

**“CLINICAL STUDY ON DEMOGRAPHIC PROFILE,
MATERNAL AND PERINATAL OUTCOME IN
PREGNANT WOMEN WITH HYPERTENSIVE
DISORDERS”**

Dissertation submitted for

M.S., DEGREE EXAMINATION

M.S. OBSTETRICS AND GYNAE COLOGY

BRANCH II



**CHENGALPATTU MEDICAL COLLEGE,
CHENGALPATTU**

**THE TAMIL NADU DR.M.G.R. MEDICAL UNIVERISTY
CHENNAI – TAMILNADU**

MAY - 2018

CERTIFICATE

This is to certify that the dissertation titled **“CLINICAL STUDY ON DEMOGRAPHIC PROFILE MATERNAL AND PERINATAL OUTCOME IN PREGNANT WOMEN WITH HYPERTENSIVE DISORDERS”** is a bonafide work done by **DR.P.SUGANTHI** in **CHENGALPATTU MEDICAL COLLEGE**, during the academic year 2015-2018 submitted to the **TAMILNADU Dr.M.G.R. Medical University** in partial fulfillment of University regulation for M.S. Branch - II obstetrics and Gynaecology degree examination of The Tamilnadu Dr.M.G.R Medical University.

Dr.USHA SADHASIVAN M.D.,Ph.D.,
The DEAN
Chengalpattu medical college& Hospital
Chengalpattu-603 001

CERTIFICATE BY THE HEAD OF THE DEPARTMENT

This is to certify that the dissertation titled “**CLINICAL STUDY ON DEMOGRAPHIC PROFILE MATERNAL AND PERINATAL OUTCOME IN PREGNANT WOMEN WITH HYPERTENSIVE DISORDERS**” is a bonafide work done by **Dr.P.SUGANTHI** in the **CHENGALPATTU MEDICAL COLLEGE**, during the academic year **2015-2018** under My direct supervision and guidance, submitted to the Tamilnadu Dr. M. G. R. Medical University in partial fulfillment of University regulations for **M.S.Branch- II** Obstetrics and Gynaecology degree examination of the **Tamilnadu Dr.M.G.R.Medical University**.

Place: Chengalpattu

Date:

Prof.Dr.SAMPATHKUMARI M.D.D.G.O

Head of the Department

Department of obstetrics and Gynaecology

Chengalpattu medical college

Chengalpattu.

CERTIFICATE BY THE GUIDE

This is to certify that the dissertation titled “**CLINICAL STUDY ON DEMOGRAPHIC PROFILE MATERNAL AND PERINATAL OUTCOME IN PREGNANT WOMEN WITH HYPERTENSIVE DISORDERS**” is a bonafide work done by **DR.P.SUGANTHI** in **CHENGALPATTU MEDICAL COLLEGE** during the academic year 2015-2018 under my direct supervision and guidance, submitted to the Tamilnadu **Dr. M.G.R. Medical University** in partial fulfillment of University regulation for M.S. Branch II Obstetrics and Gynaecology degree examination of the Tamilnadu Dr. M.G.R. Medical University.

Place:Chengalpattu

Date:

Prof.Dr.SAMPATHKUMARI. M.D.D.G.O

Head of the Department

Department of obstetrics and Gynaecology

Chengalpattu medical college

Chengalpattu.

DECLARATION BY THE CANDIDATE

I, **DR.P.SUGANTHI** solemnly declare that the dissertation titled **“CLINICAL STUDYON DEMOGRAPHIC PROFILE MATERNAL AND PERINATAL OUTCOME IN PREGNANT WOMEN WITH HYPERTENSIVE DISORDERS”** has been prepared by me. I also declare that this bonafide work or a part of his work was not submitted by me or any other for any award, degree, diploma to any other University board either in India or abroad. This is submitted to The Tamilnadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the rules and regulation for the award of M.S. Degree Branch- II Obstetrics and Gynaecology.

Place: Chengalpattu

Signature of the candidate

Date:

[DR.P.SUGANTHI]

ACKNOWLEDGEMENT

I humbly submit this work to the Almighty who has given the health and ability to pass through all the difficulties in the compilation and proclamation of my dissertation.

My sincere thanks to our **DEAN, Dr.USHA SADHASIVAN M.D., Ph.d**, Chengalpattu Medical College, Chengalpattu for granting me permission to undertake the clinical study in the hospital.

First , I would like to express my sincere gratitude to my beloved HOD , My **Prof.Dr.SAMPATHKUMARI M.D.,D.G.O.**, Professor and Head of the department of obstetrics and Gynaecology, Chengalpattu for the able guidance and encouragement all along in completing my study.

I sincerely thank my **ASSOCIATE PROFESSOR Dr. THENMOZHI, and ASSISANT PROFESSORS Dr.MALINI, Dr.SREEPRITHIKA, Dr.VINITHRA, Dr.SHRUTHI** for giving me practical suggestions and permitting me to carry out this study in our patients. Had it not been for her whole hearted support throughout the period of this study, extending from vast knowledge, invaluable advice and constant motivation, I truly would not have been able to complete this dissertation topic in its present form.

I am thankful to **Dr. NIVEDITHA CRRI** for her kind co operation in helping me to do this study.

I am indebted to all teaching staffs and colleagues of my department for their valuable suggestions and auxillary attitude and extremely thankful to all the patients who were the most important part of the study.

I would like to thank the institutional Ethical Committee for approving my study.

I thank my parents and all the family members who have been solid pillars of everlasting support and encouragement and for their heartfelt blessings.

Urkund Analysis Result

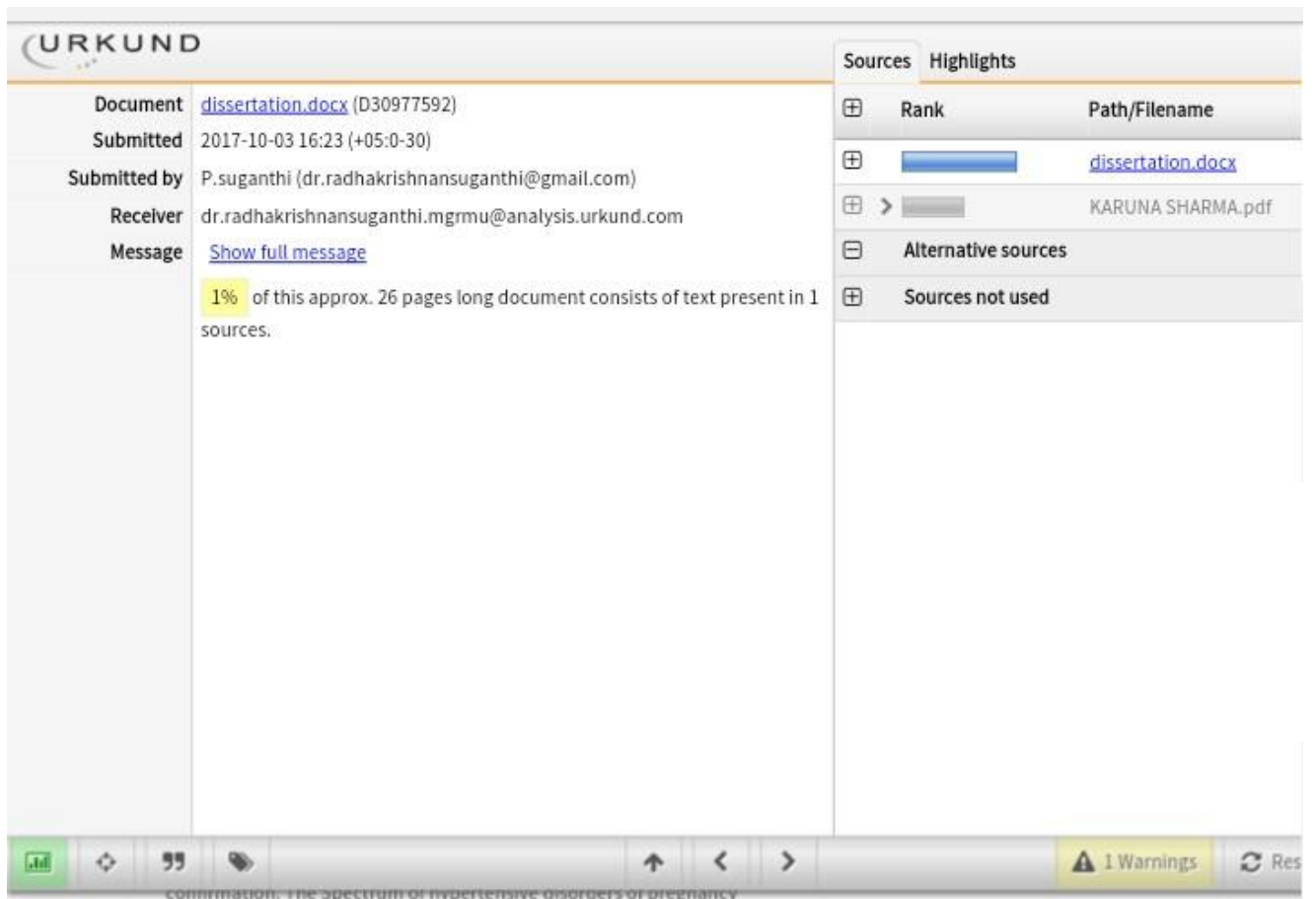
Analysed Document: dissertation.docx (D30977592)
Submitted: 10/3/2017 12:53:00 PM
Submitted By: dr.radhakrishnansuganthi@gmail.com
Significance: 1 %

Sources included in the report:

dissertation.docx (D30977553)

Instances where selected sources appear:

2



The screenshot displays the Urkund analysis interface. On the left, a metadata panel shows details for the document 'dissertation.docx (D30977592)', including the submission date (2017-10-03 16:23), the submitter (P. suganthi), and the receiver (dr.radhakrishnansuganthi.mgrmu@analysis.urkund.com). A message snippet indicates that 1% of the document's text is derived from sources. On the right, a 'Sources' panel is active, showing a table with columns for Rank and Path/Filename. The table lists 'dissertation.docx' as the primary source and 'KARUNA SHARMA.pdf' as an alternative source. Below the table, there are sections for 'Alternative sources' and 'Sources not used'. At the bottom of the interface, a status bar shows navigation icons, a warning icon with the text '1 Warnings', and a refresh icon labeled 'Res'.

