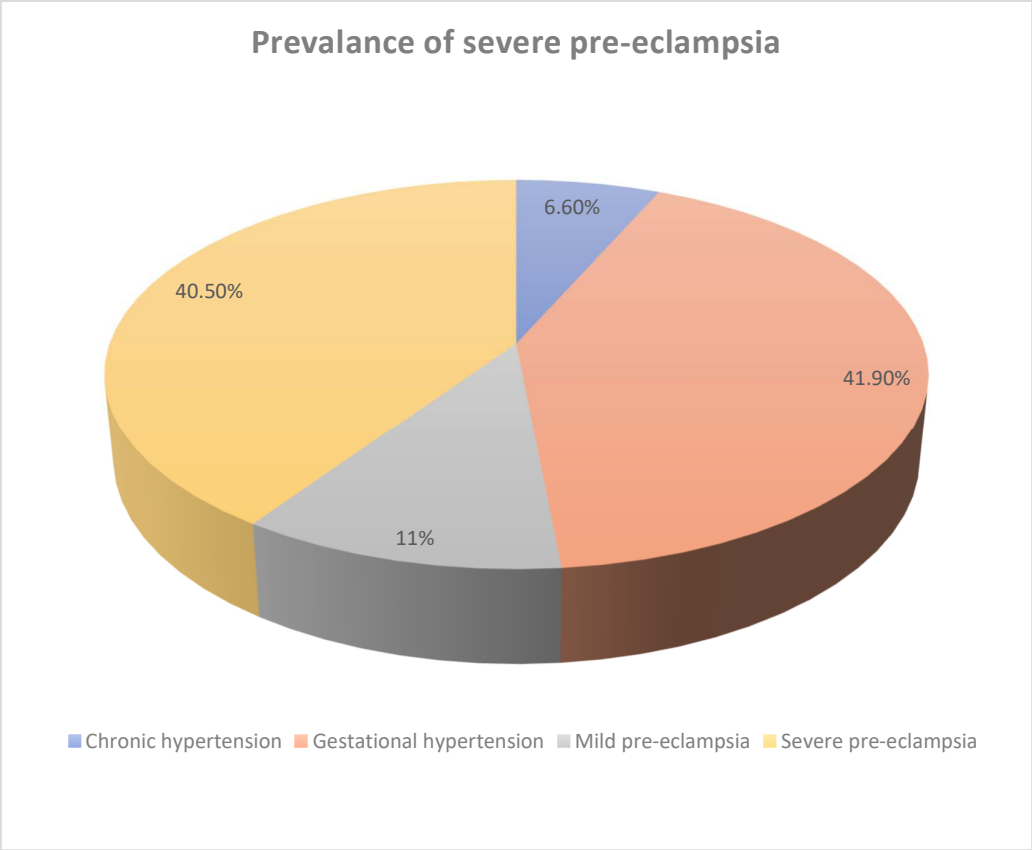


Table 3. Distribution of study participants in women according to age group:

Category	Sub-category	Number(n=116)	Percentage
Age	18-20	13	11.2%
	20-25	45	38.7%
	26-30	43	37%
	31-35	10	8.6%
	>35	05	4.5%

Most of the study population i.e 45 participants were in the age group of 20-25 years with the mean age being 25.87 years with standard deviation of 4.8 years showing that severe pre-eclampsia is more common in the younger age group. The maximum age observed was 41 years and the minimum age observed was 19 year and 84% women were from rural area

Figure 11: Distribution of study population according to age group

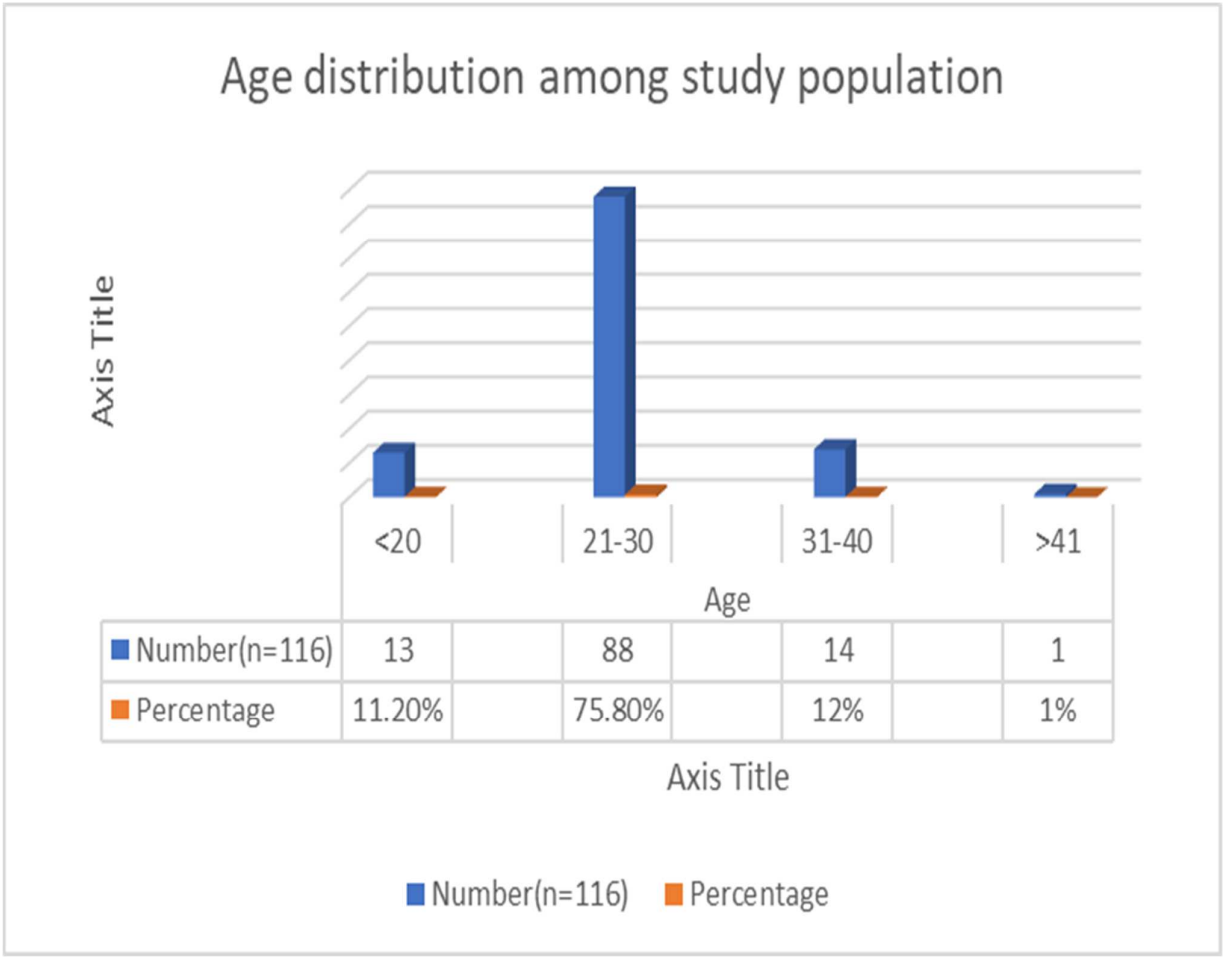


Table 4. Distribution of study population according to gravida status

Category	Sub-category	Number(N=116)	Percentage
Gravida status	Primigravida	59	51%
	G2	30	26%
	G3	21	18%
	More than gravida 4	06	5%

Our study shows among women with severe pre-eclampsia 51% were primigravida and 57 patients were multigravida.

Figure 12. Distribution of study population according to gravida status

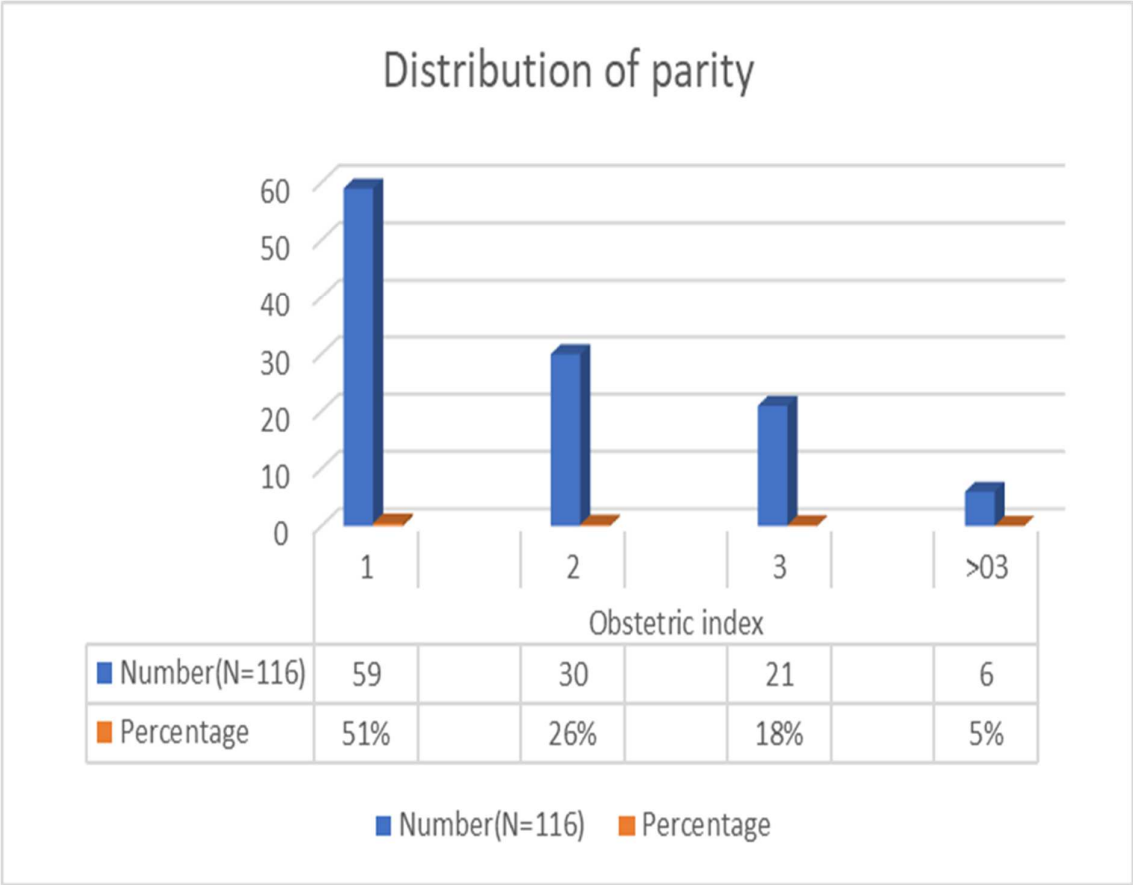


Table 5. Distribution of study population according to gestational age at diagnosis

Gestational age category	N=116	%
<20weeks+6 -24^{+6}	01	1%
25 – 28^{+6}	10	9%
29- 32^{+6}	20	17.2%
32-36^{+6}	38	32.7%
37-40	44	38%
>40 weeks	03	2.1%

In present study, most of the women (38%) were diagnosed at gestational age of around 37 weeks-40 weeks The mean gestational age at diagnosis was 35.32weeks with standard deviation of 3.7weeks. The maximum gestational age observed was 41 weeks and the minimum observed was 24 weeks.

Figure 13. Distribution of study population according to gestational age

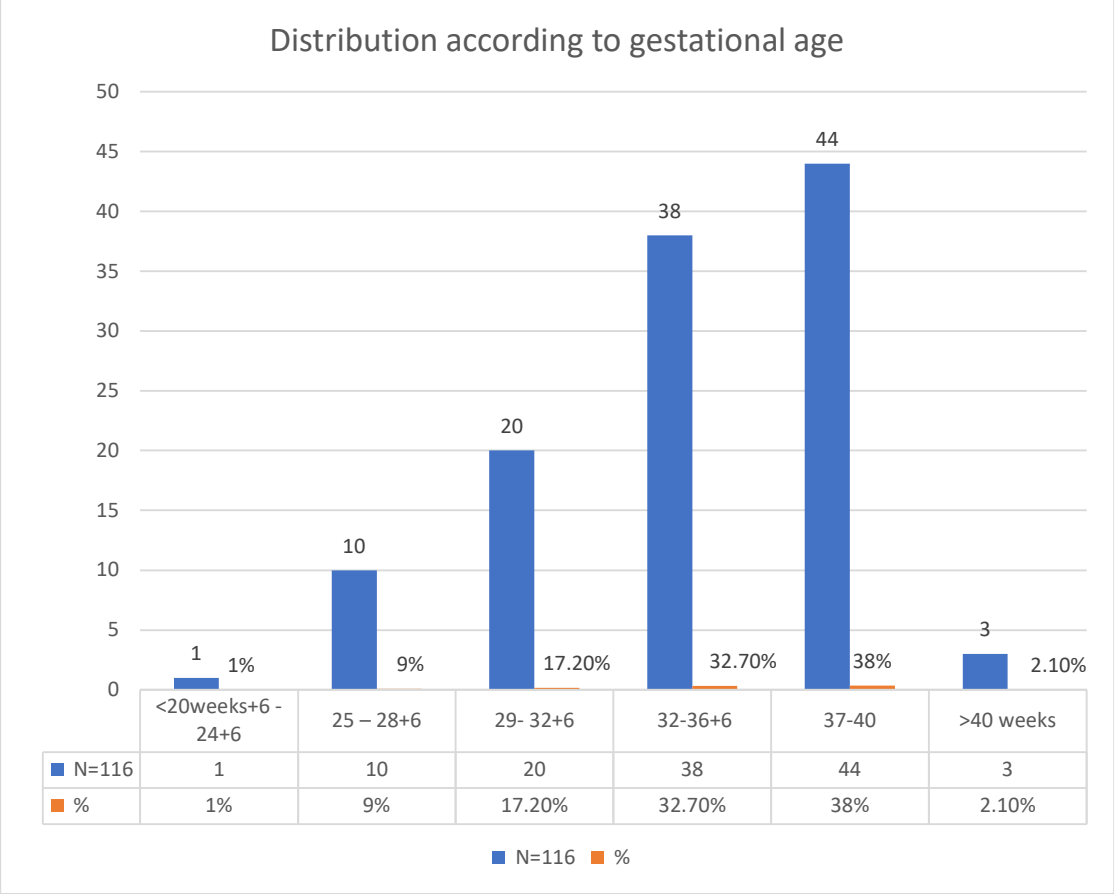


Table 6. Distribution of study population according to registration status:

Category	Sub-category	N=116	%
Registration status	Registered	32	27.5%
	Unregistered	84	72.5%

In the current study setup, majority of the women i.e 72.5% were unregistered cases.