Document	dissertation.docx (D30977592)
Submitted	2017-10-03 16:23 (+05:0-30)
Submitted by	P. suganthi (dr.radhakrishnansuganthi@gmail.com)
Receiver	dr.radhakrishnansuganthi.mgrmu@analysis.urkund.com
Message	Show full message

Rank	Path/Filename
1	dissertation.docx
2	KARUNA SHARMA.pdf

1% of this approx. 26 pages long document consists of text present in 1 sources.

1 Warnings

CERTIFICATE

This is to certify that this dissertation work titled **“CLINICAL STUDY ON DEMOGRAPHIC PROFILE , MATERNAL AND PERINATAL OUTCOME IN PREGNANT WOMEN WITH HYPERTENSIVE DISORDERS “** of the candidate **Dr. P. SUGANTHI** with registration number 221516252 for the award of M.S., in the branch of **OBSTETRICS AND GYNAECOLOGY**. I have personally verified urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains introduction to conclusion pages and the result shows 1 percentage of plagiarism in the dissertation.

**GUIDE AND SUPERVISOR
SIGNATURE**

INSTITUTIONAL ETHICAL COMMITTEE

CHENGALPATTU MEDICAL COLLEGE, CHENGALPATTU

Title of Work : **Clinical Study on Demographic Profile Maternal and Perinatal Outcome in Pregnant women with Hypertensive Disorder**

Principal Investigator : Dr.P.Suganthi, MS.,

Designation : Post graduate in Obstetrics and Gynaecology,

Co-Investigators : 1.Dr.S.Nesam Susanna Minnalkodi, MS.,
Professor & HOD of Obstetrics and Gynaecology,
2.S. Sampathkumari, MS.,
Associate professor of Obstetrics and Gynaecology,

Department : Obstetrics and Gynaecology

The request for an approval From the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on.25.10.2016 at the Medical Education Unit, Government Chengalpattu Medical College, Chengalpattu at 12.00 PM.

The Members of the committee, the Secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

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

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


MEMBER SECRETARY,

24/12/17
IEC, CHENGALPATTU MEDICAL COLLEGE
CHENGALPATTU.


DEAN

24/12/17
CHENGALPATTU MEDICAL COLLEGE
CHENGALPATTU

CONTENTS

S.NO	TITLES	PAGE NO
1	INTRODUCTION	1
2	REVIEW OF LITERATURE	3
3	AIMS & OBJECTIVES	40
4	MATERIALS & METHODS	41
5	RESULTS & ANALYSIS	49
6	DISCUSSION	77
7	CONCLUSION	82
8	LIMITATIONS	83
9	<ul style="list-style-type: none">• ANNEXURES• BIBLIOGRAPHY• ABBREVIATIONS• PROFORMA• INFORMATION SHEET• CONSENT FORM• MASTER CHART	

INTRODUCTION

Hypertension during pregnancy is one of the well known complications dating from ancient times. It remains as one of the leading causes of maternal mortality and morbidity through its myriad complications.

This common medical disorder of pregnancy represents about 7 to 15% of all pregnancies and accounts for nearly 25% of admissions in antenatal wards¹. And hypertensive disorders account for 16% of maternal mortality worldwide, according to the WHO analysis of causes of maternal death². Of the studies conducted in India, the prevalence of gestational hypertension was found to be about 7.8%³.

However this figure is increasing as women postpone their first pregnancy and also because of increasing metabolic complications like obesity. But, the incidence of complications is decreasing in developed nations due to better antenatal services. Apart from the various deleterious antenatal complications, pregnancy induced hypertension also poses a life long risk for the woman by transforming itself into chronic hypertension and also by increasing the woman's cardiovascular risk. Hypertension during pregnancy along with proteinuria also adversely affects the neonate's health by being the leading cause of stillbirth, fetal growth restriction, low birthweight and admissions to neonatal intensive care units. For a disorder with such vast implications, it is a relatively easier condition to diagnose provided a woman is

given proper antenatal care and blood pressure monitoring. Hence, the importance of studying this disorder in great lengths arises.

This study was undertaken to evaluate the demographic profile and the present status of maternal and perinatal outcomes throughout the spectrum of hypertensive disorders of pregnancy including mild, severe preeclampsia, eclampsia and chronic hypertension at Chengalpattu Medical College Hospital, Chengalpattu in a period of 1 year from October 2016 to September 2017.

REVIEW OF LITERATURE

National High Blood Pressure Education Program Working Group (NHBPEP 2000) working along with American College of Obstetricians and Gynecologists (ACOG) has set the definition and classification for the ‘hypertension during pregnancy’⁴. Accordingly, Hypertension in Pregnancy is defined as Systolic pressure more than 140 mmHg and/or Diastolic pressure more than 90 mmHg (Korotkoff V/Disappearance of sound). Two pressure readings taken at 4 to 6 hours apart are needed for confirmation.

The Spectrum of hypertensive disorders of pregnancy consists of the following five:

- Gestational Hypertension
- Preeclampsia
- Eclampsia
- Preeclampsia superimposed on Chronic hypertension
- Chronic Hypertension

Gestational Hypertension is the elevated blood pressure of more than 140/90 mmHg with its first onset noted after 20 weeks of gestation. It may also be noted during labour or in the first 24 hours postpartum but there must be no proteinuria. The elevated blood pressure has to resolve to normal within 12 weeks postpartum and hence the final diagnosis is made only postpartum.

Blood pressure is measured in upright or left lateral recumbent position. Appropriate bladder cuff encircling two-thirds of the arm has to be used at the level of heart. Mercury sphygmomanometers remain the gold standard for measurement. Earlier, a raise of 30 mmHg systolic or 15 mmHg diastolic above the baseline used to be a diagnostic criterion but is now abandoned⁵.

It is termed as Preeclampsia when gestational hypertension is associated with proteinuria. Proteinuria is defined as excretion in the urine of more than 300 mg protein in 24 hours or 1+ by reagent strips (30mg/dl) in atleast 2 random urine samples 4 to 6 hours apart. A spot urine protein:creatinine ratio is also currently preferred and has to be a value >0.3

Even in the presence of proteinuria, it is classified as Severe Preeclampsia when the pressure exceeds 160/110 mmHg and the proteinuria more than 2g per 24 hours or 2+ by reagent strips. The presence of imminent symptoms like persistent headache, persistent epigastric pain and oliguria also qualify for classification under severe preeclampsia. These persistent symptoms, earlier referred to as ‘imminent eclampsia’ are nothing but the manifestations of multiorgan involvement of the disease. In addition, microangiopathic hemolysis, elevated liver enzymes, elevated serum creatinine (>1.2 mg/dl), reduced platelets ($<1,00,000/\mu\text{l}$) and pulmonary edema characterize severe preeclampsia.

When a patient with preeclampsia presents with convulsions that cannot be attributed to other causes, then it is Eclampsia.

Chronic hypertension is when hypertension (blood pressure of more than 140/90 mmHg) is diagnosed preconceptionally or before 20 weeks of gestation and remains elevated after 12 weeks postpartum. Naturally, it may either be essential hypertension with no underlying cause or secondary hypertension with some underlying cause like renal, endocrine or vascular disorders.

It is termed as Preeclampsia superimposed on chronic hypertension when a woman who was previously a hypertensive presents in pregnancy after 20 weeks with features of preeclampsia such as new-onset proteinuria or even worsening of already existing proteinuria/hypertension.

INCIDENCE

Hypertensive disorders of pregnancy totally represent about 7 to 15% of all pregnancies and makes up about 25% of antenatal admissions¹. These also account for 16% of maternal mortality worldwide, according to the WHO analysis of causes of maternal death². In India, the prevalence of gestational hypertension is around 7.8%³. Describing the trends of maternal mortality in United States, in around the year 1930 when the mortality figure was around 1%, hypertension during pregnancy made a sizable 20% of it. As of now, the mortality has dropped down and is around 15/100,000. But hypertension still contributes a whopping 10% of these maternal deaths⁶. Coming to figures from UK, hypertensive disorders are the second leading cause maternal mortality and make up about one-third of antenatal morbidity⁷.