Figure 14. Distribution of study population according to registered cases

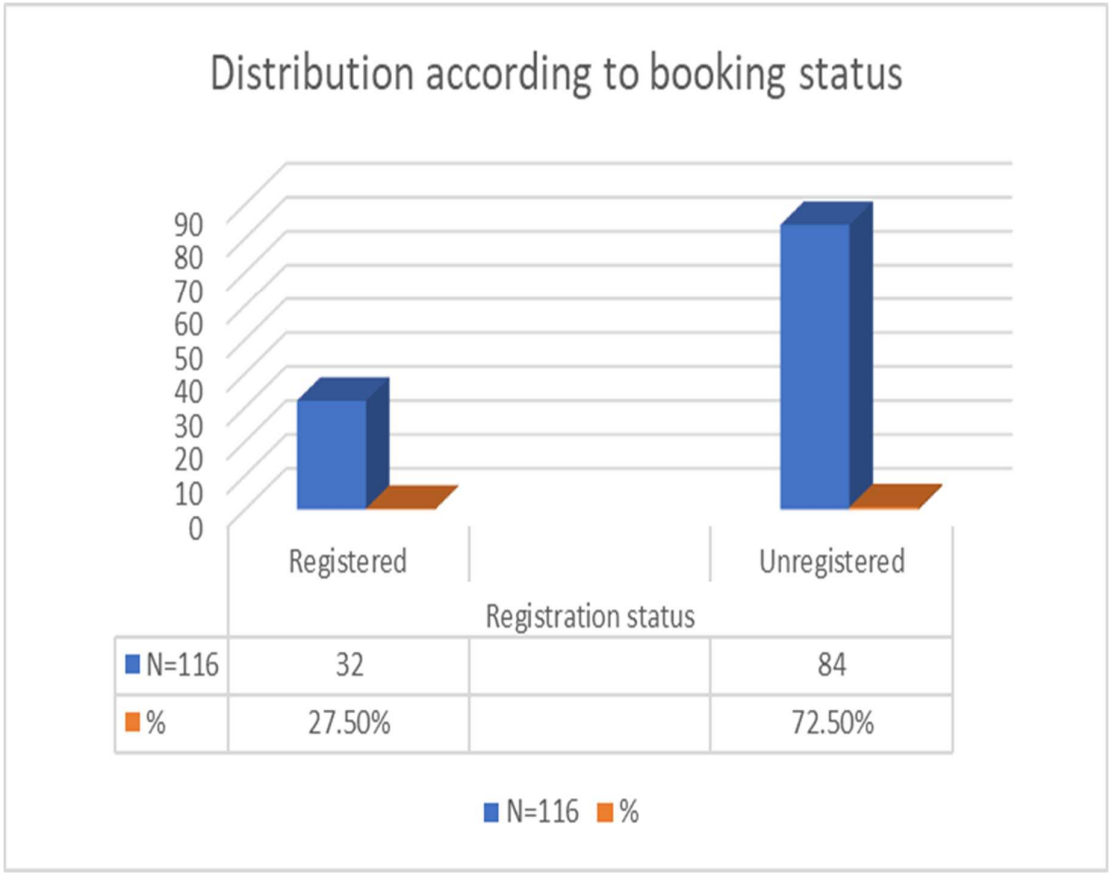


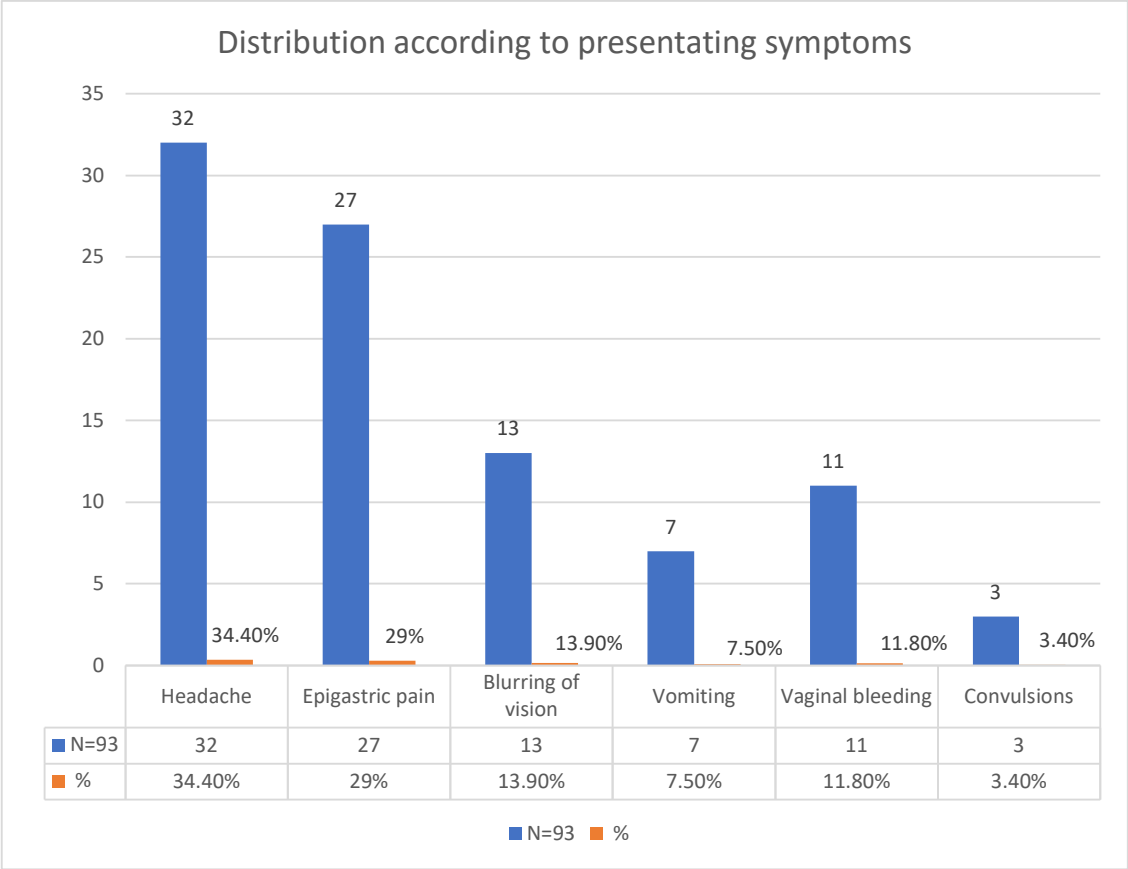
Table 7. Distribution of study population according to presenting symptoms

Presenting features	N=93	%
Headache	32	34.4%
Epigastric pain	27	29%
Blurring of vision	13	13.9%
Vomiting	07	7.5%
Vaginal bleeding	11	11.8%
Convulsions	03	3.4%

In the present study, 83 patients presented with symptoms and rest 33 patients did not have any symptoms. In the 83 patients around 10 patients had presented with more than one symptoms.

In present study group majority of women presented with the complaint of headache 34.4%, followed by epigastric pain 29%, visual defects 13.9 % and vomiting 7.5 %

Figure 15. Distribution of study populationaccording to presenting symptoms



Laboratory parameters:

Table 8.Distribution of study population according to urine albumin levels

Urine albumin	N=116	%
</1+	27	23%
>/2+	47	41%
>/3+	42	36%

In the present study, 41% of patients had an albumin count of more than 2+ and 36% patients had an albumin count of more than 3+.

Figure 16. Distribution of study population according to urine albumin levels

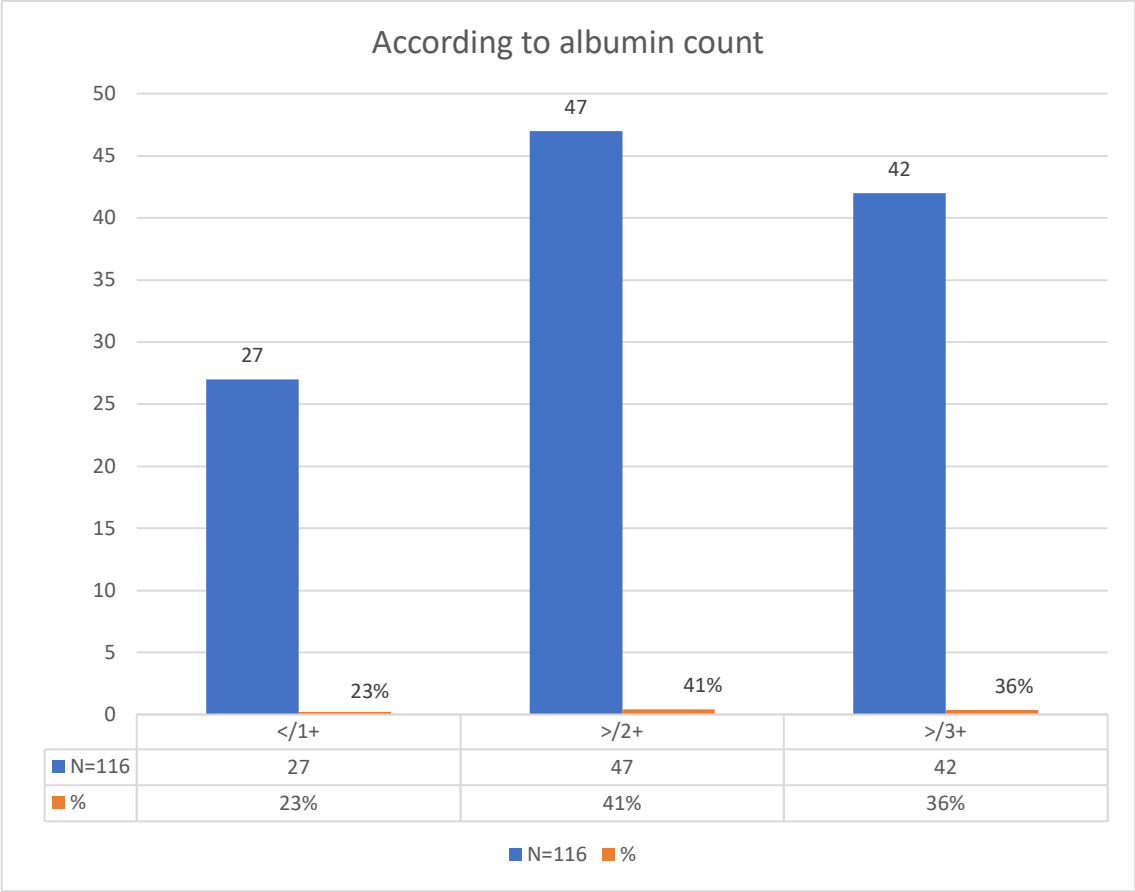


Table 9. Distribution of study population according to platelet count

Platelet count per microliter	N=116	%
<50.000	09	8%
>51.000-1 lakh	09	8%
>1 lakh- 1.5 lakh	48	41%
>1.5 lakh	50	43%

In the current study , according to Mississipi classification, out of 116 patients 9 patients(7.7%) had a platelet count of less than 50,000 , 9 patients(7.7%) between 50,000 to 1 lakh , 48 patients(41%) had a 1 lakh to 1.5lakh and 50 patients(43%) more than 1.5 lakh with a mean platelet count was 1.81 with standard deviation of 0.829

Figure 17. Distribution of study population according to platelet count

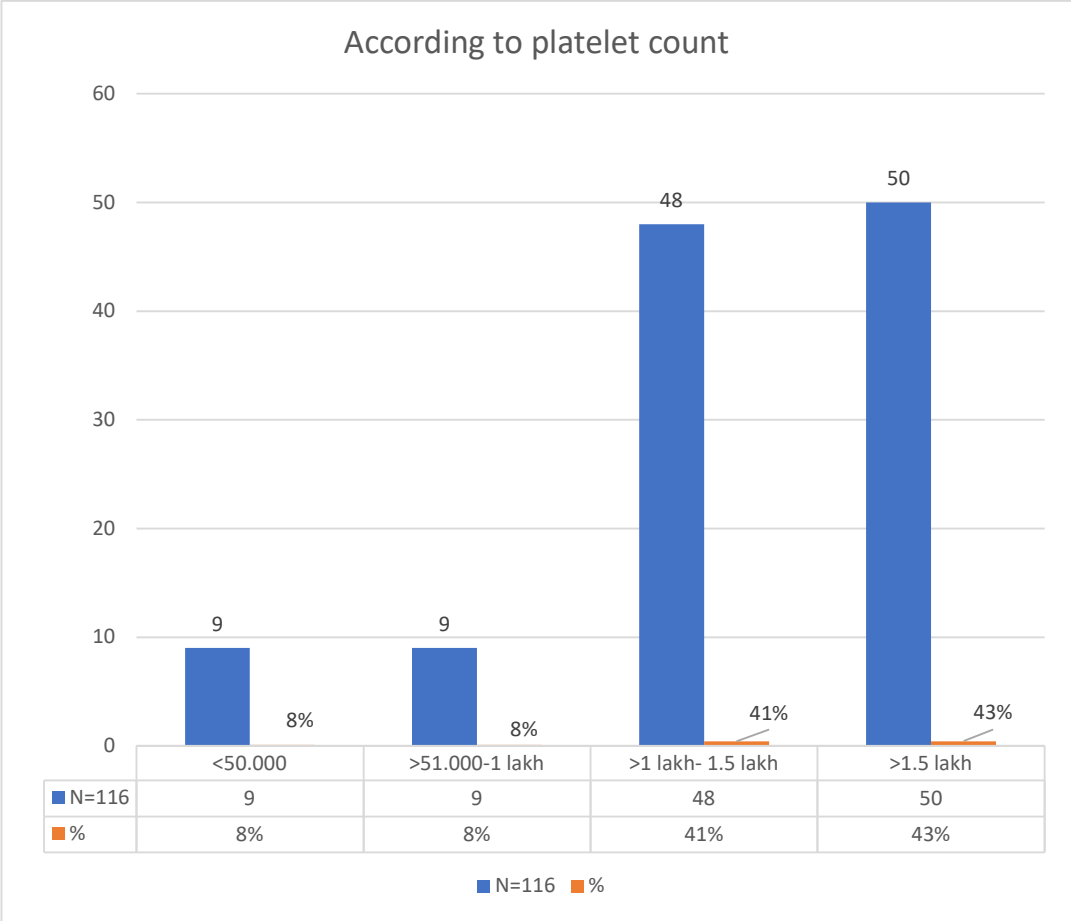
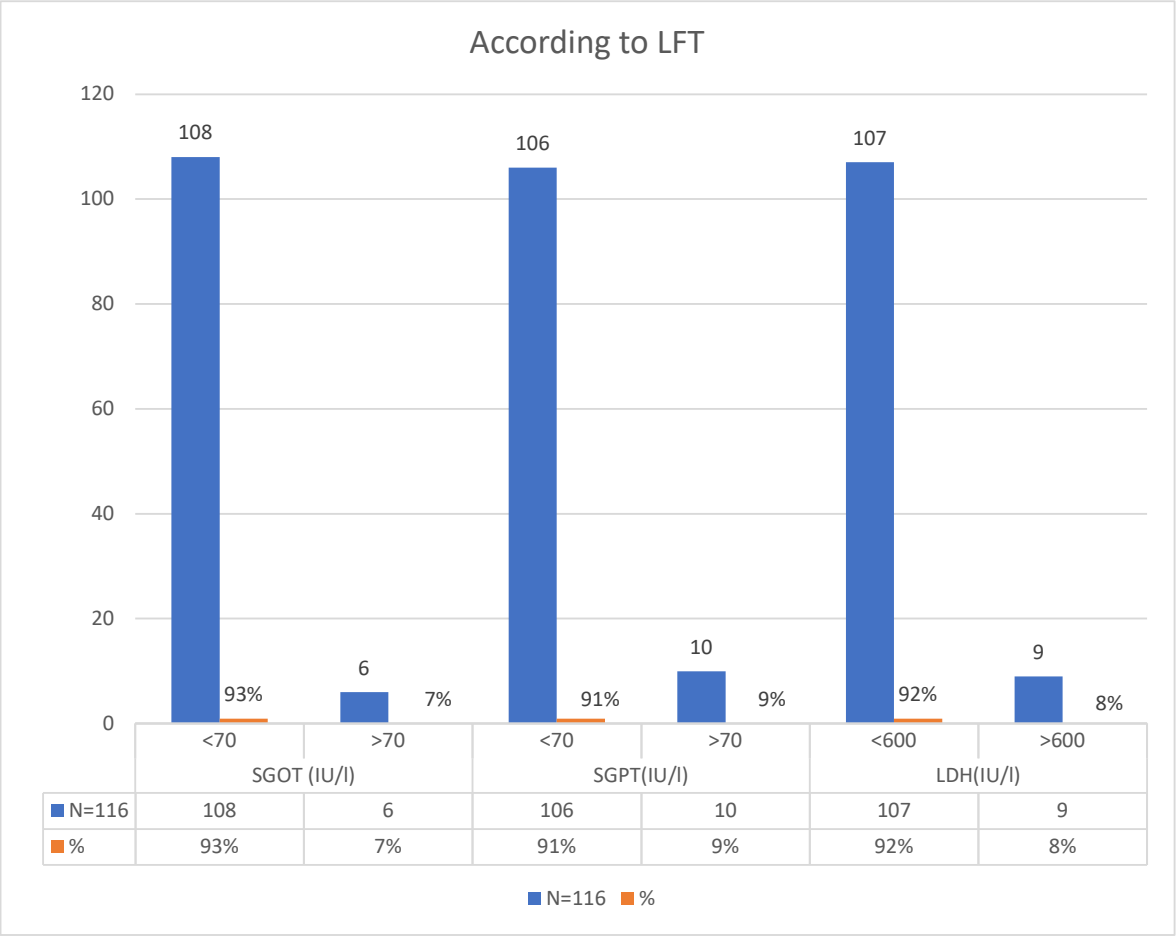


Table 10.Distribution of study population according to LFT

LFT	Levels	N=116	%
SGOT (U/l)	<70	108	93%
	>70	06	07%
SGPT(U/l)	<70	106	91%
	>70	10	09%
LDH(U/l)	<600	107	92%
	>600	09	08%

According to Mississipi classification,108 patients(93%) had AST levels less than 70 and 8 patients(6.8%) had more than 70 . 106 patients (91%) had ALT levels of less than 70 and 10 patients(8.6%) had a count of more than 70 .107 patients (92%) had LDH levels of less than 600 and 9 patients (8%) had LDH levels of more than 600. In the present study it was observed that, the mean SGOT was 39.58 with standard deviation of 49.33, mean SGPT was 33.08 with standard deviation of 50.86 and a mean LDH 396.7 with standard deviation of 275.6.