RISK FACTORS FOR PREECLAMPSIA

1. Maternal Age

Increasing maternal age in itself is a risk factor for preeclampsia. The incidence of preeclampsia is around 9.4% in women aged above 35 years in comparison to the figure of 6.4% in younger women according to the study by Lamminpa et al in the year 2012⁸. Of strategic importance is also increasing incidence in women of the other extreme of age-the teenagers. The study by Robillard and Halsey outline the predisposition of teenagers to develop the condition⁹.

2. Parity

Preeclampsia is essentially a disease of first pregnancy. Women who have previously had a successful or failed pregnancy are at reduced risk of developing preeclampsia. Mac Gillivray reported that primigravidae have 15 times more chances of developing proteinuria¹⁰.

3. Family history of Preeclampsia

Genetic inheritance is a key factor to be noted while studying the risk factors for preeclampsia. The predisposition to develop preeclampsia which is genetically inherited was studied by Chesley and Cooper¹¹. Another study by Cincotta et al¹² demonstrated that the risk of severe preeclampsia is about four times higher in primigravidae with family history. John Studd¹³ has also pointed out that the risk of developing preeclampsia is about 25% and eclampsia is about 3% for daughters born to mothers with history of preeclampsia.

4. Race and ethnicity

Excluding the lifestyle, education and other demographic factors, ethnicity in itself alone has been found to be a risk factor for developing preeclampsia according to Bouthoornsh et al¹⁴.

5. Paternity

Primipaternity is also considered an important factor in the incidence of preeclampsia. Not only is the first pregnancy at increased risk of developing preeclampsia, but also the first pregnancy by the current partner has to be taken into account according to the study by Deswriet et al¹⁵. Paternal risk factor is also highlighted by the fact that fathers who have already fathered a preeclamptic pregnancy are at nearly twice the risk of again fathering a preeclamptic pregnancy with another woman

6. Obesity

Obesity remains a risk factor for many afflictions of pregnancy including hypertensive disorders of pregnancy. It is believed that with each 5 to 7 kg/m² increase in BMI, the risk of developing preeclampsia doubles

7. Obstetric factors

The study of preeclampsia risk factors is incomplete without considering the other obstetric risk factors that might complicate the pregnancy such as

- Multiple gestation
- Preeclampsia/Gestational hypertension in prior pregnancy
- Hydatidiform mole

- Hydrops fetalis
- Abnormal uterine artery Doppler at 18-24 weeks

Studies dedicated to twin gestation have been done which indicate the increased risk of preeclampsia, which is about 13% when compared to the 5% in singleton pregnancy in a study by Sibai et al¹⁶.

8. Pre-existing medical disorders

- Naturally chronic hypertension is an important risk factor for developing preeclampsia with an increased risk of 25-50% more than normal pregnancies
- Diabetes mellitus is the next important condition which along with gestational hypertension contributes to a combined incidence of 30%
- Autoimmune disorders
- Renal diseases
- Thrombophilias
 - ◆ Antiphospholipid antibody syndrome
 - ◆ Factor V Leiden deficiency
 - ◆ Activated Protein C resistance
 - ◆ Hyperhomocysteinemia

9. Placental and Fetal factors:

Impaired placentation is the most important risk factor for preeclampsia which is at the crux of its pathogenesis. Inadequate autophagy and poor placentation as causative factors for preeclampsia is explained by Saito et al¹⁷. In addition as mentioned above, multiple pregnancies is associated with higher risk¹⁶. Also fetal abnormalities such as hydrops fetalis¹⁸, hydatidiform mole, trisomies and triploidy and advancing gestational age also contribute to increased risk.

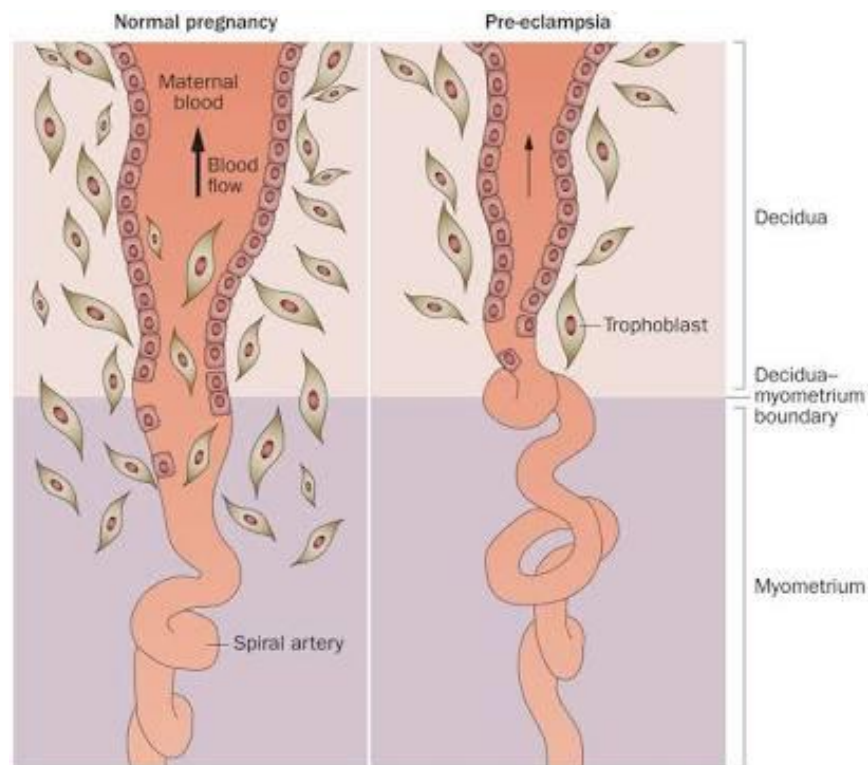
ETIOPATHOGENESIS

Etiopathogenesis of this common disorder stands elusive in spite of its wide prevalence. Several theories have been put forth, none of which is completely satisfactory. However, the most widely accepted root cause is that of abnormal placentation explained by the Two-Stage Disorder Hypothesis. The two stages are:

- Stage 1: Incomplete migration of the endovascular trophoblastic cells in the spiral arteries resulting in decreased uteroplacental perfusion
- Stage 2: The above explained defective uteroplacental perfusion resulting in the maternal syndrome of preeclampsia

ABNORMAL PLACENTATION

The migration of the endovascular cytotrophoblastic cells into the endothelium and smooth muscle layer of spiral arterioles in the placenta is a vital event that occurs in normal pregnancies in the first trimester. It is because of this important event that the spiral arterioles are converted into low resistance channels which is essential for maintaining the vascularity of the placenta. This is completed by about mid-pregnancy in the second trimester.



In Preeclampsia however, the process is maladapted. The migration that takes place in two stages falters in the second stage due to defective differentiation of the trophoblasts which succeed in only penetrating the endothelium and not the muscle layer. Thus, the spiral arterioles maintain their limited caliber. Also they remain responsive to vasomotor influences.

PLACENTAL HYPOPERFUSION AND HYPOXIA

The above mentioned placental maladaptation obviously leads to placental hypoperfusion and hypoxia. In addition, this stage is also further modified by other factors that can reduce placental perfusion such as diabetes, connective tissue disorders and so on. Thus, the repeated and prolonged drop in placental perfusion in turn releases placental debris into the intervillous spaces of maternal circulation. These products thus released spike maternal endothelial dysfunction.