Figure 18. Distribution of study population according to LFT



N=116	108	6	106	10	107	9
%	93%	7%	91%	9%	92%	8%

Table 11. Distribution of study population according to RFT

RFT	Levels	N=116	%	Mean	S.D
Blood urea (mg/dl)	<40	112	96%	**23.25**	**3.18**
	>40	04	04%		
Serum creatinine (mg/dl)	<0.8	112	96%	**1.09**	**0.23**
	>0.8	04	04%		
Uric acid levels (mmol/l)	<7.8	102	88%	**1.64**	**6.2**
	>7.8	14	12%		

In the correct study it was observed that around 18% of study population had a deranged renal function and mean uric acid with standard deviation of 1.64 was 6.2 , mean urea level of 23.25 with a standard deviation of 3.18 and mean creatinine level of 1.09 with standard deviation of 0.23.

Figure 19.Distribution of study population according to RFT levels

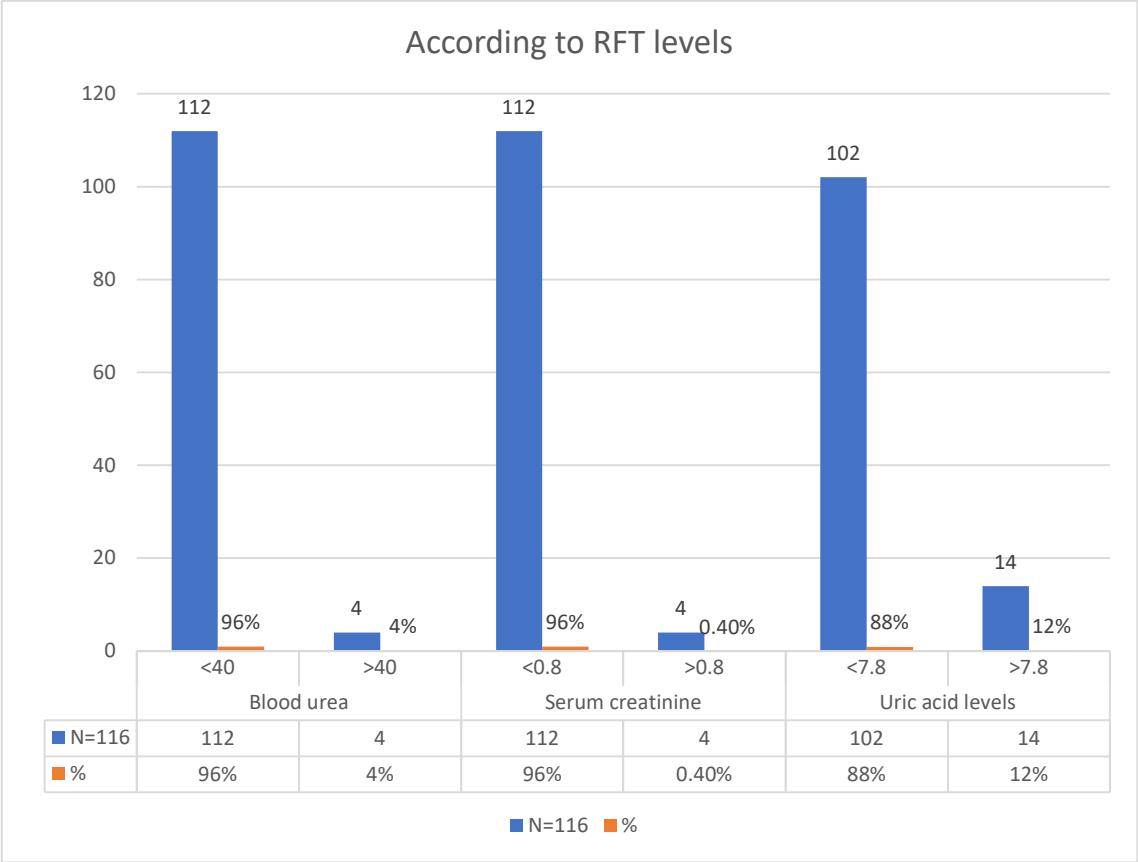


Table 12.Distribution of study population according to DIC profile:

DIC profile	Levels	N=116	%	Mean	S.D
PT	<15.6 >15.6	114 02	98% 02%	**14.22 ± 1.1**	**1.1**
INR	<1.2 >1.2	113 03	97% 03%	**1.03**	**0.5**
APTT(seconds)	<12.7 >12.7	115 01	99% 01%	**30.6 ± 6.39**	**6.49**
D-dimer (ng/dl)	<2000 >2000	113 03	97% 03%	**1079±2.36**	**0.6**
Sr.Fibrinogen (ng/dl)	>160 <160	113 03	97% 03%	**300**	**1.2**

In the current study it was observed that around 3% of the study population had a deranged DIC profile.

Figure 20.Distribution of study population according to DIC profile

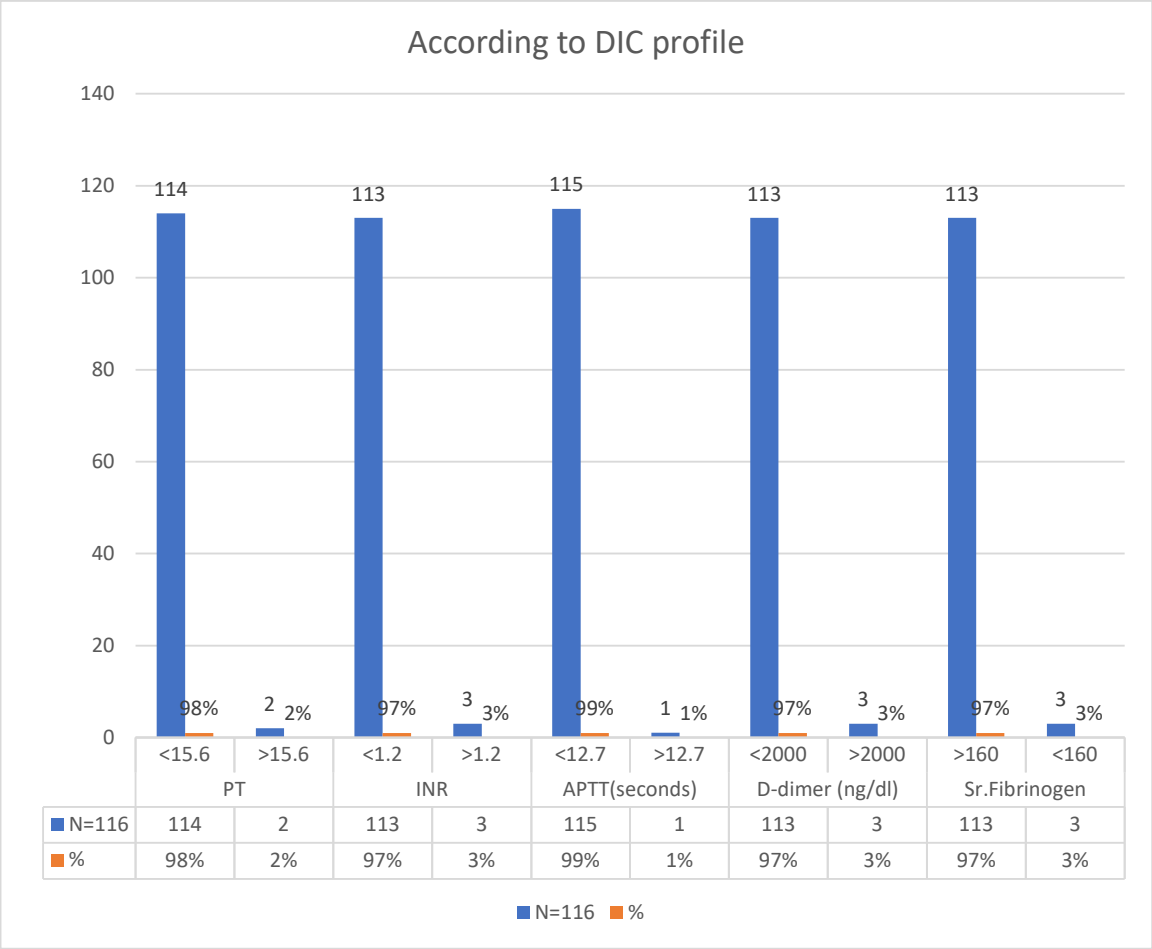


Table 13.Distribution of study population according to fundoscopy changes

Fundoscopy changes	N=09	%
Grade 1 papilledema	06	67%
Grade 2 papilledema	03	33%

In the present study , out of 116 cases 9 patients had fundoscopy changes in which 67% of the study population had grade 1 papilledema and 33% of study population had grade 2 papilledema

Figure 21.Distribution of study populationaccording to fundoscopy changes

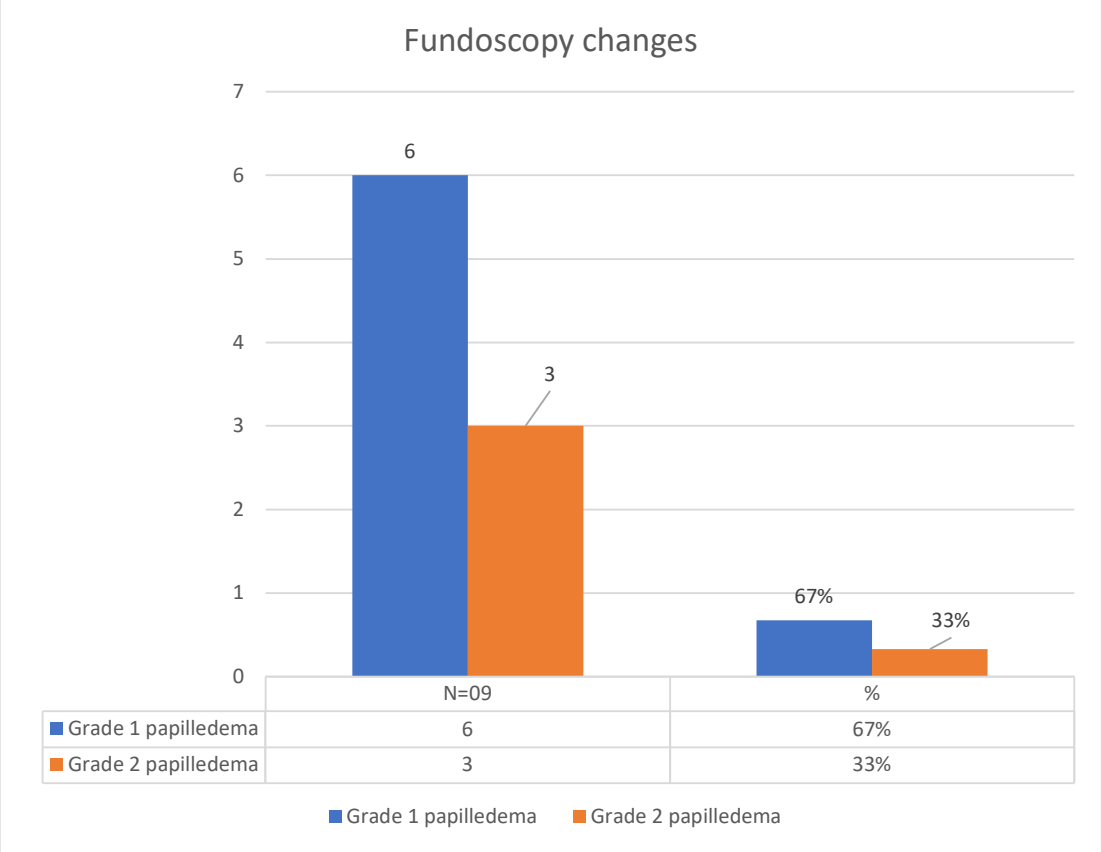


Table 14.Distribution of study population according to mode of management

Category	Sub-category	N=116	%
$Mgso_4$ administration	Given	13	11%
	Not given	103	89%
Anti-hypertensive given	Nifedepine	90	76%
	Labetalol Intravenous	15	15%
	Nifedepine+Labetalol	11	09%
Steroid administration	Given	50	43%
	Not given	66	57%

Out of 116 cases , 13 patients i.e 11% were given $Mgso_4$ .Nifedipine was the most commonly used drug in the present study i.e 90 participants (76%), either singly or in combination. IV labetalol was given in around 9% of study population on admission. Labetalol was also used in combination with nifedipine . Antenatal steroids were given in 50 participants (43%)

Figure 22. Distribution of study population according to mode of management

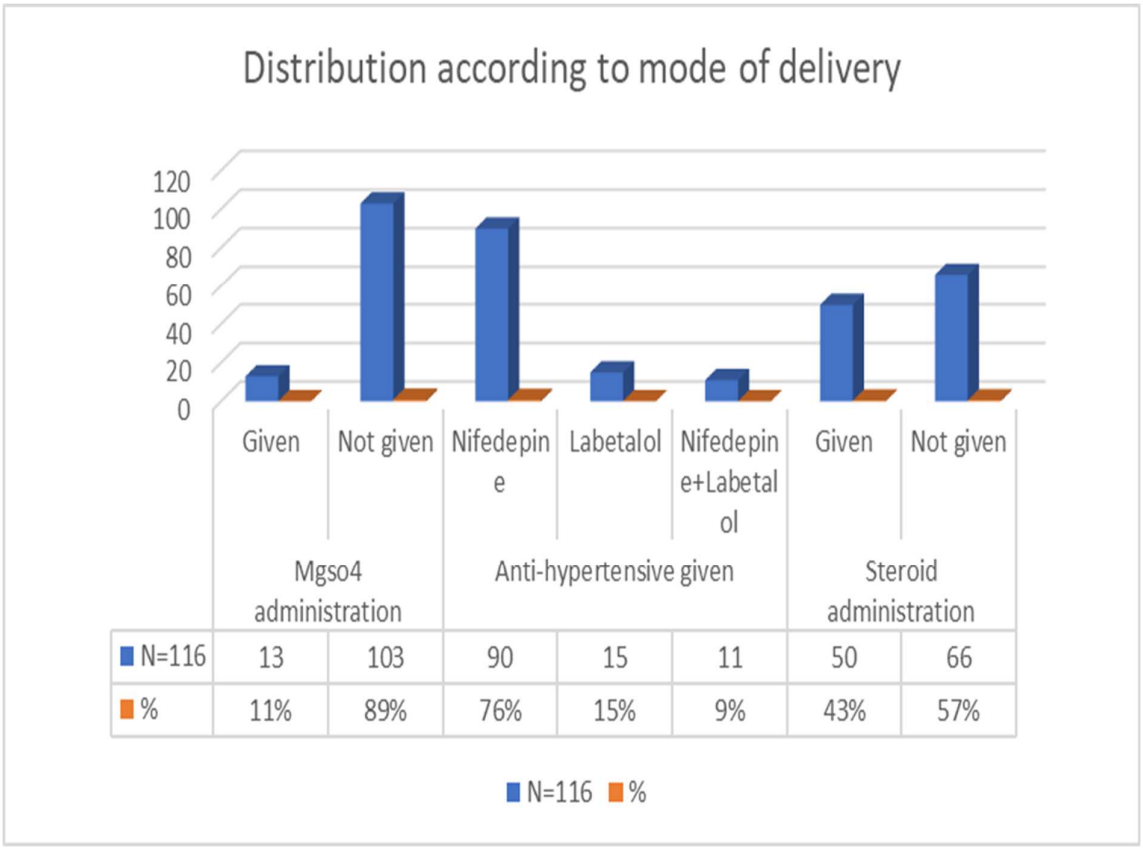


Table 15.Distribution of study population according to the days gained at each entry gestation in the study

Category	Sub category	Number	%
Conservative treatment	Given	13	11.2%
	Not given	103	88.7%
Days gained Gestational age	24-28	04	Mean 4
	28.1-30	05	5
	30.1-32	02	2
	32.1-4	01	1
Days gained	1 day	7	
	2-4 days	4	
	>4 days	2	

Among 116 participants only 13 patients (11.2%) were given expectant management. Overall, in our study population, 88.7 % of women were not eligible for expectant management. The median number of days of pregnancy prolongation was 1.5 days (1-5 days). The days gained were significantly higher among those who had expectant management between 28.1-30.

Figure 23. Distribution of study population according to conservative treatment given.