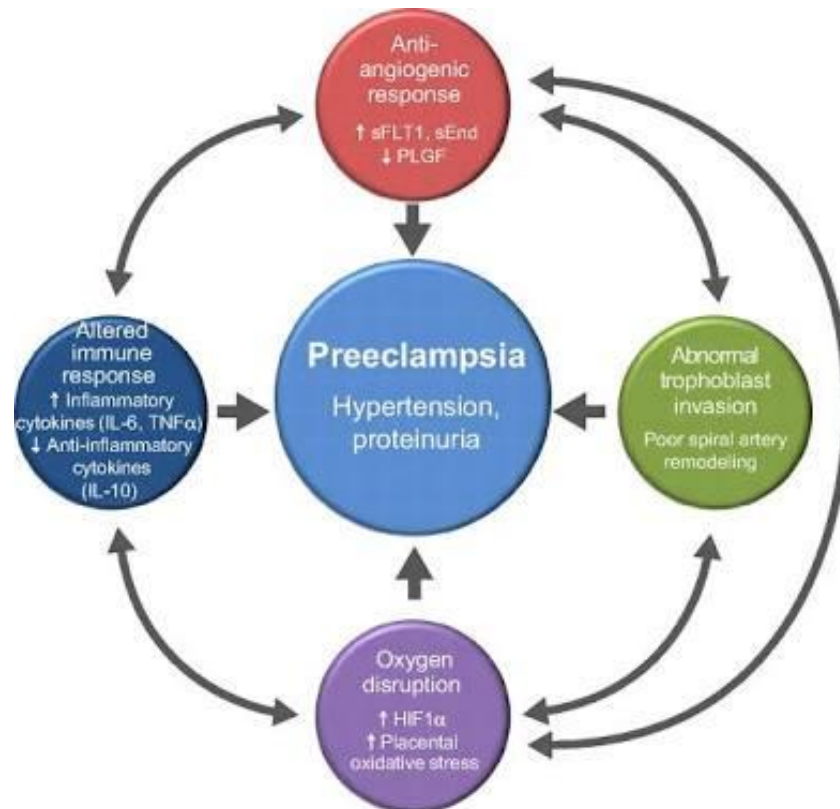
ENDOTHELIAL DYSFUNCTION AND VASOSPASM

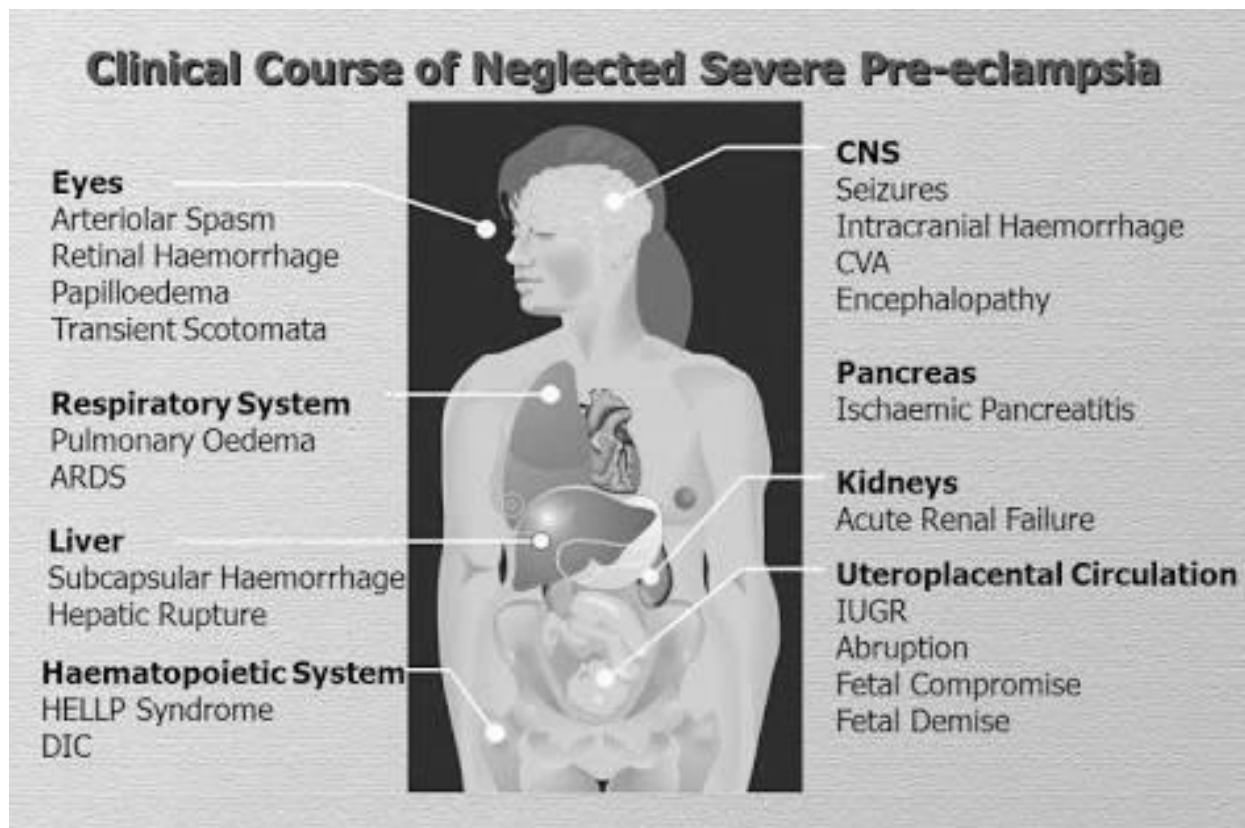
The normal balance between the proangiogenic factors like VEGF (vascular endothelial growth factor) and PlGF (placental growth factor) and antiangiogenic factors like soluble Fms-like tyrosine kinase-1 (sFlt-1) is essential for optimal vascularity in pregnancy. However in case of preeclampsia, this balance is tipped in balance of excess antiangiogenic factors by the gradual uteroplacental hypoxia. Thus the resultant excessive sFlt-1 is responsible for the decreased production of the vasodilatory prostaglandins (PGI₂) and Nitric oxide which causes vasospasm, the main event behind preeclampsia

Also contributing to endothelial dysfunction is the inflammation caused by cytokines like TNF α and Interleukins on the endothelium. These cytokines are released by the syncytiotrophoblastic debris of the placenta provoking

inflammation. These cytokines cause an oxidative stress by forming free radicals.

The combined effect of endothelial inflammation and drop in the level of vasodilators lead to platelet aggregation and vasospasm. Platelet aggregation along with microvascular coagulation results in thrombi formation and thrombocytopenia. On the other hand, the intense and generalized vasospasm leads to increased vascular resistance and decreased organ perfusion which leads to necrosis, hemorrhage and other manifestations of end-organ failure.





IMMUNOLOGIC FACTORS

An immunologic tolerance to the paternal and fetal antigens is very much essential in a normal pregnancy in order to facilitate the implantation of placenta and further growth of fetus. However due to an error in this immune tolerance that occurs in preeclampsia, the normal trophoblastic invasion that occurs is error prone and hence predisposed to develop preeclampsia.

GENETIC FACTORS

Genetic predisposition to preeclampsia is a well established one. MTHFR gene coding for methylene tetrahydrofolate reductase and Factor V Leiden gene have been found to coexist with thrombophilia genes. In addition, the NOS3

gene plays an important role in the etiology of preeclampsia since it encodes for the placental nitric oxide production which is affected here.

PATHOLOGY

HEMODYNAMIC CHANGES

The major point to be noted is that the normal increasing cardiac output and decreasing peripheral resistance of pregnancy is inverted in preeclampsia. With worsening preeclampsia, there is decrease in cardiac output and increase in peripheral resistance. Also there is an increased vascular sensitivity to vasopressors.

The severe vasospasm along with microvascular leak shifts the fluid from intravascular to interstitial compartment. This fluid leak along with reduced oncotic pressure (caused by proteinuria and resultant protein loss) leads to the pathological edema of preeclampsia. In addition, the loss of intravascular fluid leads to relative hypovolemia along with hemoconcentration which is the trademark of severe preeclampsia. It is this hypovolemia which also contributes in the causation of growth restriction, oligohydramnios and preterm labour.

HEMATOLOGICAL CHANGES

The high cardiac output in severe preeclampsia along with the increased peripheral resistance causes traumatic lysis of the red blood corpuscles leading to microangiopathic hemolytic anemia. The other common hematological

abnormality noted is drop in the platelet count; which on exceeding the 1,00,000/ μ l mark denotes severe preeclampsia. These both combined together is noted in HELLP syndrome, a dreaded complication of preeclampsia.

Though thrombocytopenia and consumptive coagulopathy are obvious complications noted here, overt coagulation defects are rare and are observed in cases of abruption alone. This rules out the need for regular use of coagulation investigations like PT, aPTT and plasma fibrinogen in cases of preeclampsia.

RENAL CHANGES

The trademark lesion that is found in preeclampsia is ‘Glomerular capillary endotheliosis’ which is nothing but the enlargement of the endothelial cells of the glomerular capillaries which is evident on electron microscope. This in turn leads to a reduced blood flow and hence glomerular enlargement. The reduced renal blood flow in turn leads to drop in glomerular filtration.

The proteinuria that typically characterizes preeclampsia is due to increased vascular permeability to proteins in the glomeruli. This along with drop in glomerular filtration cause increased levels of serum creatinine and uric acid levels.

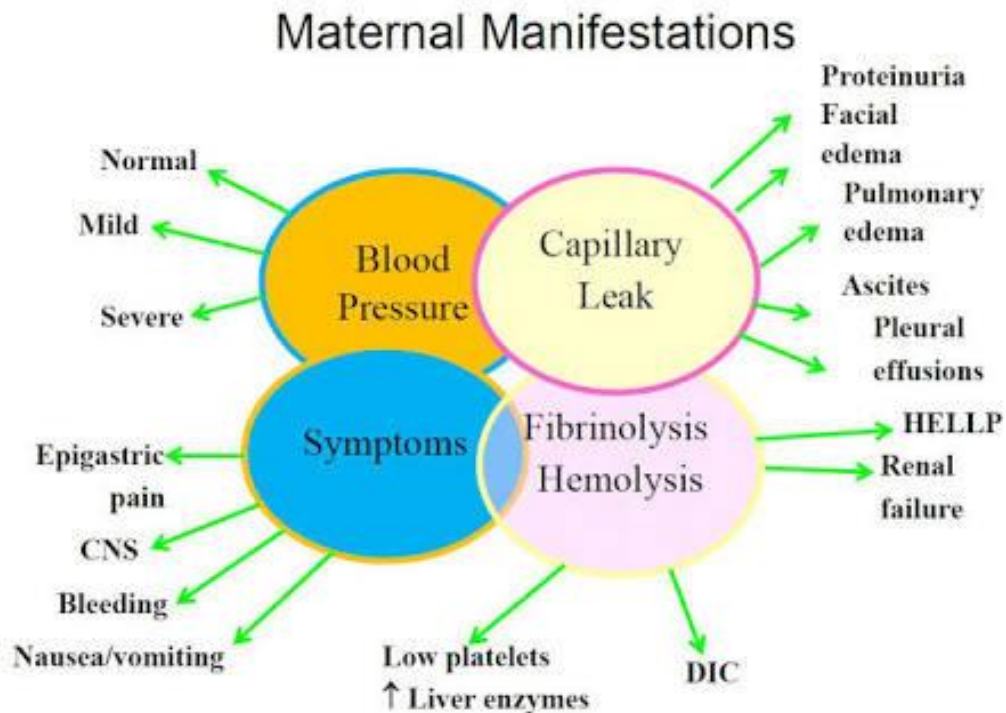
In addition, the rennin-angiotensin system gets activated due to the renal pathology which in turn leads to sodium and chloride retention.

Acute renal failure (ARF) that occurs in preeclampsia is almost always due to acute tubular necrosis (ATN). This occurs whenever there is profuse hemorrhage and also hypovolemia and hypotension.

HEPATIC CHANGES

The intense vasospasm in the hepatic sinusoids along with fibrin deposition result in infarction and periportal hemorrhages. These hemorrhagic infarctions result in the formation of hematoma beneath the Glisson's capsule, the so called subcapsular hematoma.

The hematoma thus formed in need of space expands and stretches the Glisson's capsule, thus causing the characteristic epigastric pain of severe preeclampsia. In addition, the infarct and cellular damage to hepatic cells cause release of hepatic enzymes into the blood, thus elevating the blood levels of SGOT and SGPT which also indicates severe preeclampsia.



As long as the hemorrhage remains subcapsular, the patient also remains hemodynamically stable but in severe pain. But when the hemorrhage is combined with DIC, the hemorrhage becomes major and may even lead to liver rupture.

CENTRAL NERVOUS SYSTEM CHANGES

The obvious and dreaded neurologic complication of preeclampsia is Eclampsia. This occurs in the setting of intense vasospasm leading to ischemia and infarction of neurons. Also more than half of eclamptic women have intracranial bleed (both cortical and subcortical hemorrhages) which is due to uncontrolled hypertension. In addition, the widespread endothelial damage causes vascular leak and thus perivascular edema. Thus, the pathology involves softening, necrosis, infarct and hemorrhages. Vasospasm occurring in different

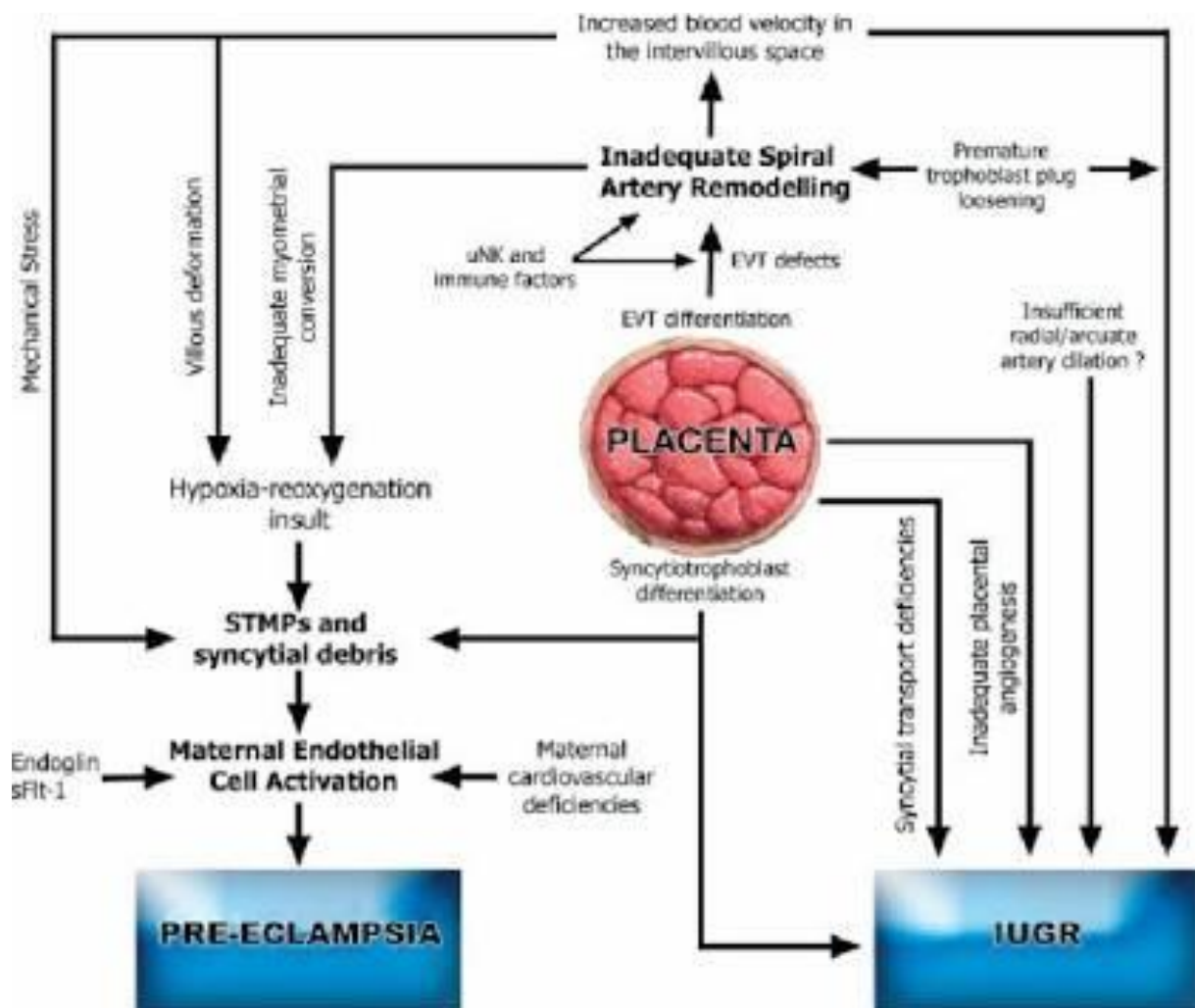
areas of motor cortex lead to different manifestations: severe headache unrelieved by analgesics when frontal area is involved, cortical blindness when occipital area is involved and henceforth. Thus control of blood pressure within normal limits is very vital to prevent cerebrovascular accidents. Widespread cerebral edema also occurs which can manifest as coma and confusion.

Eden's criteria are used for assessing the severity of eclampsia and also used as indications for CT scan. They are:

- Coma of >6 hours
- Temperature > 39⁰ C
- Pulse rate > 120/min
- Systolic BP >200 mmHg
- Respiratory rate >40/min
- More than 10 convulsions

PLACENTAL CHANGES

The basic underlying pathology of incomplete trophoblastic invasion that occurs in the placenta is responsible for the placental changes in preeclampsia which occur even before the clinical manifestations are evident. The narrow and necrosed spiral arteries along with the compromised uterine blood flow are the main culprits in causing fetal growth restriction and preterm labour.



Also characteristic changes in the placenta include increased apoptosis and necrosis of syncytiotrophoblast. Thus, increased syncytial knots, cytotrophoblastic proliferation, fibrinoid necrosis and calcified and hyalinised villi are the findings observed under microscopy¹⁹.

PREDICTIVE TESTS FOR PREECLAMPSIA:

CLINICAL HISTORY

Detailed clinical history taken at the time of antenatal booking serves as the first step in evaluating a patient for preeclampsia. The points that raise suspicion in favour of preeclampsia include advancing maternal age, primiparity, primipaternity, multiple gestation, a previous history of preeclampsia, antiphospholipid antibodies and so on. These risk factors already discussed above along with routine measurement of blood pressure in clinical visits help in clinching an early diagnosis²⁰.

CLINICAL TESTS

Mean Arterial Pressure of more than 90 mmHg measured in the second trimester is predictive of preeclampsia since this value normally drops at this point of time. MAP is calculated from diastolic and pulse pressure. Though the sensitivity of mean arterial pressure is very low²¹ in the range of 0-9%, its specificity is comparatively high in the range of 53-97%. This is the initial step in predicting preeclampsia.

Roll over test which is done between 28 to 32 weeks is a routine test which is recommended for the early diagnosis of gestational hypertension²². This test involves determining the difference in blood pressure that is noted

when the patient is in supine position and she rolls over to left lateral position. A difference in diastolic pressure of more than 20 mmHg is considered positive.

Hand grip test is another test which is advocated for predicting preeclampsia. It involves maintaining the pressure at 200 mmHg for 3 mins by holding the BP cuff in the hand. Later, the diastolic pressure is reassessed. A rise of more than 20 mmHg is considered positive. The sensitivity of the test in prediction of preeclampsia is around 80% while the specificity is 92%²³. However the test is found unsuitable when employed as a screening test because of its time consuming nature. It is limited to research purposes.

BIOCHEMICAL PARAMETERS

When microalbuminuria is detected around 18 weeks by measuring urine albumin excretion of more than 30 mg in 24 hours, it can serve as an excellent predictor of preeclampsia that is about to subsequently develop²⁴.

Reduced levels of urate excretion leads to raised serum uric acid levels. Plasma uric acid level of more than 5.9 mg/dl measured at around 24 weeks has a positive predictive value of preeclampsia of around 33 percent according to study by Jacobson and colleagues (1990).