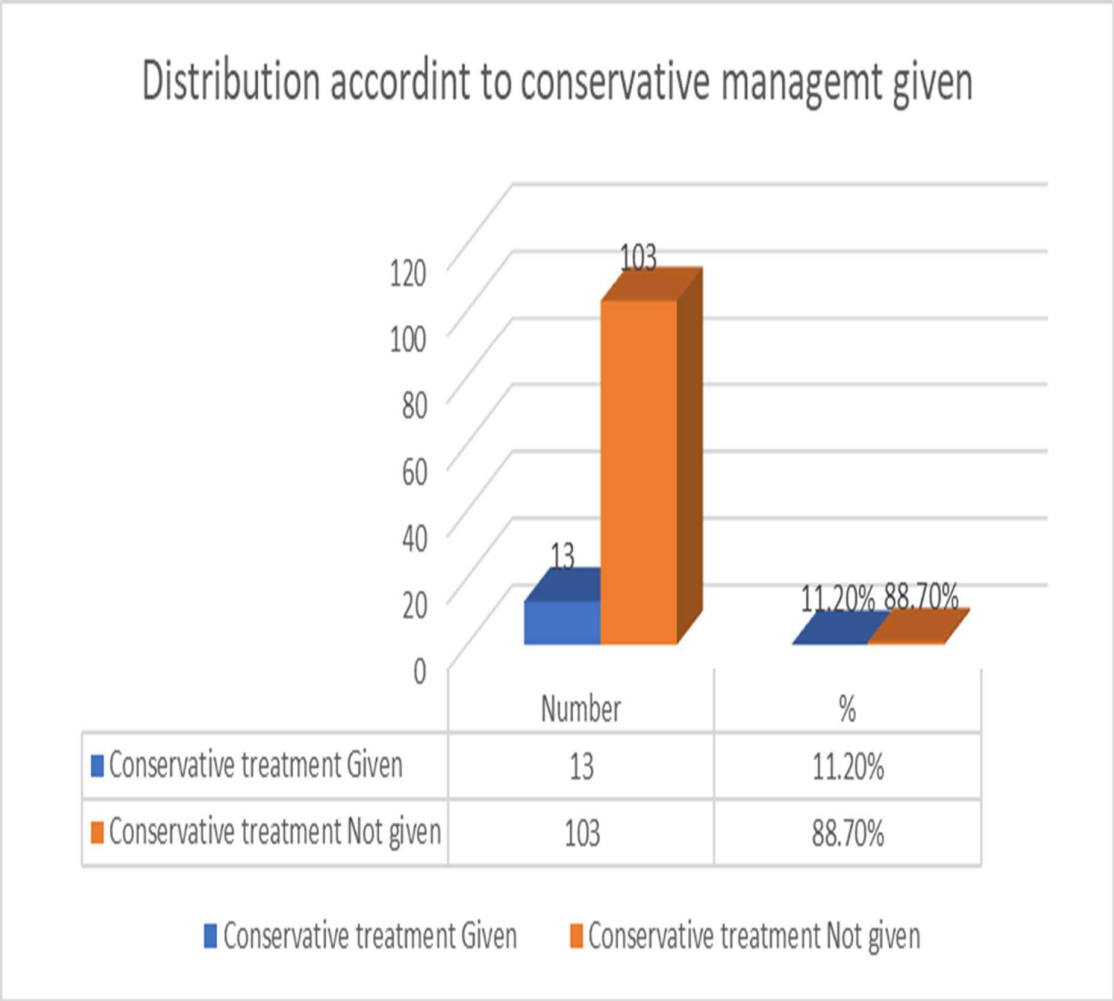


Table 16.Distributionof study population according to mode of delivery

Category	Sub-category	Number(N=116)	%
Vaginal delivery	Spontaneous	10	8.6%
	Induced	13	11.2%
	Instrumental	03	2.5%
Caesarean Section	Elective LSCS	05	4.3%
	Emergency	85	73.4%

Among the study population, 73.4% women underwent LSCS in comparison to 22.4% women who delivered vaginally out if which 3 had instrumental delivery i.e ventouse delivery and there were no forceps used .

Figure 24. Distribution of study population according to mode of delivery

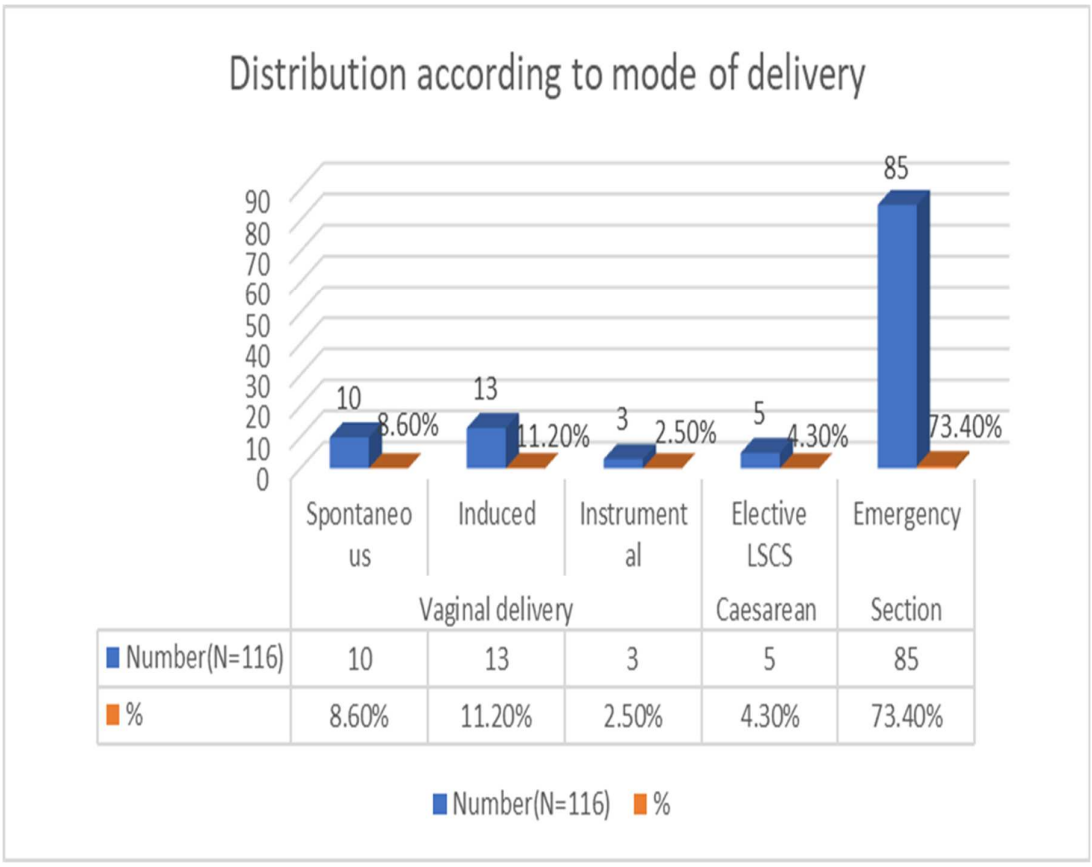


Table 17. Distribution of study population according to mode of delivery according of gestational age

Category	Sub-category	Number (N=116)	%
Vaginal delivery	Preterm:20-24^{+6}	01	0.8%
	25-28^{+6}	10	9%
	29-32^{+6}	05	4.3%
	33-36^{+6}	03	2.5%
	Term:37-40	06	5.1%
	>40	01	0.8%
Caesarean section	Preterm: 20-24^{+6}	0	0%
	25-28^{+6}	0	0%
	29-32^{+6}	15	13%
	33-36^{+6}	35	30.1%
	Term: 37-40	38	32.7%
	>40	02	1.7%

In the present study, it was concluded that around 70% of pre-term delivery were done vaginally. In cases of LSCS, 50% were pre-term and 50% were term.

Figure 25. Distribution of study population of mode of delivery according of gestational age

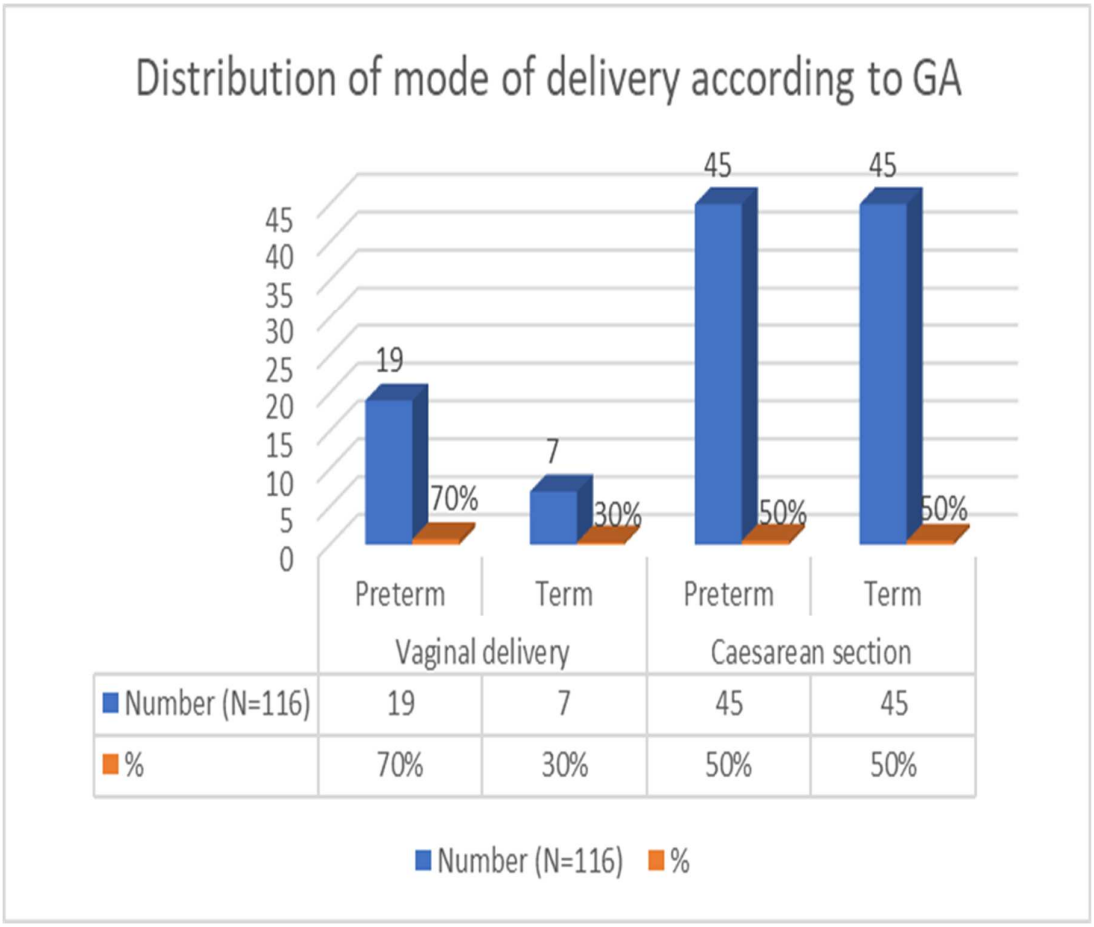
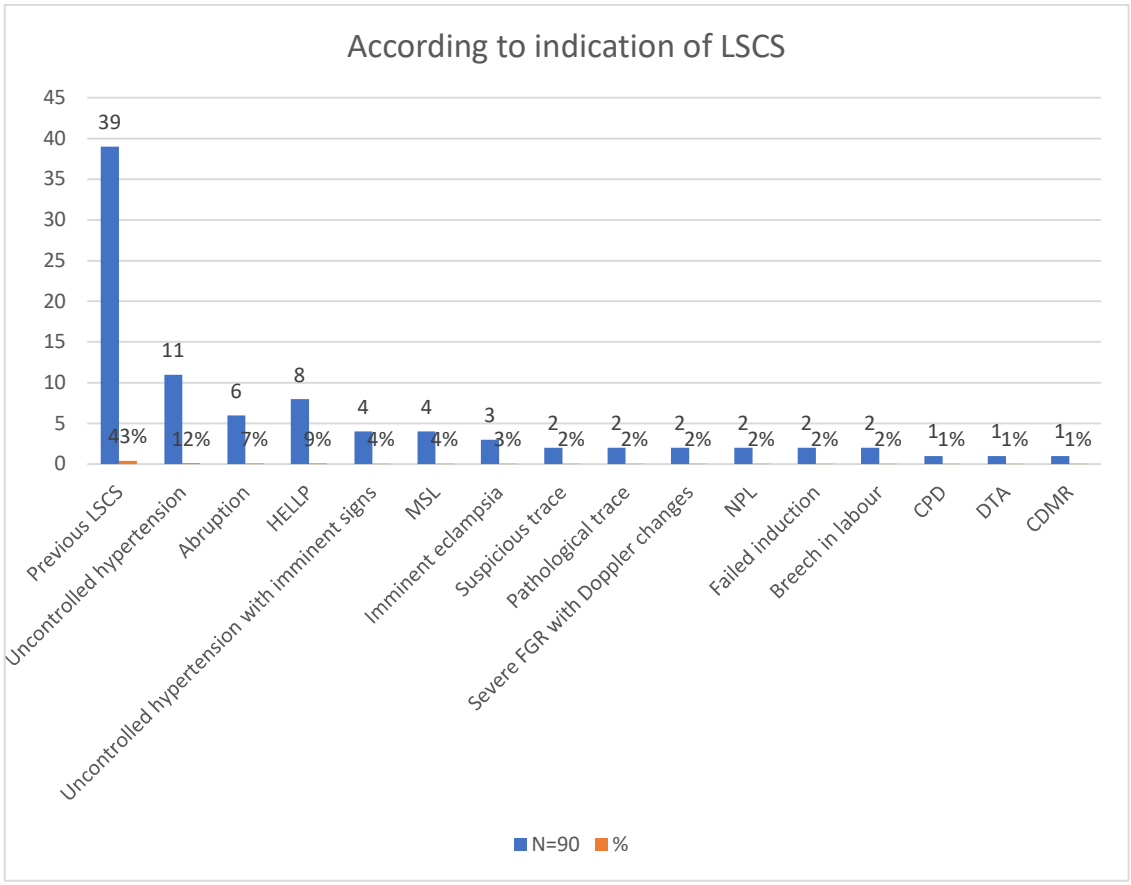


Table 18. Distribution of study population according to the indication of LSCS

Indication	N=90	%
Previous LSCS	39	43%
Uncontrolled hypertension	11	12%
Abruption	06	07%
HELLP	08	9%
Uncontrolled hypertension with imminent signs	04	4%
MSL	04	4%
Imminent eclampsia	03	3%
Suspicious trace	02	2%
Pathological trace	02	2%
Severe FGR with Doppler changes	02	2%
NPL	02	2%
Failed induction	02	2%
Breech in labour	02	2%
CPD	01	1%
DTA	01	1%
CDMR	01	1%

In the current study, previous LSCS was the most common indication among women with severe pre-eclampsia who underwent LSCS accounting to 43% and this serves as main reason for which caesarean rate was high among women with severe pre-eclampsia in our study. Followed by uncontrolled hypertension which accounts for 12 % and 7% of cases were due to abruption.