Even in the presence of normal uric acid levels, when plasma urea and creatinine values are raised, they denote renal impairment and also in a way predict preeclampsia, though they have a low predictive value.

Hypocalciuria of less than 195 mg per 24 hours is found to be a predictor of preeclampsia in later pregnancy according to Maikranz et al²⁵.

When the calcium creatinine ratio is measured, it is around 0.44 ± 0.32 in normotensive women. However the value drops and it is around 0.03 in preeclamptic women and 0.20 in chronic hypertensive women. Thus, a ratio of less than 0.06 can be said to be strongly indicative of preeclampsia. However this has a false positive rate of around 33% according to Saudan et al²⁶.

Also, plasma fibronectin is found to be elevated in women with preeclampsia and eclampsia as early as first trimester itself according to a study by Ballegeer et al²⁷.

When inactive urinary Kallikrein to creatinine ratio is measured in a random urine sample which is collected between 16 to 20 weeks of pregnancy, it is found to have a predictive value in the diagnosis of preeclampsia²⁸.

Since nitric oxide is implicated in the pathogenesis of preeclampsia, when its metabolites such as nitrite/nitrate are measured in urine, their total urinary excretion can give an idea about the severity of preeclampsia according to the study by Begum et al²⁹.

HEMATOLOGICAL TESTS

As discussed in the pathology of preeclampsia, hematological alterations that occur in preeclampsia can be measured. Such tests include platelet count,

hematocrit, hemoglobin levels, RBC morphology and coagulation profile. Also, D-dimer and Antithrombin 3 levels are also measured in patients with preeclampsia. However, the test that is believed to have some predictive value is that of serum ferritin since hyperferritinemia is considered to be a result of placental damage.

PRESSOR RESPONSE TEST

As already discussed in etiopathogenesis, in addition to the abnormal placentation in preeclampsia, the spiral arterioles also retain their response to the vasopressor agents. Hence, Angiotensin Sensitivity Test (AST) where we look for the dose of infused Angiotensin II that raises the BP by 20 mmHg is thought to be a better predictor of PIH. The specificity of the test is 85% though the sensitivity is only 22%²⁷.

HORMONAL TESTS

Hyperprolactinemia is an important criterion to be considered while predicting the risk of developing preeclampsia. Both the blood pressure and prolactin secretion are influenced by diminished dopaminergic activity in the CNS³⁰.

UTERINE ARTERY DOPPLER VELOCIMETRY

However when compared to all the above, the test which is of clinical importance today is Uterine artery Doppler which is commonly done in second

trimester. Even in early gestation, a drop in the flow of uterine artery can be picked up by Doppler velocimetry. The presence of diastolic notching and a high resistance or pulsatility index or S/D ratio is suggestive of impaired blood flow³¹. However more than predicting preeclampsia, they are more useful in predicting fetal growth restriction.

NEWER MARKERS

Newer biochemical markers such as ADAM 12, free β -hCG, Inhibin A, Activin A, PP13, PIGF, PAPP-A, sFlt-1, P-selectin performed in the first trimester have shown to have a predictive value for preeclampsia. These along with Uterine artery Doppler are promising recent trends³²

LABORATORY INVESTIGATIONS:

- Complete Blood Count and blood film
- Blood urea and creatinine
- Serum uric acid
- Liver function tests
- Urine Albumin and Sugar
- Serum electrolytes
- Coagulation profile
- 24 hour urinary protein
- Lactate dehydrogenase

- Creatinine clearance
- Antiphospholipid antibodies
- Fundus opinion
- Obstetric Ultrasound with Doppler
- Plasma and urinary metanephrines
- Serum renin and aldosterone levels
- Electrocardiogram

PREVENTION OF PREECLAMPSIA:

Diet and Supplementation: Although it has been publicized that salt restriction, antioxidants, marine oils, proteins, magnesium supplementation and folic acid have a role in preventing preeclampsia, trials have failed to establish a concrete proof for the same.

According to the study by Hofmeyr et al³³, preeclampsia incidence can be reduced to half by supplementing 1.5-2 g of calcium carbonate or elemental calcium; especially for high risk women and those with low calcium intake.

Since L-Arginine is the precursor of vasodilator Nitric Oxide, it is naturally expected that supplementation of L-Arginine might reduce the incidence of preeclampsia. But again, this point lacks proof from studies.

Antioxidants such as Vit C and Vit E are expected to limit the oxidative stress, prevent endothelial injury and hence reduce the incidence of

preeclampsia. However, studies have shown little benefit and even benefit for harm.

Exercise

Though exercise and physical activity are very much linked with reduced incidence of hypertension in non-pregnant population, prospective population based studies have failed to establish a link between exercise and incidence of preeclampsia³⁴.

Low Dose Aspirin

National Institute of Clinical Excellence, UK³⁵ places a recommendation of low dose supplementation of Aspirin (75 mg) daily for high risk women in order to prevent preeclampsia. This suppresses the thromboxane synthesis in platelets and prevents microthrombi formation.

CLASP (Collaborative Low Dose Aspirin Study in Pregnancy), a large randomized controlled trial done in this respect showed a reduction of about 12% in the incidence of preeclampsia with low dose aspirin³⁶.

But, a significant reduction in the incidence of preeclampsia of about 50% was noted by Bujold et al when he started low dose aspirin before 16 weeks of gestation³⁷.

MANAGEMENT OF HYPERTENSION DURING PREGNANCY

ANTIHYPERTENSIVE DRUGS

1. Labetalol

This combined alpha and beta blocker drug is now the preferred first line therapy in the treatment of preeclampsia. The onset of action of the drug is 5 to 10 mins

Orally, the dosage of the drug is 100-400 mg twice daily for mild preeclampsia. The maximum dose of the drug is 1200 mg. In case of severe hypertension (BP>160/110 mmHg), intravenous route of the drug is given. It is started as an IV bolus of 10-20 mg. This is then followed up with 20-80 mg IV dose every 30 mins until we reach the maximum dose of 300 mg.

The drug is attractive in the fact that the decline of blood pressure is rapid but smooth and also there is no adverse effect on the fetal circulation. Also the drug rarely causes maternal hypotension and tachycardia.

But, caution has to be exercised in patients with asthma and congestive heart failure where it is preferable to avoid this drug

2. Nifedipine

This calcium channel blocker is the next drug of choice in the treatment of preeclampsia. The onset of action is 10 to 15 mins

The drug is given at a dose of 10-20 mg twice or thrice daily with the maximum dose being 180 mg per day.

Short acting Nifedipine is appealing in the fact that it gives a more rapid control of hypertension when compared with labetalol. Hence it is effective in the acute treatment of severe hypertension in pregnancy. However as with other antihypertensives, it causes maternal hypotension in addition to causing fetal distress.

3. Alpha Methyl Dopa

Alpha Methyl Dopa is a centrally acting Alpha adrenergic agonist which inhibits adrenergic receptors in the vasomotor center. When the drug is given orally, it takes 3-6 hours to act. It acts by decreasing cardiac output, peripheral resistance and renal blood flow.

The dosage of the drug is 250-500 mg twice or thrice daily to a maximum of 2g per day.

While sedation and fatigue are the common side effects, it also causes altered liver function tests, skin rash and cholestatic jaundice

4. Hydralazine

This drug acts by directly causing relaxation of the smooth muscles of the arterioles and venules. Onset of action is from 10 to 20 mins.

An intermittent bolus of 5 mg IV once in about 30 mins or an infusion at the rate of 0.5-10 mg per hour is the route in which the drug is given. The maximum dose of the drug is 30 mg.

In spite of the long experience of the drug in clinical setting, its disadvantage lies in the fact that its adverse effects like severe headache (due to

raised intracranial pressure) often mimic the features of severe preeclampsia. Also fetal heart monitoring reveals signs of abnormal fetal heart pattern in addition to low one-minute Apgar scores. Hydralazine is also known to cause maternal hypotension, placental abruption, caesarian section and oliguria³⁸.

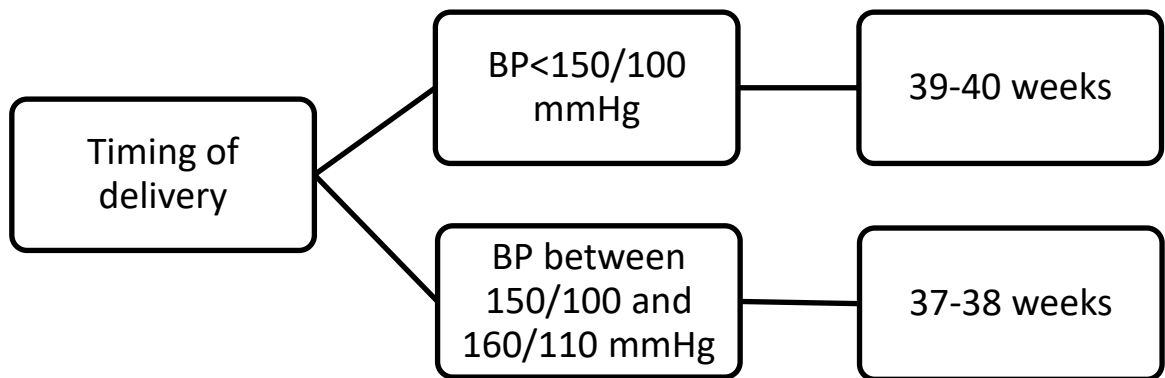
5. Sodium Nitroprusside

This is a short acting antihypertensive which relaxes both venous and arteriolar smooth muscles. Onset of action of the drug is less than one minute and its duration is limited to 1 to 3 minutes and hence the drug is valuable in its use in Hypertensive Emergencies.