Figure 26. Distribution of study population according to the indication of LSCS



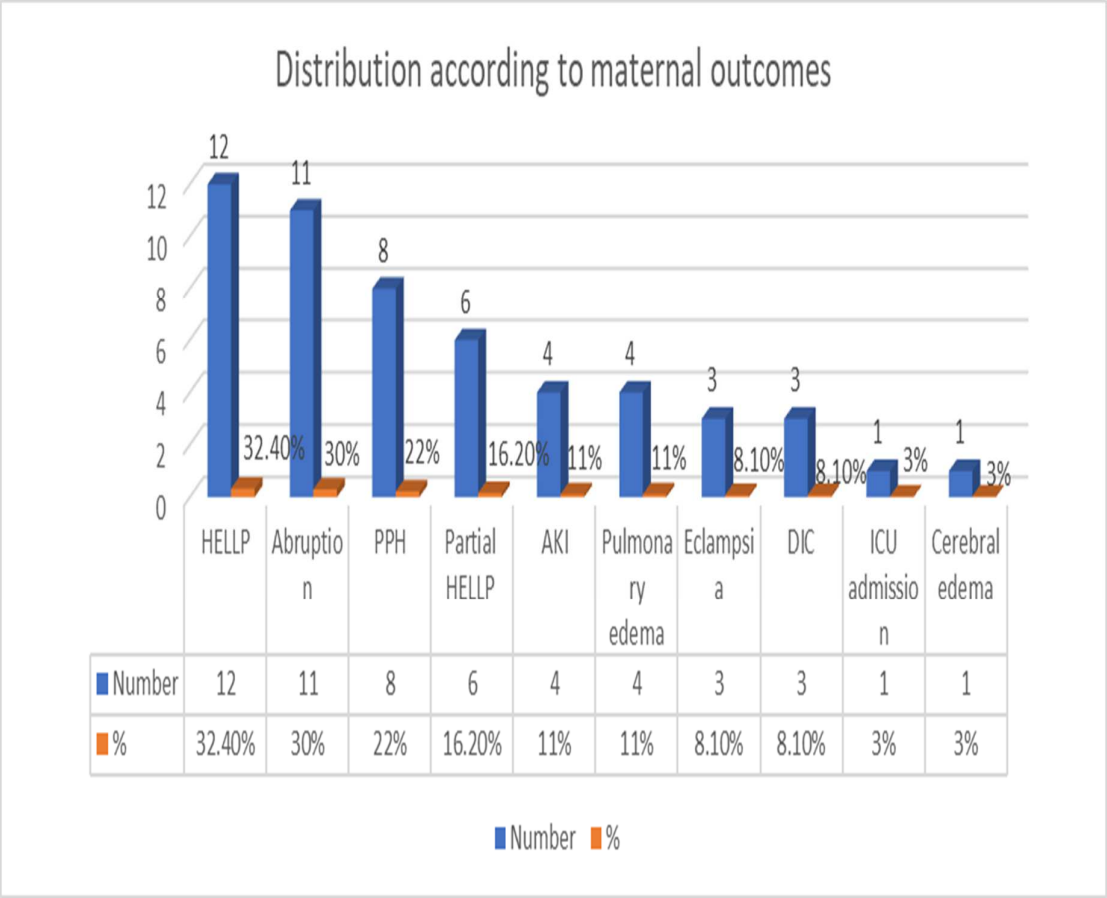
Maternal and perinatal outcomes in severe pre-eclampsia:

Table 19. Distribution of study population according to maternal complications:

Complication	Number	%
HELLP	12	32.4%
Abruption	11	30%
PPH	08	22%
Partial HELLP	06	16.2%
AKI	04	11%
Pulmonary edema	04	11%
Eclampsia	03	8.1%
DIC	03	8.1%
ICU admission	01	03%
Cerebral edema	01	03%

Among 116 cases, 37 women developed complications and it was found that HELLP was seen in 32.4% patients, abruption in 30% , PPH in 22% , Partial HELLP in 16.2%, pulmonary edema in 11% , eclampsia & DIC in 8.1%, whereas maternal ICU admission occurred in 3% of patients , there was no maternal mortality noted due to severe pre-eclampsia .

Figure 27. Distribution of study population according to maternal outcomes



Perinatal outcomes:

Table 28: Distribution of study population of study population according to baby characteristics:

Category	Sub category	Number(N=116)	%
Sex	Male	76	66%
	Female	40	34%
Birth weight	<1.0kg	08	6.8%
	1.1-1.5kg	26	22.4%
	1.6-2.0kg	18	15.5%
	2.1-2.5kg	31	26.7%
	2.6-3.0kg	19	16.3%
	>3.0kg	14	12.3%

In present study, 66% were male babies and 22,4% babies weight are in the range of 1.1 -1.5kg 26.7% babies weight were in the range of 2.1- 2.5kgand 14% babies weight was more than 3kg.

Figure 28. Distribution of study population according to birth weight

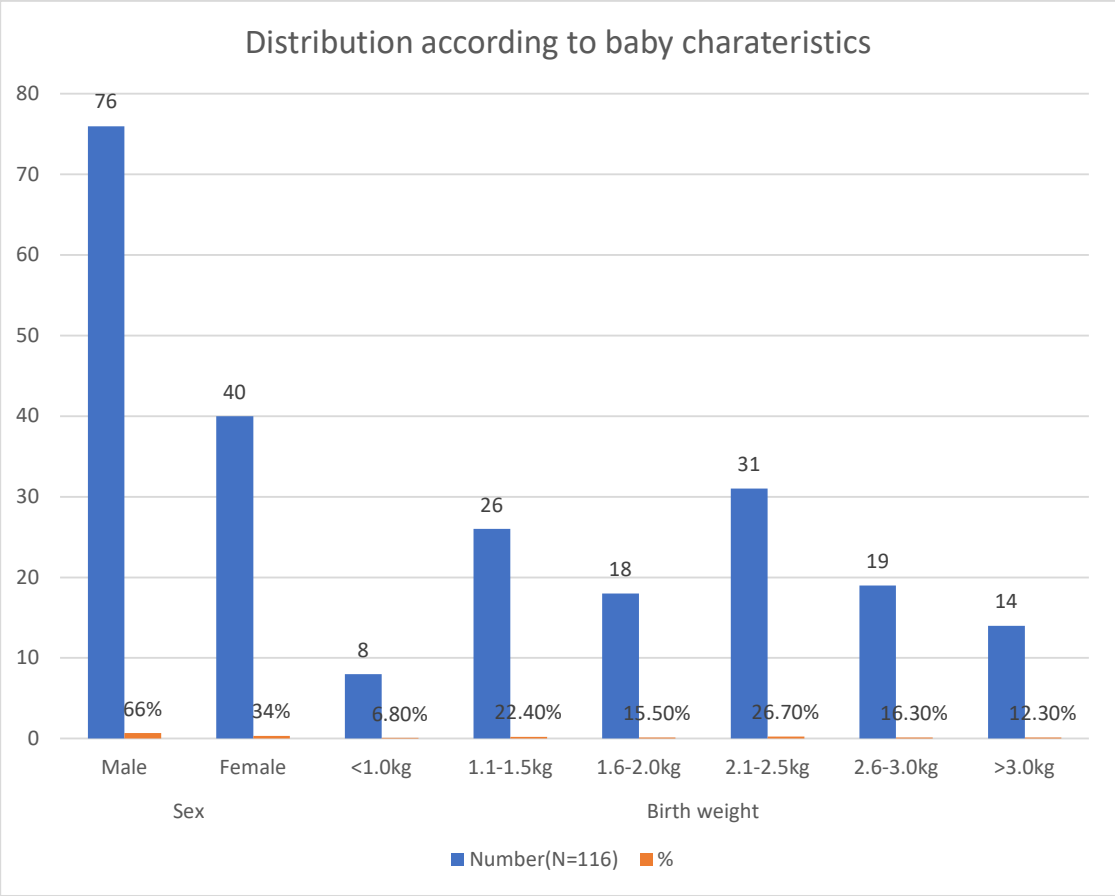


Table 21.Distribution of study population according to apgar score :

Apgar score	N=105	%
0-5	08	7.6%
5-7	06	6%
7-10	91	86.4%

In the current study , out of 105 live babies it was found that 7.6% of babies had a apgar score between 0-5 and 6% of babies had a apgar score between 5-7 .

Figure 29.Distribution of study population according to apgar acore:

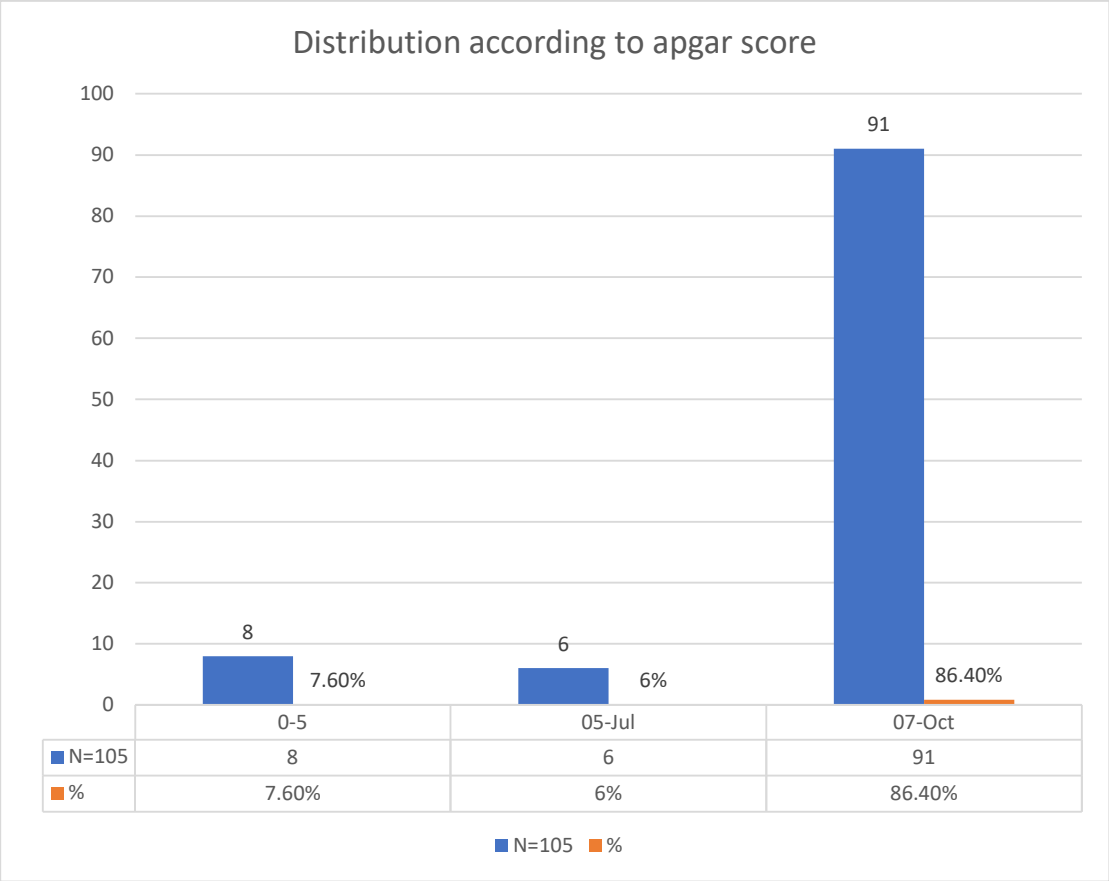


Table 22. Distribution of study population according to perinatal complications

Complications	Number (N=95)	%
Prematurity	50	53%
FGR	15	15.7%
IUD	11	12%
Low apgar	14	14.3%
Perinatal death	05	5%

In the present study , out of 116 babies there were 105 live births in which around 95 babies developed perinatal complication out which prematurity was observed in 55% of babies , which is the most common neonatal outcome of severe pre-eclampsia , 11 babies were IUD in which 5 babies were FSB and 6 babies were MSB . 5 babies died in the neonatal period in which 3 babies died on day 5 of life due to extremely low birth weight , 1 baby died on day 8 of life due to inborn error of metabolism and 1 baby died of day 15 of life due to sepsis.

Figure 30. Distribution of study population according to perinatal outcome

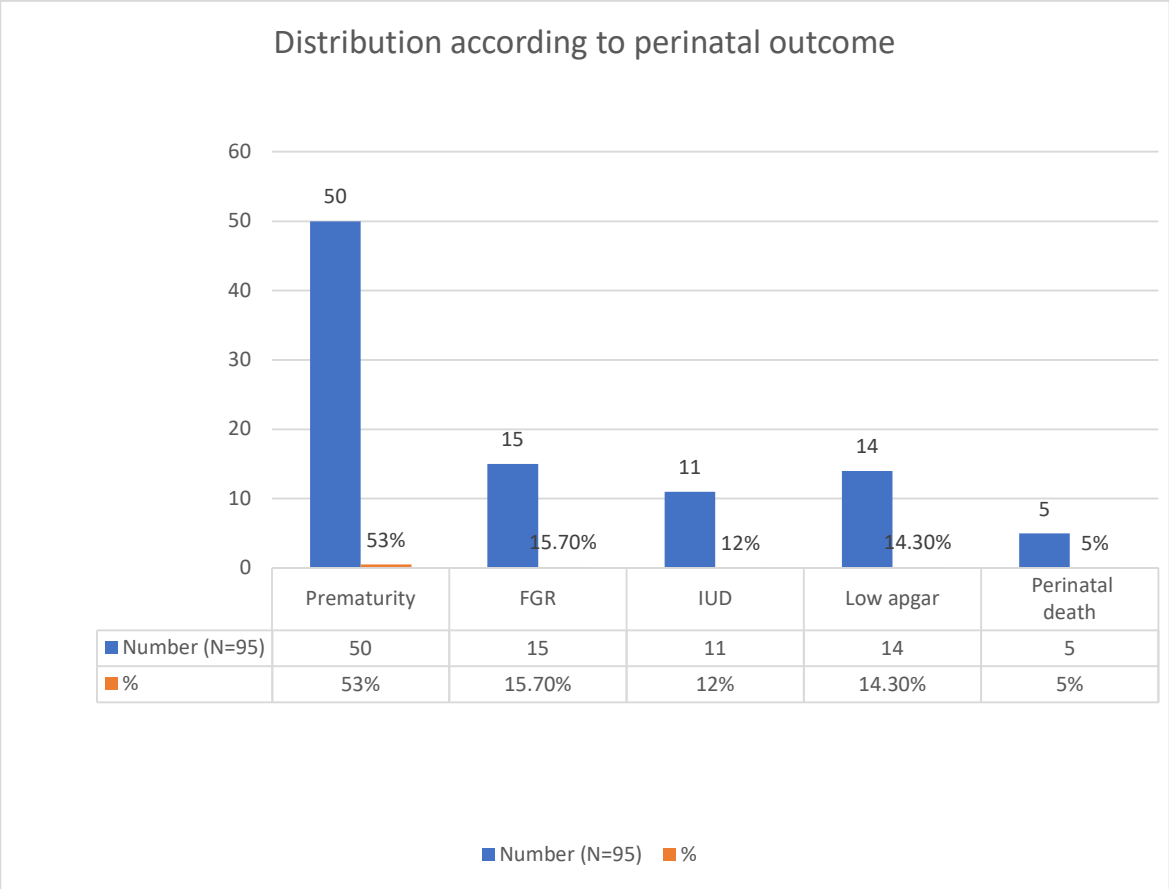
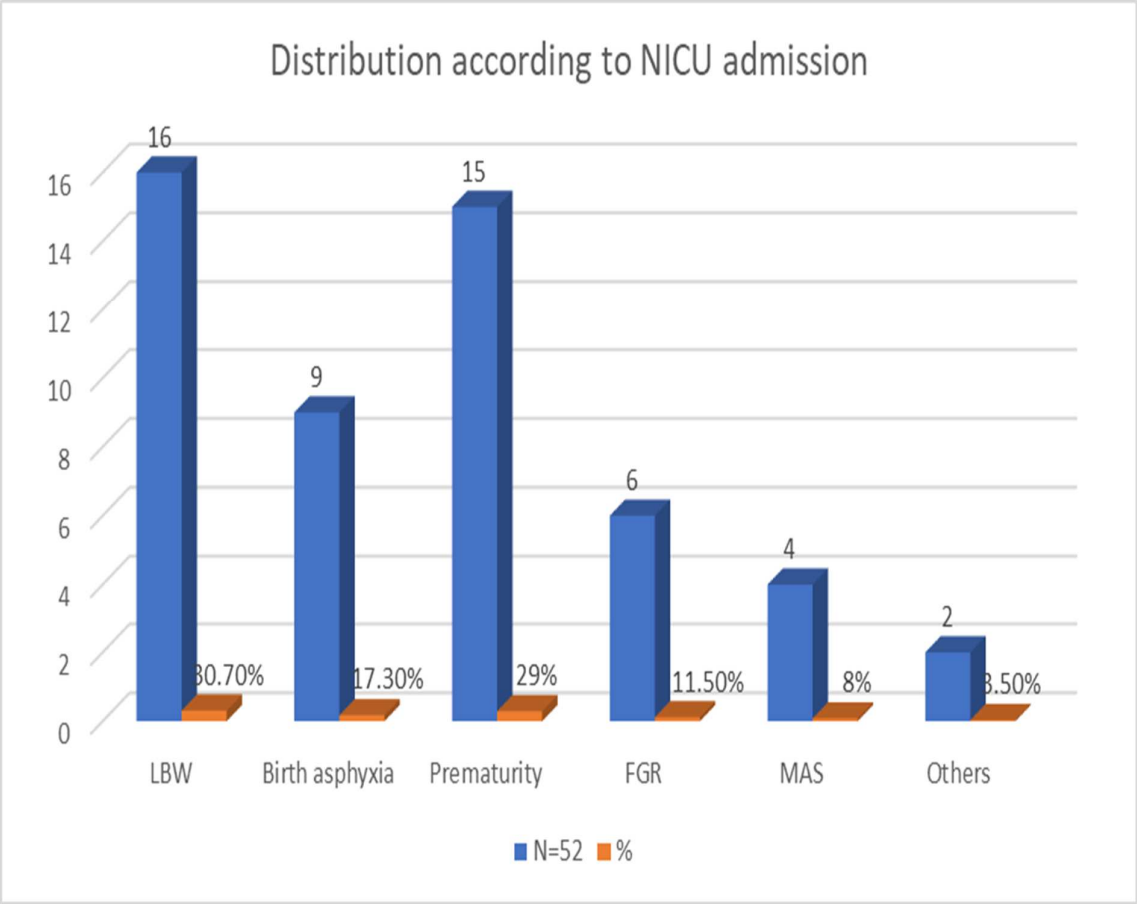


Table 23. Distribution of study population according to indications for NICU admission

Indications	N=52	%
LBW	16	30.7%
Birth asphyxia	09	17.3%
Prematurity	15	29%
FGR	06	11.5%
MAS	04	8%
Others	02	3.5%

In present study, total of 52 babies were shifted to NICU for various indications, most of them were observed for a period of 48-72hrs, given antibiotics and IV fluids .The most common indications were low birth weight, birth asphyxia and preterm babies. Other indication for which 2 babies were shifted to NICU was one in view of congenital diaphragmatic hernia and another one in view of hyperbilirubinemia.

Figure 31. Distribution of study population according to indications for NICU admission



DISCUSSION

Hypertensive disorder of pregnancy represents significant public health problem throughout the world, and pre-eclampsia is most common of these disorders. It complicates 5-10% of all pregnancies and is a primary cause of maternal and neonatal morbidity and mortality. Severe pre-eclampsia occurs in about 25% of all cases. The present study is a prospective observational study conducted in KAHER'S Dr Prabhakar kore hospital attached to JNMC to know the effect of severe pre-eclampsia on women with its maternal and perinatal outcome in study period from January 2021 to December 2021.

Prevalence of severe pre-eclampsia: In present study, total no of deliveries were 3154 , in which the hypertensive disorders are 26 cases of chronic hypertension , 183 women were gestational hypertension , 47 women were mild pre-eclampsia and 177 women were severe pre-eclampsia .Hence the prevalence of severe pre-eclampsia is about 5.7% among the total deliveries in the current study and the prevalence of severe pre-eclampsia was 40.5% among hypertensive disorder. In that 177 women , 22 women were twin pregnancy , 10 patients had overt DM and 5 patients had cardiac disorder . Therefore only 116 patients were included in the study.In a study conducted in China reported that the overall prevalence of severe pre-eclampsia is about 4% [49]. Also a study conducted in south part of India reported that a overall prevalence of severe pre-eclampsia in their study was about 4.7% similar to our study [50].

Age distribution: Age is considered as an supreme factor because it is associated with increased maternal and perinatal risks. The current study demonstrated that the mean age of the studied women was 25.87 years with standard deviation of 4.8 years showing that severe pre-eclampsia is more common in the younger age group . 38.7%

of the women were in the age group of 20-25 years. 8.6% were in the age group of 31-35 years .Similar findings have been reported by Sujatha Patnaik et al , in which 70% of the women were in the age group of 20-30 years and only 10% were in the age group of more than 30 years [35]. In a similar study done by Saxena et al also reported that around 71% of the study population were in the age group of 20-30 years [10]. However, in contrast to our findings, Sahu *et al* reported maternal age to be significantly higher in pregnancy-induced hypertension associated pregnancy [36]. Results in our study could be because of a large number of younger age group of pregnant patients as girls in our country get married at an early age.

Parity distribution: Primigravida has been found to be associated with pre-eclampsia. In the present study it has been observed that pre-eclampsia occurred more frequently in primigravida (51%) as compare to multigravida (49%). In total of 116 women, 59 women were primigravida and 57 of the study population were multiparous. In a study conducted by Ketz et al also reported that 70% women as primigravida in the study population similar to our study[37].

Similar studies conducted by Swamy et al [38] and Shobana Pillai et al [22] also reported 50% and 61% of study population as primigravida.

Sibai and Cunningham reviewed a number of worldwide studies and concluded that the incidence of pre-eclampsia in nulliparous populations was more than that for multiparous . Many other studies have also reported primiparous as a risk factor for severe pre-eclampsia. It could be because of the failure of normal invasion of trophoblastic cells that leads to maladaptation of the spiral arterioles. However,in contrast to the present study a lower percentage of 21.3% for primigravidas was reported in the study of Pramana et al [39] .

Gestational age distribution: In the present day, it was concluded that about 38% of study population were between the gestational age of 37 weeks to 40weeks and 10% of the women were in the gestational age of less than 28 weeks .In contrast to the current study ,a study conducted by Ngwenya S et al [1] and Shobana pillai et al [22] reported that around 50 % and 53% respectively were in the gestational age of less than 34 weeks . This variation in study results might be due to the fact that ours being a teritery care centre received a large number of women as undiagnosed and unbooked cases in the last trimester.

Registration status distribution: In the current study, as told earlier 75% i.e majority of the patients were unregistered cases and 27% of women were registered cases. Similar findings are noted in the study done by saxena et al that around 58 % were unbooked cases [10] .In contrast to the present study , a study conducted by shobana pillai reported 78% of registered cases in the study population which correlated with the fact that more women were in the gestational age group of less than 34 weeks [22].

Presenting symptoms: In terms of clinical presentation , headache was the most common antecedent symptom present in 34.4% of the patients followed by epigastric pain(29%) and blurring of vision (13.9%).Around 33 patients did not present with any symptoms and 10 patients had more than one symptoms. Similarly , Douglas et al also reported headache, epigastric pain and blurring of vision in 50%, 19% and 19% patients respectively [40].In a study conducted by Rekha, et al. study, cases manifested with edema with 90%, jaundice in 57%, nervous system involvement in 42%, visual symptoms in 6.4%, vaginal bleeding in 11.30%, and HELLP syndrome in 2.80% of cases [41]. In many studies, it has been observed that pedal edema has the most common presenting symptom, but in the current study pedal edema was not taken into account

the fact that pedal edema is not a significant feature for severe pre-eclampsia as it can be both physiological as well as pathological.

Laboratory parameters distribution: In present study among 116 cases , liver functions were deranged in 23.2% of cases and renal functions were deranged in about 8% with a mean uric acid of 1.64 with standard deviation 6.2 , mean urea level of 23.25 with a standard deviation of 3.18 and mean creatinine level of 1.09 with standard deviation of 0.23 in another 5% whereas in a study done by Singhal SR et al showed that 20% patients had deranged liver function tests while 27% patients had deranged renal functions [43].

In the current study ,16% patients had low platelet counts. PT/ INR was deranged in 03% patients with mean of 14.22 and standard deviation 1.1 and mean of 1.03 with standard deviation of 0.5 respectively who were managed by giving FFP transfusions.12% of patient had elevated uric acid levels and 36% of patients had proteinuria of more than 3+ .In the study conducted by Saxena et al reported that liver functions were deranged in 24% of our cases out of which 17.3% cases had SGOT> 100 IU and another 18.67% cases had SGPT>100 IU. Renal functions were deranged in 59% of our patients, who most commonly had raised serum creatinine levels [10].

It has been concluded that in almost 50% of the studies levels of liver function and renal function are deranged when compared to other parameters. In the present study, 9 patients presented with fundoscopy changes with 67% of patients with grade 1 papilledema and 33% patients with grade 2 papilledema. In a study conducted at Haryana showed 3% patients with hypertensive changes and 3% patients with papilledema. Fundoscpy findings correlated with the fact that ophthalmic changes are rare to be seen in severe pre-eclampsia patients.

Mode of management : In the present study patients were given anti-hypertensive to control blood pressure ,in which nifedepine is the most commonly used anti-hypertensive among 90 patients i.e 75% .Labetalol was in combination with nifedipine in 9% of patients respectively in the current study .

IV labetalol was used as emergency drug on admission in around 15% of the patients. In the study conducted by Shobana pillai reported the same as our study that nifedipine was the most commonly used drug either singly or in combination with labetalol in about 39% and 24% respectively. They have also reported that alphamethyldopa was also used when patients presented with severe pre-eclampsia before 28 weeks of gestation[22] .

In the current study, out of 116 cases $Mgso_4$ was administered in around 11% of patients and 43% of patients were given antenatal steriods . In a study conducted by Sujata Patanail et al concluded , that around 83 patients have received $Mgso_4$ in combination with nifedepine and labetalol [24] .

In the present study , expectant management was also given to patients. Out of 116 patients , 13 patients was given conservative management with median prolongation of 1.5 days. The minimum days gained was 1 day and maximam days gained was 5 days. In a study conducted by Swamy et al , reported the median number of days of pregnancy prolongation was 5 (1–24) and the days gained were significantly higher among those who had expectant management between 28.1 and 30 weeks (14 days) compared with the other two groups—30.1–32 weeks (5 days) and 32.1–34 weeks [38].

Mode of delivery: The mode of delivery was determined by severity of maternal condition, bishop's score, gestational age, fetal condition, USG and laboratory investigations. In the current study , out of 116 cases the most common mode of delivery was lower segment cesarean section in 78% of the cases and the most common indication was previous LSCS 43.3% in the present study. 22.3% of the cases delivered normally in which 8.6% were spontaneous , 11.2% were induced and 3 patients delivered by ventouse delivery.

In a study by Singhal et al reported 33% caesarean section rate [43] and Tufnell et al[44] reported as high as 72% caesarean section rate in BJOG . Caesarean section rates of 71% and 78% respectively were reported by Miguel Met al and Dissanayake VH et al [44].In Tavassoli, et al. study, caesarean section was 47.1% in severe pre eclampsia[45] . A study by Saxena N, et al. showed caesarean section rate of48.2% and vaginal delivery 51.8% [10].The high rate of caesarean section in the present study is due emergency delivery approach taken to prevent further maternal and fetal complications due to severe preeclampsia or eclampsia especially in cases where the cervix is unfavourable for induction. 20% of women delivered by vaginal delivery and 2% of women delivered by instrumental delivery.

Maternal outcomes: In the present study out of 116 cases, 37 patients (32%) had maternal complication, which included HELLP in 12 cases followed by abruption in 09, PPH in 08, partial HEELP in 6 patients. Regarding ICU admission, the present study displayed that 0.3% of cases were admitted to ICU. In contrast, Patnaik et al reported that 4.16% of cases with severe preeclampsia were admitted to ICU [35] .This illustrates the proper care which the cases received in the current study.

In the study by Shaikh S et al and Murphys DJ reported that abruption cases are 9 in number [24] .Whereas another study done by Singhal SR et al showed only one case had abruption [43].

Lakshmi Narayana Kota reported a maternal mortality of 61.66% [21]. Curiel Balsera et al, Ngwenya S. et al and Quah et al.14 reported 1.5%, 1.7% and 1.3% maternal mortality in their settings, respectively [1].

It is heartening to know that no maternal mortality occurred in the present study as compared to 1.5%9 and 0.9% in other studies. This can be explained by the multidisciplinary approach and the excellent care given to the mother admitted in the labour room in present setup.

Perinatal outcomes: In the present study ,overall the rate of FGR was 15.7 %. 105(87 %) babies were born alive, with 14(13.5 %) of these infants having an Apgar score <7 at 5 min. In the current study about 7.6% of babies had an apgar 0-5 and 6% had a score of 5-7 at 5 mins of birth .The total NICU admission required among 105 live born infants was 52 babies. They stayed in the NICU for a median number of 12 days. There was no statistically significant difference among different gestational age groups regarding the rate of FGR, NICU admission or NICU stay. Neonatal death of 5% reported in the present study .

In a study conducted by Turgut A. et al reported that the mean apgar score at one minute was 6.39 and at 5 mins interval its around 7.39 respectively[46] .

Prematurity is commonly associated with severe pre-eclampsia. Most of the cases 66% had preterm delivery. Almost similar findings were reported by Tuffnell et al 65.3% of infants were pre mature [47].The high incidence of preterm delivery could

be attributed to the early intervention and induction of labour or LSCS done to avert further maternal and perinatal complications.

In the current study ,out of 116 babies 5 babies died which accounts for 4.5% of perinatal mortality .In the study conducted by Shobana pillai reported a perinatal mortality rate of 18% [22]. Similar findings were also noted in a study conducted by saxena et al and Shahin et al from Pakistan reported a perinatal mortality rate of 30% and 41.6% respectively [10,48] . Perinatal mortality rate is comparatively less in the current study when compared to other studies , proving the factor that prompt treatment to the mother and timely delivery of the baby and proper NICU care in the current setup.

In the present study , the most common indication for NICU admission was due to low birth weight and fetal distress of 53% and 15.7% respectively . In a study by Turgut A. et al. the rate of NICU admission was 69.5% and most common cause was hyperbilirubinemia [27].

The strengths of the study is that it is a prospective study and all details of the study population were taken from the patient in person and babies were followed up till discharge .

The major limitation of the current study is that its a one year study which has been conducted only in a single teritary care centre .Henceforth ,to emphasise the maternal, fetal and neonatal outcomes of severe pre-eclampsia on a broader scale, further multicenter studies should be carried out across various years. We were not able to study the fetomaternal outcomes in case of severe pre-eclampsia in a larger scale since it was a one year study and the sample size was limited.

Despite advances in medical practice, severe pre-eclampsia has remained a leading cause of maternal mortality throughout the world. It is a common problem in developing countries because of illiteracy, poor antenatal care, lack of health awareness and poverty.

CONCLUSION

The current study was done to know the maternal and fetal outcomes in severe pre-eclampsia so that the natural course of disease can be blocked at the secondary and tertiary levels of prevention. While early detection and prevention of occurrence of the disease per se is called for the allying of the severity of the disease and thereby reducing the complications prompt the mainstay in the present times. The morbidity and mortality of the severe pre-eclampsia in mother and the neonate is considerably reduced with effective management. The present study emphasizes the fact that proper antenatal care and management of severe pre-eclampsia in a teritary care setup can effectively reduce the maternal and perinatal morbidity and mortality.

SUMMARY

- Majority of the study population i.e 88 participants were in the age group of 21-30 years with the mean age being 25.87 years with the maximum age observed was 41 years and the minimum age observed was 19 year.
- In the present study shows among women with severe pre-eclampsia
- 51% were primigravida and 57 patients were multigravida.
- Most of the women (51%) were diagnosed at gestational age of around 38 weeks-40 weeks The mean gestational age at diagnosis was 35.32weekswith standard deviation of 37 weeks. The maximum gestational age observed was 41 weeks and the minimum observed was 24 weeks.
- Majority of women presented with the complaint of headache 24.1%, followed by epigastric pain 23%, visual defects 8.6 % and vomiting 3.4% .
- Nifedipine was the most commonly used drug in the present study
- Overall, in our study population, 88.7 % of women were not eligible for expectant management. The median number of days of pregnancy prolongation was 1.5 days (1-5 days). The days gained were significantly higher among those who had expectant management between 28.1-30.
- 73.4% women underwent LSCS in comparison to 22.4% women who delivered vaginally out if which 3 had instrumental delivery (ventouse).
- Previous LSCS was the most common indication among women with severe pre-eclampsia who underwent LSCS.

- It was found that HELLP was seen in 32.4% patients, Abruption in 30%, PPH in 22%, Partial HELLP in 16.2%, pulmonary edema in 11%, eclampsia &DIC in 8.1%, whereas maternal ICU admission occurred in 3% of patients, there was no maternal mortality noted due to severe pre-eclampsia.
- There was no maternal mortality.
- Preterm was observed in 55% of babies, which is the most common neonatal outcome of severe pre-eclampsia.
- Out of 75 live births 45 babies required NICU admission and the most common cause of NICU admission was low birth weight and fetal distress accounting to 35% and 20% respectively.
- The total early neonatal death was 4.2%

WAIVER OF CONSENT

K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed – to- be- University)
Accredited 'A' Grade by NAAC (2nd Cycle) Placed in Category 'A' by MHRD (GoI)
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
Website: http//www.jnmc.edu
E-Mail : dome@jnmc.edu
Phone: (+ 91-(0)831 Office : 2472550
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref: MDC/DOME/ 290 **Date: 22/12/2021**

To,

REG. NO. BJ0120009
J. N. Medical College,
BELAGAVI.