The drug is given intravenously at the rate of 0.25 µg/kg/min. The maximum dose tolerated is 8 µg/kg/min.

MANAGEMENT OF GESTATIONAL HYPERTENSION

- Gestational hypertension is managed as outpatient with the patient coming for weekly antenatal visit during which we look for blood pressure, proteinuria, signs of preeclampsia, platelet count and liver enzymes.
- The patient is advised daily fetal movement monitoring/fetal kick count to which is added biweekly non-stress test and biophysical profile.
- Fetal growth monitoring is done once in 3 weeks
- Antihypertensives are given when pressure exceeds 150/100 mmHg

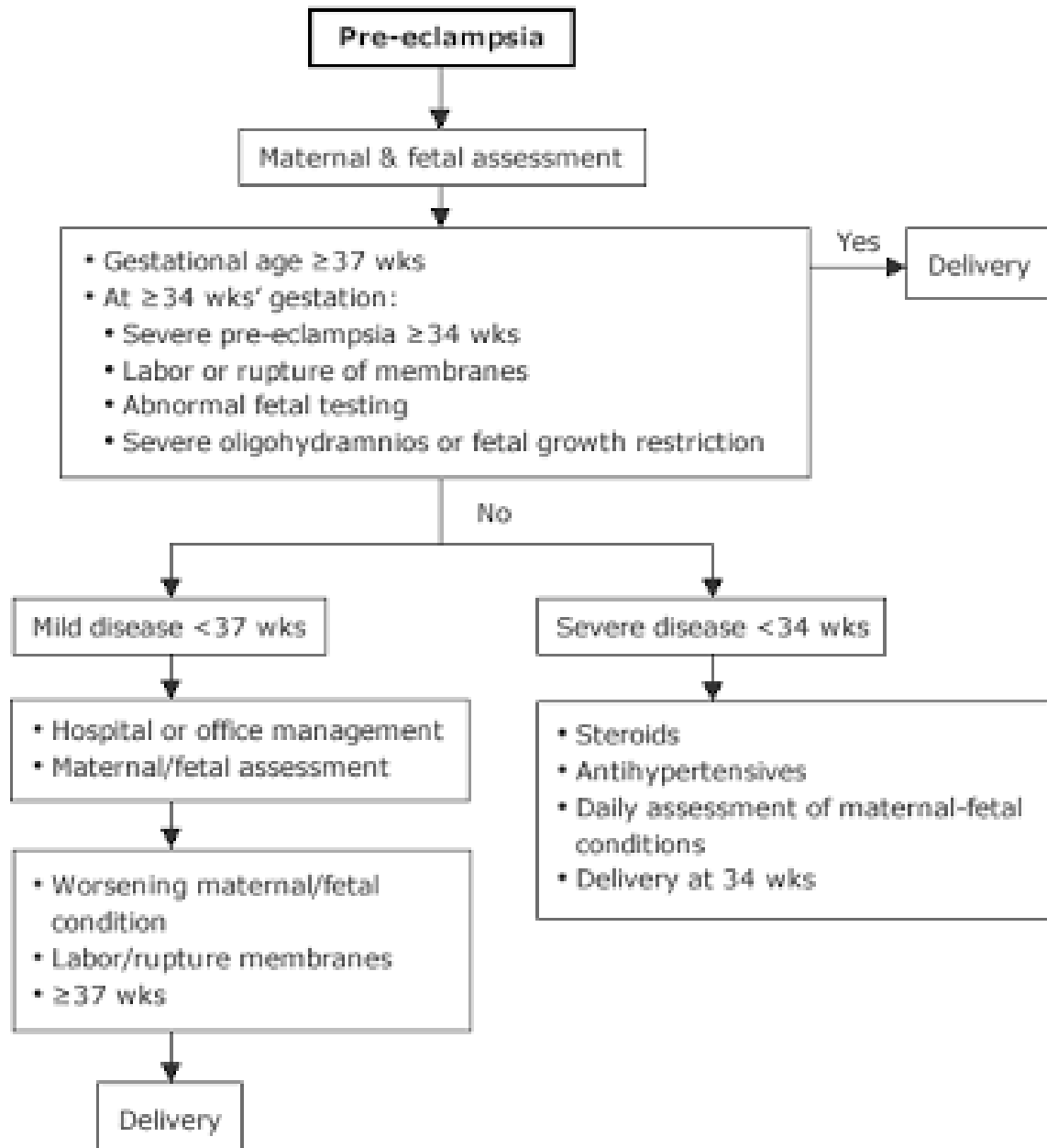


- Severe gestational hypertension is when pressure is more than 160/110 mmHg but without proteinuria. Such patients are managed as like severe preeclampsia

MANAGEMENT OF MILD PREECLAMPSIA

- Patients with mild preeclampsia are hospitalized and monitored for BP, proteinuria and signs and symptoms of severe preeclampsia.
- A reduced level of physical activity with 4-6 hours of bed rest in a day is advised to halt the progression to severe preeclampsia.
- In addition to the daily fetal movement monitoring, weekly NST and biophysical profile and fetal growth monitoring once in 2-3 weeks is recommended.
- Antihypertensive drugs are given to retain the BP within normal limits and this also decreases the risk of developing severe preeclampsia by half³⁹.

- Since prolonging the pregnancy beyond 37 weeks only increases the risk of progression to severe preeclampsia, mild preeclampsia is generally terminated at 37 weeks of gestation



MANAGEMENT OF SEVERE PREECLAMPSIA

The definitive management of severe preeclampsia lies in delivering the fetus and placenta. But when in early gestation, the risk of delivering a preterm neonate has to be weighed against the risk of continuing the pregnancy.

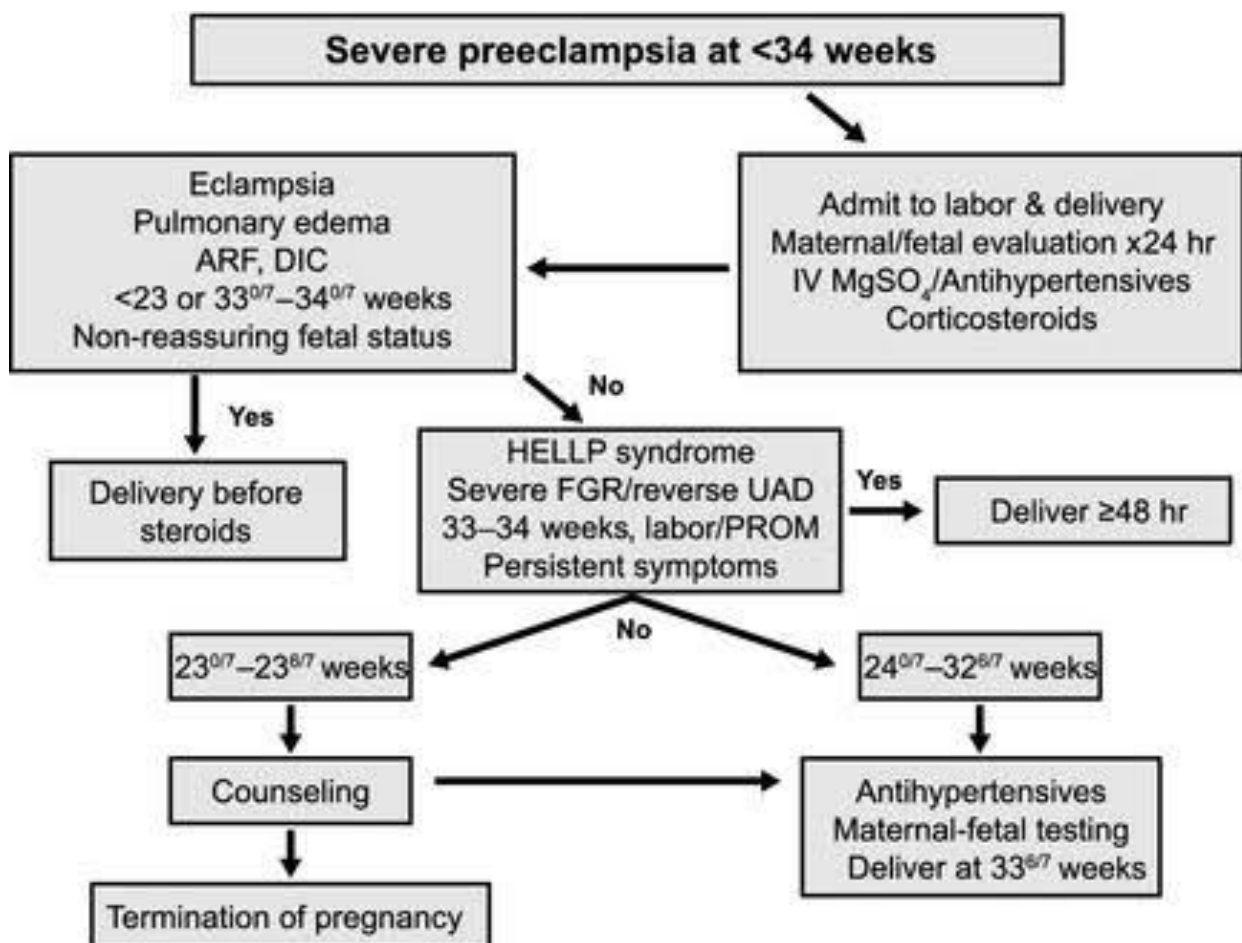
Expectant Management

- Prolongation of the pregnancy up to 34 weeks though of no benefit to the mother with even a slight complication rate of 5% improves fetal survival chances
- This has to be undertaken only in a tertiary care centre with neonatal intensive care facilities
- 4th hourly BP monitoring, strict input-output chart, daily urine protein are to be undertaken along with appropriate oral antihypertensives
- Twice weekly maternal blood count, liver and renal function tests are done
- Close fetal monitoring with daily fetal kick chart and biweekly NST and biophysical profile are done
- Elective delivery is undertaken at 34 weeks by vaginal delivery; resorting to caesarean route only when obvious obstetrical indications are present