With reference to the above, we wish to inform you that your proposed research project titled

"One year observational study of maternal and perinatal outcome in severe pre-eclampsia at tertiary care hospital, Belagavi" collected. The waiver of consent has been approved for the proposed research project and has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

(Dr. Smita Sonoli)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

(Dr. Harsha Hegde)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi

ANNEXURE 1: PROFORMA

TITLE: "One Year Observational Study of Maternal and Perinatal Outcome in Severe Pre-Eclampsia At Tertiary Care Hospital, Belagavi".

Screening Id: ☐☐☐☐ Study Id: ☐☐☐☐

Age: ☐☐

Date Of enrollment: ☐☐☐☐☐☐

COMPLAINTS-

PERIOD OF AMMENORRHOEA-

IMMINENT SIGNS-

1) Headache	☐ Yes	No ☐
2) Blurring of vision	☐ Yes	No ☐
3) Epigastric discomfort	☐ Yes	No ☐
5) Vomiting.	☐ Yes	No ☐
4) Seizures	☐ Yes	No ☐

Yes:

- Number of episodes-
- Duration-
- LOC-
- Lucid Interval-

Obstetric history:

Married Life (years): ☐ ☐

Obstetric score: G ☐ P ☐ L ☐ A ☐ D ☐

Menstrual History:

LMP-

EDD/CEDD-

Period of gestation-

Past History: Diabetes ☐ Yes ☐ No

Tuberculosis ☐ Yes ☐ No

Hypertension ☐ Yes ☐ No

Asthma ☐ Yes ☐ No

Personal History:

DIET:

BOWEL AND BLADDER`:

APPETITE:

HABITS: Smoking/Tobacco/Alcohol

☐ ☐ ☐

General Examinations:

PULSE RATE (Beats per minute): bpm

BLOOD PRESSURE : / mmhg

PALLOR: ☐ Yes No ☐

ICTERUS: ☐ Yes No ☐

PEDAL EDEMA: ☐ Yes No ☐

PA:

INVESTIGATION:

DATE OF ADMISSION:	
COMPLETE BLOOD COUNT: Hemoglobin Hematocrit Total count Rbc	
PLATLET COUNT:	
URINE EXAMINATION : PROTEIN SUGARS	
LIVERFUNCTION TEST: Total bilirubin Direct bilirubin Indirect bilirubin ALP SGOT SGPT Albumin A:G ratio Serum LDH RENALFUNCTION TEST: Serum uric acid: Urea:	

Serum creatinine:	
COAGULATON PROFILE: APTT: C: T: R: PT: C: T: R: INR: TT: C: T R: SERUM FIBRINOGEN: D-DIMER:	
PHERIPHERAL SMEAR:	

Management:

1) Date of delivery

2) Mode of delivery VAGINAL DELIVERY C-Section ☐

3) Induction Augmentation ☐

4) Duration of Labour

5) Intrapartum Complication Yes ☐ No ☐

6) Indication of LSCS-

1)previous lscs not w/f vabc

2)NPL

3)Uncontrolled hypertension

4)Pathological trace

5)MSL

6)Others

BLOOD LOSS :

Examination of Placenta:

Weight of Placenta-

Retroplacental Clots Yes ☐ No ☐

Placental Infarction Yes ☐ No ☐

Maternal Outcome:

Peripartum Complications:

1) Convulsions Yes ☐ No ☐

2) DIC Yes ☐ No ☐

3) Abruptio Placenta Yes ☐ No ☐

4) Peripartum cardiomyopathy Yes ☐ No ☐

5) Respiratory Complications Yes ☐ No ☐

6) HELLP Yes ☐ No ☐

7) ACUTE KIDNEY I Yes ☐ No ☐

8) Pulmonary Edema Yes ☐ No ☐

9)Pulmonary Embolism Yes ☐ No ☐

10) PPH Yes ☐ No ☐

13) ICU Admissions- Yes ☐ No ☐

Duration of Stay-

Condition at discharge-

Cause of death if so-

Perinatal Complication:

1) Condition of birth ☐ ALIVE ☐ IUD ☐ MSB ☐ FSB

2) TERM ☐ PRETERM ☐

3) Sex - FEMALE. ☐ MALE ☐

4) WEIGHT- <1 Kg

1-1.5 Kg

1.5-2 Kg

2-2.5 Kg

>3 Kg

5) APGAR-

6) NICU admission YES ☐ NO ☐

7) Indication of NICU admission-

1] FETAL DISTRESS. YES ☐ NO ☐

2] MSL YES ☐ NO ☐

3] IUGR YES ☐ NO ☐

4] LOW BIRTH WEIGHT YES ☐ NO ☐

5]PRETERM YES ☐ NO ☐

6] ASPHYXIA YES ☐ NO ☐

7] NON REASSURING CTG YES ☐ NO ☐

8] OTHERS

8) Condition of baby at discharge-

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s number	AGE	REG-1,UNREG-2	POG	EO-1,LO-2	PARITY,PRIMI-1,MULTI-2	BP	IMMINENT SIGNS,NO:0,HEADACHE-1,BLURRING-2,EPIGASTRIC,3,VOMITING:4	PALLOR,PRESENT:1,ABSENT:2	ICTERUS,PRESENT:1,ABSENT:2	PEDAL EDEMA,PRESENT:1,ABSENT:2	HYPOTHYROIDISM,PRESENT:1,ABSENT:2	URINE ALBUMIN	PLATLET	AST/OT	ALT	LDH	RFT,DERANGED-1,RANGED-2	URIC ACID	DIC,DERANGED-1,RANGED:2	MODE OF DELIVERY,NORMAL-1,SECTION:2	INDICATION	IUGR,PRESENT-1,ABSENT-2	DOPPLER CHANGES,NORMAL-1,ABNORMAL-2	FUNDOSCOPY,NORMAL-1,ABNORMAL-2	MGSO4 ADMINISTRATION,GIVEN-1,NOT GIVEN-2	MATERNAL OUTCOME,1-CONVULSION,2:DIC,3-ABDRUPTIO,4-CVS,5-RESPIRATORY,6-HELLP,7-RENAL FAILURE,8-PULMONARY EDEMA,9-PULMONARY EMBOLISM,10-PPH,11-ICU,12-PARTIAL HELLP	MATERNAL MORTALITY	ANTIHYPERTENSIVE GIVEN-1,NOT GIVEN-2	BLOOD TRANSFUSION,GIVEN-1,NOT GIVEN-2	CONSERVATIVE,GIVEN-1,NOT GIVEN-2	PERINATAL OUTCOME,1-LIUD:2,FSB:3	BIRTH WEIGHT,<1KG:1,1.0-1.5KG:2,1.6KG-2:3,2.1 -2.5:4,2.6-3.5:>3:6	NICU ADMISSION O:DIED,1:YES,2:NO	INDICATION,FETAL DISTRESS:1,MSL:2,FGR:3,LBW:4,PRETERM:5,AXPHYSIA:6,NST:7,OTHERS:8	CONDITION AT DISCHARGE
1	26	2	32+3	1	1	180/110	0	2	2	1	1	4+	2L	18	11	301	2	6.9	2	2	SEV PE WITH UNCONTROLLED HTN	2	1	1	2	0		1	2	1	1	2	1	5	KMC
2	28	2	39+6	2	1	180/110	0	2	2	1	1	3+	2.33	19	12	280	2	5.6	2	2	MSL	2	1	1	2	10		1	1	2	1	5	1	1	STABLE
3	19	2	38+6	2	1	170/100	3,4	2	2	2	2	3+	2.17	21	10	403	2	5.6	2	1		2	1	1	1	0		1	2	2	1	4	2	NO	STABLE
4	32	2	40	2	2	160/100	0	2	2	2	1	TRACE	3.54	21	13	251	2	5.4	2	2	MSL	2	1	1	1	0		1	2	2	1	5	2	NO	STABLE
5	36	1	34+1	2	2	174/110	0	2	2	1	1	3+	73000	21	10	337	2	6.6	2	2	HELLP	2	1	1	1	6		1	1	2	1	2	1	5	STABLE
6	23	1	25+5	1	1	160/110	0	2	2	1	1	2+	2.41	34	19	330	2	7.3	2	1		1	2-AEDF	1	1	0		1	1	2	1	1	1	4,5	EXPIRED(D1)
7	23	2	36+1	2	1	160/110	0	2	2	1	1	3+	1.36	45	35	379	2	5	2	2	SEV PE WITH UNCONTROLLED HTN	2	2-AEDF	1	1	0		1	2	2	1	4	2	NO	STABLE
8	31	1	33+4	1	1	170/110	0	2	2	1	1	3+	1.54	29	17	354	2	5.7	2	2	PREV LSCS	2	2-INC RESIST	2	1	0		1	2	1	1	3	1	5	KMC
9	19	2	34+4	2	1	190/110	0	1	2	1	2	2+	52000	43	15	697	2	5.2	1	1		1	1	1	1	2,3,6		1	1	2	2	3	2	NO	STABLE
10	26	1	38+1	2	1	160/100	0	2	2	2	2	2+	1.42	20	10	277	2	6.9	1	1		2	1	1	1	0		2	2	2	1	4	2	NO	STABLE
11	25	1	32+1	1	1	156/110	1	2	2	1	2	3+	1.91	85	107	546	1	7.4	2	2	SEV PE WITH FGR	1	1	1	1	0		1	2	2	1	2	1	1	STABLE
12	24	2	31+1	1	1	160/110	0	1	2	1	2	3+	43000	49	16	250	1	7.1	1	2	ABRUPTIO	1		1	2	3,7,10,11		1	1	2	2	2	2	NO	STABLE
13	30	2	39+3	2	2	170/110	4	2	2	1	2	3+	2.96	18	10	188	2	4.3	2	2	SEV PE	2	1	1	1	0		1	2	2	1	4	2	NO	STABLE
14	25	1	38+5	2	2	164/100	0	2	2	2	2	3+	2.73	43	33	351	2	4.4	2	1		2	1	1	2	0		2	2	2	1	5	2	NO	STABLE
15	32	1	33+3	1	1	160/100	3,4	2	2	1	2	1+	2.45	10	10	204	2	4.6	2	2	PATHOLOGICAL TRACE	2	1	1	1	0		1	2	2	1	4	2	NO	STABLE
16	20	1	37+3	2	1	160/110	3	2	2	1	2	2+	2.19	36	38	252	2	4	2	2	NPL	2	1	1	2	0		2	2	2	1	4	2	NO	STABLE
17	41	2	38+1	2	2	174/108	0	2	2	1	2	1+	1.25	36	38	350	2	6.4	2	2	PREV LSCS WITH SEV PE	2	2--AEDF	1	2	0		1	2	2	1	4	2	NO	STABLE
18	24	2	41	2	1	170/100	1,4	2	2	1	2	2+	1.39	22	11	570	2	6.7	2	2	DTA	2	2-AEDF	1	1	0		1	2	2	1	6	2	NO	STABLE
19	23	1	39	2	1	160/100	0	1	2	1	2	2+	95000	19	11	363	2	3.4	2	2	PARTIAL HELLP	2	1	1	2	12		1	1	2	1	6	2	NO	STABLE
20	25	1	40+1	2	2	160/100	0	2	2	1	2	3+	2.6	69	176	710	1	4.5	2	2	PREV LSCS	2	1	1	1	8,12		1	1	2	1	6	2	NO	STABLE
21	19	1	37+4	2	2	160/100	0	2	2	1	2	2+	1.07	45	29	450	2	3.9	2	2	SEV PE WITH UNCONTROLLED HTN	2	1	1	2	0		1	2	2	1	4	2	NO	STABLE
22	25	1	40	2	2	160/90	3,4	2	2	1	2	2+	2.87	16	10	505	2	4.9	2	2	NPL	2	1	1	2	0		2	2	2	1	5	2	NO	STABLE
23	21	1	36+3	2	2	160/100	0	2	2	1	2	1+	2.52	22	20	450	2	6.1	2	2	ABRUPTION	2	1	1	2	3		1	2	2	1	3	1	NO	STABLE
24	30	2	32	1	2	180/100	1	1	2	1	2	3+	2.47	23	13	417	2	4.7	2	2	ABRUPTIO	2	1	2	1	3		1	2	2	1	2	1	4	STABLE
25	26	1	39	2	2	170/110	1	2	2	2	2	3+	3.44	21	12	244	2	5.1	2	1		2	1	1	1	0		1	2	2	1	6	2	NO	STABLE
26	30	1	36+3	2	2	160/110	1,4	2	2	1	2	3+	2.71	29	15	450	2	6.5	2	2	MSL	2	1	1	1	0		1	2	2	1	3	1	2	STABLE
27	23	1	39	2	2	160/110	3	2	2	2	2	3+	2.42	16	12	281	2	7.3	2	2	SUSPICIOUS TRACE	2	1	1	2	0		1	2	2	1	4	2	NO	STABLE
28	30	1	39+3	2	2	160/100	0	2	2	1	2	2+	1.81	23	10	471	2	3	2	2	MSL	2	1	1	2	0		1	2	2	1	6	1	2	STABLE
29	22	2	37+4	2	2	170/120	1	1	2	1	2	3+	2.3	20	7	450	2	7.4	2	2	SEV PE	1	1	1	2	0		1	2	2	1	2	1	4	STABLE
30	20	2	36+5	2	1	170/100	1	2	2	1	2	2+	2.24	52	29	351	2	4.5	2	1		2	1	1	2	0		1	2	2	1	5	2	NO	STABLE
31	21	2	40+2	2	1	160/100	1,4	2	2	1	2	3+	96000	50	187	350	2	8.9	2	2	PARTIAL HELLP	2	2-INC RESIST	1	1	12		1	1	2	1	6	1	1	STABLE
32	21	2	40+2	2	1	160/100	3,4	2	2	2	2	1+	96000	50	187	189	2	8.9	2	2	SEV PE WITH PARTIAL HELLP	2	1	1	2	12		1	1	2	1	6	1	1	STABLE
33	23	2	28+4	1	1	170/100	1	2	2	1	2	2+	23900	28	32	560	2	5.6	2	1		2	2-AEDF	1	1	6		1	1	2	3	1	0	NO	FSB
34	37	2	33+4	2	2	160/100	1,3	2	2	2	2	1+	2.52	30	10	396	2	7.2	2	2	PREV LSCS	1	1	1	2	0		1	2	2	1	3	2	NO	STABLE
35	24	2	28	1	1	180/110	1	2	2	1	2	3+	2.79	21	18	269	2	9.3	2	2	SEV PE WITH UNCONTROLLED HTN	2	2-AEDF	1	1	10		1	2	2	1	2	1	4	STABLE
36	36	2	39+1	2	2	160/100	3	1	2	1	2	1+	2.05	23	14	260	2	6.9	2	2	BREECH IN LABOUR	2	1	1	2	0		1	1	2	1	5	2	NO	STABLE
37	29	1	35	2	2	160/100	4	2	2	1	2	3+	1.07	48	36	472	2	7.2	2	1		1	2-INC RESIST	1	2	0		1	2	1	1	2	1	3	STABLE
38	29	1	35	2	2	160/100	0	2	2	1	2	3+	1.07	48	36	477	2	7.2	2	2	SEV PE WITH UNCONTROLLED HTN	2	1	1	1	0		1	2	2	1	2	1	3	STABLE
39	30	2	31+5	1	1	170/110	0	1	2	1	2	3+	1.32	43	27	436	2	6.9	2	1		2	1	1	2	0		1	2	1	3	2	0	NO	FSB
40	20	2	38+1	1	1	180/90	3	1	2	1	2	2+	1.93	14	74	334	2	9.7	2	2	UNCONTROLLED HTN WITH IMMINENT SIGNS	2	1	1	1	0		1	1	2	1	5	2	NO	STABLE
41	27	1	36+2	2	2	160/100	1,4	2	2	2	2	2+	1.45	90	10	213	2	6.9	2	2	SEV PE WITH UNCONTROLLED HTN	2	1	1	1	0		1	2	2	1	5	2	NO	STABLE
42	22	2	32+2	1	1	170/100	0	2	2	1	2	1+	2.1	15	8	337	2	4.8	2	2	SEV FGR WITH PATHOLOGICAL TRACE	1	2-INC RESIST	2	2	0		1	2	2	1	2	1	3	STABLE
43	30	1	38+1	2	2	170/100	0	2	2	2	2	2+	2.48	16	15	299	2	3	2	1		1	1	1	2	10		1	2	2	1	4	2	NO	STABLE
44	30	2	34+1	2	1	180/90	1,3	2	2	1	2	2+	1.3	21	14	275	2	5.1	2	2	SEV PE	2	1	2	1	0		1	2	1	1	4	1	5	STABLE
45	26	1	38+1	2	2	160/100	0	2	2	1	2	1+	2.09	16	17	257	2	3.4	2	2	SUSPICIOUS TRACE	1	1	1	2	0		1	2	2	1	3	1	2	STABLE
46	27	2	31+1	1	2	160/100	1,3	2	2	2	2	3+	2.22	18	20	401	2	11.6	2	2	FGR WITHAEDF	1	2-AEDF	1	2	0		1	2	2	1	2	1	3	EXPIRED(D2)
47	22	1	38	2	2	160/100	0	2	2	1	2	1+	1.23	23	11	353	2	6.9	2	2	PREV LSCS	2	1	2	2	0		1	2	2	1	5	2	NO	STABLE
48	24	2	36	2	2	180/90	0	2	2	2	2	1+	2.24	18	30	280	2	5.2	2	2	PREV LSCS WITH SEV PE	2	1	1	2	0		1	2	2	1	4	1	8	CDH
49	40	1	36+6	2	2	170/110	1,4	2	2	1	2	3+	1.92	21	12	327	2	6.9	2	2	SEV PE	2	1	1	2	0		1	2	2	1	4	1	1	STABLE
50	30	2	34+5	2	2	162/100	1	2	2	2	2	2+	2.37	25	15	436	2	8.9	2	2	SEV WITH PREV LSCS	2	1	1	2	0		2	2	2	1	3	1	1	STABLE
51	22	2	39	2	1	160/110	0	2	1	2	2	2+	85000	203	80	315	2	5.6	2	2	HELLP	2	2-AEDF	1	1	6		1	1	2	1	5	2	NO	STABLE
52	20	2	35+2	2	1	190/100	1,3	2	2	1	2	3+	1.25	31	17	630	2	6.9	2	2	SEV PE WITH UNCONTROLLED HTN	2	2-INC RESIST	1	1	0		1	2	2	1	3	1	1	STABLE
53	28	1	32+6	1	2	160/90	1	2	2	1	2	3+	23000	63	27	409	2	6.2	2	1		2	1	1	1	6		1	1	1	1	3	1	4	STABLE
54	22	2	28+6	1	1	170/110	1	2	2	1	2	4+	1.83	50	37	424	2	7	2	2	IMMINENT ECLAMPSIA	2	2-AEDF	1	1	0		1	2	2	1	1	1	4	EXPIRED(D3)
55	29	1	38+3	2	2	160/110	0	2	2	2	2	2+	2.22	20	14	251	2	6.9	2	2	SEV WITH PREV LSCS	2	1	1	2	0		1	2	2	1	5	2	NO	STABLE
56	21	1	34+1	2	1	160/100	0	2	2	1	2	2+	1.23	20	18	233	2	6.9	2	2	UNCONTROLLED HTN WITH IMMINENT SIGNS	2	1	1	2	0		1	2	1	1	4	2	NO	STABLE
57	28	2	36	2	2	190/100	1,4	2	2	2	2	2+	3.09	32	32	363	2	7.6	2	2	ABRUPTIO	2	1	1	1	3		1	2	2	1	4	2	NO	STABLE
58	30	2	35+2	2	2	180/110	1,4	2	2	2	2	2+	98000	35	32	495	2	6.5	2	1		2	1	1	1	12		1	1	2	2	2	0	NO	MSB
59	24	2	24+5	1	1	180/110	0	2	1	2	1	1+	49000	139	208	471	2	6.2	2	1		2	2-AEDF	1	2	6		1	1	2	3	1	0	NO	FSB
60	29	1	34+3	2	2	180/100	0	2	2	1	2	2+	2.2	19	17	252	2	5.4	2	2	PREV LSCS WITH SEV PE	2	1	1	2	0		1	2	1	1	5	2	NO	STABLE
61	30	1	36+5	2	2	180/120	1,4	2	2	1	2	2+	2.06	13	8	258	2	2	2	2	PREV LSCS WITH SEV PE	1	1	1	1	0		1	2	2	1	4	2	NO	STABLE
62	32	1	38+6	2	2	160/110	1	2	2	1	2	1+	1.5	15	7	436	2	3.4	2	2	SEV PE WITH TRANSVERSE LIE	2	1	1	2	0		1	2	2	1	6	2	NO	STABLE
63	31	1	37+2	2	2	160/100	0	2	2	1	2	2+	1.96	19	11	400	2	5.7	2	2	SEV WITH PREV LSCS	2	1	1	2	0		1	2	2	1	4	2	NO	STABLE
64	20	2	32+3	1	1	180/90	0	2	2	2	2	2+	2.23	37	16	267	2	7.8	2	1		2	1	1	2	0		1	2	1	1	3	2	NO	STABLE
65	25	2	39+1	2	2	180/120	0	2	2	1	2	2+	1.49	18	10	300	2	7.5	2	2	SEV PE WITH UNCONTROLLED HTN	2	1	1	1	10		1	1	2	1	6	2	NO	STABLE
66	33	2	38+5	2	1	170/110	1	2	2	1	2	2+	1.78	29	18	244	2	6.5	2	2	SEV PE WITH UNCONTROLLED HTN	2	1	1	1	0		1	2	2	1	6	2	NO	STABLE
67	19	1	37+2	2	1	160/120	1,2,3	2	2	1	2	2+	86000	38	21	357	2	5.3	2	2	PARTIAL HELLP	2	1	1	1	12		1	1	2	1	4	2	NO	STABLE
68	22	2	39+4	2	1	180/110	0	2	2	1	2	2+	3	18	3	393	2	6.4	2	2	SEV PE WITH FETAL DISTRESS	1	1	1	1	0		1	2	2	1	4	1	1	STABLE
69	25	2	30+5	1	1	160/100	1,3	2	2	1	2	2+	1.85	16	32	376	2	6.8	2	1		1	2-AEDF	1	2	0		1	1	2	3	1	0	NO	FSB
70	29	2	32	1	1	160/100	0	2	2	2	2	2+	1.5	14	9	200	2	8	2	2	FGR WITHAEDF	1	2-AEDF	1	2	0		1	2	2	1	2	1	5	STABLE
71	23	1	38+3	2	2	160/100	0	2	2	2	2	1+	2.5	19	10	425	2	5.9	2	2	PREV LSCS	1	1	1	2	0		1	2	2	1	4	2	NO	STABLE
72	26	2	37	2	1	190/110	1,2,4	2	2	1	2	2+	1.71	24	14	462	2	5.6	2	2	SEV PE WITH IMMINENT SIGNS	1	1	1	1	13		1	2	2	1	4	2	NO	STABLE
73	23	2	38+4	2	1	190/110	1	2	2	2	2	3+	2.22	49	29	301	2	6.6	2	2	BREECH IN LABOUR	2	1	1	1	0		1	2	2	1	4	2	NO	STABLE
74	28	2	37+1	2	2	160/100	0	1	2	1	2	1+	2.55	16	22	480	2	6.2	2	2	ABRUPTIO	1	1	1	2	3		1	1	2	1	4	2	NO	STABLE
75	25	1	38+2	2	1	160/100	0	1	2	1	2	1+	38000	15	9	239	2	5.5	2	2	UNCONTROLLED HTN WITH IMMINENT SIGNS	2	1	1	1	10		1	1	2	1	4	2	NO	STABLE
76	28	1	33+1	1	2	150/110	0	1	2	2	2	1+	2.37	46	36	399	2	5.8	2	2	SEV PE	2	1	1	2	10		1	1	2	1	3	1	4	STABLE
77	26	2	38+4	2	1	160/100	0	2	2	1	2	3+	90000	78	120	921	2	6.4	2	2	HELLP	2	1	1	1	6		1	1	2	1	5	2	NO	STABLE

78	20	1	39+5	2	1	160/90	1,2,3,4	2	2	2	2	2+	1.96	60	10	277	2	5.5	2	2	SEV PE WITH IMMINENT SIGNS	2	1	1	1	0		1	2	2	1	5	1	8	STABLE
79	30	2	32+2	1	1	160/120	0	2	2	1	2	2+	1.22	20	13	296	2	6.6	2	2	SEV PE WITH IMMINENT SIGNS	2	1	1	1	0		1	2	2	1	2	1	2	STABLE
80	22	2	35	2	2	170/112	4	1	2	1	1	3+	26000	202	167	538	2	9.3	2	2	HELLP	2	1	1	1	6		1	1	2	1	3	2	NO	STABLE
81	**24**	1	36	2	2	160/100	0	2	2	2	2	1+	3.13	27	14	224	2	4.9	2	2	CDMR	2	1	1	2	0		2	2	2	1	5	2	NO	STABLE
82	25	2	40+2	2	2	190/120	1,2	2	2	2	2	3+	3.33	31	15	367	2	5.6	2	2	SEV PE WITH IMMINENT SIGNS	2	2-INC RESIST	1	1	0		1	2	2	1	5	2	NO	STABLE
83	26	2	33+4	1	1	160/100	0	2	2	2	2	2+	2.42	14	14	384	2	6.8	2	2	FAILED INDUCTION	2	1	1	1	0		1	2	2	2	6	0	NO	IUD
84	26	1	38+2	2	1	160/100	0	2	2	2	2	2+	1.42	20	10	275	2	6.9	2	1		2	1	1	2	0		2	2	2	1	4	2	NO	STABLE
85	21	2	39+2	2	1	170/110	3	2	2	1	2	3+	45000	435	326	978	2	8.1	1	2	HELLP	1	1	1	1	1,2,6,7		1	1	2	1	5	2	NO	STABLE
86	21	1	39+4	2	1	160/100	0	1	2	1	2	3+	2.13	0.7	30	234	2	6.7	2	2	SEV FGR WITH PATHOLOGICAL TRACE	2	1	1	2	0		1	2	2	1	4	2	NO	STABLE
87	25	2	37+5	2	2	160/100	0	1	2	1	2	3+	1.23	31	12	359	2	6.9	2	2	PREV LSCS WITH ABRUPTION	2	1	1	1	3,8		1	1	2	2	3	0	NO	IUD
88	23	1	35+6	2	2	160/90	3	2	2	1	1	2+	1.77	228	18	423	2	6.4	2	1		2	1	1	1	0		1	2	1	1	4	2	NO	STABLE
89	26	1	38+4	2	1	160/100	0	2	2	2	2	2+	2.42	14	14	384	2	6.8	2	2	FAILED INDUCTION	2	1	1	1	0		1	1	2	2	6	0	NO	IUD
90	35	2	30+6	1	2	180/110	0	2	2	1	2	2+	2.71	20	14	292	2	4.3	2	2	SEV PE WITH AEDF	2	2-AEDF	1	2	0		1	2	2	1	2	1	1	EXPIRED(D3)
91	20	1	38+6	2	2	160/100	0	2	2	2	2	2+	2.21	19	9	234	2	6	2	1		2	1	1	2	0		2	2	2	1	5	2	NO	STABLE
92	22	1	39+4	2	1	160/112	1,3	2	2	1	2	2+	1.33	16	30	234	2	5.8	2	2	UNCONTROLLED HTN WITH IMMINENT SIGNS	2	1	1	1	0		1	2	2	1	6	2	NO	STABLE
93	20	2	32+4	1	1	160/110	0	2	2	2	2	3+	1.59	42	25	555	2	8.2	2	2	ABRUPTION	2	1	1	2	3		1	1	2	2	2	0	NO	IUD
94	30	1	39+3	2	1	176/106	0	1	2	1	1	2+	2.26	25	14	252	2	5	2	2	SEV PE WITH UNCONTROLLED HTN	1	2-AEDF	1	1	0		1	1	2	1	4	2	NO	STABLE
95	24	1	34	2	2	170/110	0	2	2	1	2	2+	1.48	66	27	717	2	5.6	2	2	SEV PE	2	1	1	1	8,10		1	1	2	1	4	2	NO	STABLE
96	26	1	38	2	2	168/108	0	2	2	2	2	1+	3.14	27	30	356	2	7.2	2	2	PREV LSCS WITH UNCONTROLLED HTN	2	1	1	1	0		1	2	2	1	6	2	NO	STABLE
97	32	2	32+4	1	2	160/100	1,2,3,4	2	2	1	1	2+	1.26	33	26	384	2	5	2	2	SEV PE WITH AEDF	2	2-AEDF	1	2	0		1	2	2	1	2	1	4	STABLE
98	20	2	28+3	1	1	160/100	0	2	2	2	2	2+	68000	47	22	647	1	7.9	2	2	SEV PE WITH HELLP	2	1	1	2	6,7		1	1	2	1	2	1	4	EXPIRED(D11)
99	39	2	28+5	1	2	160/90	1,2,4	2	2	2	2	3+	1.16	16	28	420	2	6.3	2	1		2	2-AEDF	1	1	0		1	2	2	3	1	0	NO	FSB
100	24	1	31+1	1	2	180/110	1,3	2	2	2	1	1+	1.72	18	13	427	2	7.9	2	1		2	1	1	2	0		1	2	2	1	2	1	4	STABLE
101	22	1	38	2	1	180/110	1,2,3,4	2	2	1	2	1+	2.1	90	12	363	2	3.78	2	2	IMMINENT ECLAMPSIA	2	1	1	1	1		1	2	2	1	3	1	3	STABLE
102	19	2	33+2	1	1	170/110	0	1	1	1	2	4+	42000	61	177	942	1	6.8	2	1		2	2--AEDF	2	1	3,6,7		1	1	2	3	3	0	NO	FSB
103	22	1	30+1	1	1	160/90	3	1	2	1	2	1+	4.5	27	13	342	2	8.3	2	1		2	1	1	2	0		2	1	2	1	2	1	4	STABLE
104	27	1	33+5	1	2	180/110	1,3	2	2	1	2	1+	2.15	28	17	340	2	5.7	2	2	SEV PE WITH IMMINENT SIGNS	2	1	1	1	0		1	2	2	1	3	2	NO	STABLE
105	20	2	31+6	1	2	160/110	0	2	2	1	2	1+	22000	57	118	2894	1	6	2	2	SEV PE WITH HELLP	1	2-AEDF	1	1	6,7,8		1	1	2	1	2	1	4	EXPIRED(D3)
106	25		28+6	1	2	160/110	0	1	2	1	2	1+	2.07	34	12	275	2	6.5	2	2	SEV PE	2	1	1	1	0		1	2	1	1	2	1	4	STABLE
107	28		34	2	1	160/110	0	2	2	2	2	3+	2.17	20	12	224	2	3.7	2	2	SEV PE	2	2-INC RESIST	1	1	0		1	2	2	1	3	1	3	STABLE
108	23	2	28+4	1	1	170/100	1,2	2	2	1	2	2+	23900	28	32	280	2	5.6	2	1		2	1	1	1	3,6		1	1	1	2	2	0	NO	MSB
109	25		36+4	2	1	160/110	1,2,3	2	2	2	2	4+	2.26	25	12	361	2	12.6	2	2	IMMINENT ECLAMPSIA	2	1	1	1	1		1	2	2	1	4	1	5	STABLE
110	30		39+5	2	2	180/110	0	2	2	2	2	3+	1.06	30	60	240	2	6	2	2	CPD WITH UNCONTROLLED HTN	2	1	2	1	0		1	2	2	1	5	2	NO	STABLE
111	22		32+4	1	2	170/100	1,3,4	1	2	1	1	3+	1.06	27	9	344	2	6.8	2	2	SEV PE WITH IMMINENT SIGNS	1	1	1	1	0		1	1	2	1	2	1	4	STABLE
112	33		31+1	1	2	160/100	1,2	2	2	1	1	1+	2.16	19	11	337	2	3.6	2	2	SEV PE WITH IMMINENT SIGNS	2	1	1	1	0		1	2	1	1	3	1	4	STABLE
113	27		29+4	1	1	170/110	1,3,4	2	2	2	1	2+	1.19	56	45	310	2	6.9	2	1		2	1	2	2	0		1	2	2	1	1	1	4	EXPIRED(D4)
114	29		25+4	1	1	160/100	0	2	2	1	2	3+	3.02	17	10	306	2	6.08	2	1		2	2-AEDF	2	1	0		1	1	2	3	1	0	NO	FSB
115	22		36+1	2	1	150/110	3	1	2	2	2	1+	2.68	24	15	195	2	6.4	2	2	SEV PE	2	1	1	2	0		2	2	2	1	4	1	3,4,5	STABLE
116	22		33+6	1	1	160/110	0	2	2	1	2	3+	1.43	18	12	323	2	3.7	2	2	SEV FGR WITH PATHOLOGICAL TRACE	2	1	1	1	3		1	2	2	1	2	1	4	STABLE