Criteria for termination of pregnancy in Severe Preeclampsia

The expectant management is abandoned and pregnancy terminated when the following indications are present⁴⁰:

- Persistent severe hypertension unresponsive to medical therapy
- Imminent symptoms such as persistent headache, blurring of vision, right upper quadrant pain and decreased urine output.
- Eclampsia
- Thrombocytopenia ($<1,00,000/\text{mm}^3$)
- Suspected abruption placenta
- Progressive decline in hepatic and renal function
- Severe intrauterine growth restriction
- Non reassuring fetal heart rate
- Oligohydramnios



Once the patient is stabilized, close fetal surveillance and delivery is effected when the gestational age crosses 34 weeks.

MANAGEMENT OF ECLAMPSIA

Management of Eclampsia is a 3 pronged approach:

- Control of convulsions
- Control of hypertension
- Termination of pregnancy

Anticonvulsant therapy

- Initial steps of management include placing the patient in left lateral position, providing a mouth gag and using mouth suction to remove secretions and vomitus. Vitals are then monitored. Oxygen at the rate of 8-10 l/min is given to correct maternal and fetal hypoxia. If needed, IV fluids are administered but with caution lest pulmonary edema should occur.
- Magnesium Sulphate is the drug of choice for eclampsia. This drug which has a central anticonvulsant and neuroprotective effect without causing sedation or CNS depression in the mother and fetus. Maternal mortality is nearly halved in women who receive Magnesium sulphate⁴¹.
- **Pritchard's regime** is the preferred protocol which is being followed. The initial loading dose is 4g (20 ml of 20% MgSO₄) which is given as

slow IV over 4 mins which is immediately followed by 10g (20 ml of 50% MgSO₄) deep IM which is split as 5g in each buttock. The maintenance dose is 5g (10 ml of 50% MgSO₄) given as deep IM every 4 hours in alternate buttocks.

- The patients on Magnesium Sulphate are to be monitored periodically before each dose. Respiratory rate of more than 20/min, presence of patellar reflex and urine output of at least 100ml in 4 hours should be ensured; the absence of which denotes magnesium toxicity.
- The regime is given until 24 hours after delivery or 24 hours after the last seizure, whichever is later.
- Since the drug is excreted by kidneys, renal dysfunction warrants dosage adjustment. Also careful monitoring is needed to pick up magnesium toxicity and the therapeutic levels lie between 5-7 mEq/L. If at all magnesium toxicity occurs, the antidote is calcium gluconate which is given as 1g IV.
- Magnesium Sulphate also causes loss of variability in fetal heart rate which is clinically insignificant.
- Other regimens used though unpopular are Sibai regime, Zuspa regime, Cruikshank regime, Low dose regime of Sardesai and Sokoto regime.

- Though several other drugs like diazepam, phenytoin and lytic cocktail were initially advocated as alternatives for Magnesium Sulphate, these are not recommended now⁴⁰.

Antihypertensive therapy

Immediate control of blood pressure is warranted for which oral Nifedipine, IV labetalol or even IV hydralazine are employed

Obstetric management

- Occurrence of seizure causes fetal heart rate changes which recover after the episode of seizure. The woman may also be in labour. Hence, artificial rupture of membranes along with oxytocin is sufficient.
- If the patient is not in labour, the favorability of cervix is noted along with the fetal wellbeing. Then decision is taken regarding prostaglandin use to ripen the cervix or emergency shifting of patient for Caesarean section.

MATERNAL COMPLICATIONS

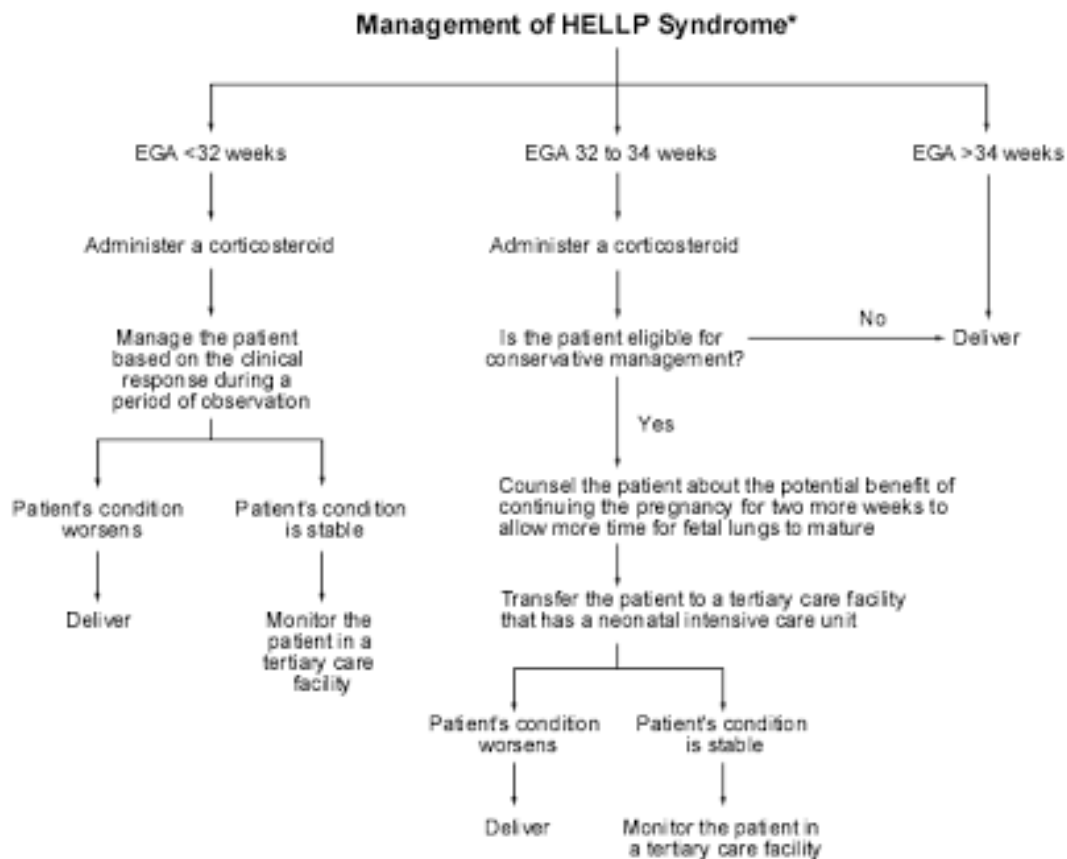
- Eclampsia is the feared complication
- Cerebral edema and cerebrovascular accidents
- Placental abruption is an important complication that can be fatal for both mother and fetus
- Pulmonary edema with or without left ventricular failure
- Microangiopathic hemolytic anemia

- Acute renal failure
- **HELLP syndrome:**

This well known complication of preeclampsia generally presents in the third trimester with an incidence of about 0.8% of all pregnancies⁴². The presence of hemolysis requires a low hematocrit, elevated bilirubin, elevated LDH (>600 IU/L) and a peripheral smear showing reticulocytes, spherocytes and schizocytes for confirmation. Along with these, we also require the evidence of elevated liver enzymes (AST and ALT) and low platelets to establish the diagnosis.

The 3 classes of HELLP syndrome are classified based on platelet counts as: Class I with platelets <50,000/mm³, Class II with counts between 50,000 to 1,00,000 and Class III with platelets between 1,00,000 and 1,50,000/mm³. Partial HELLP syndrome is a category when all the three abnormalities of HELLP (namely hemolysis, elevated liver enzymes and low

platelets) are not present together with one or two lacking.



When the diagnosis is established, platelet count and lactate dehydrogenase levels are the reliable parameters in tracking the disease status. These begin to normalize within 72 hours of delivery. Corticosteroids are given for fetal pulmonary maturity. In addition, high dose of dexamethasone (10mg IV 12th hourly) when given is found to enhance the laboratory abnormalities⁴³.

FETAL COMPLICATIONS

- Intrauterine growth restriction is an important complication as a result of drop in placental blood flow. Nearly 20% of the preterm babies and 15%

of the term babies born to preeclamptic mothers are SGA babies with birth weights less than the 10th centile.

- Prematurity is another well known complication with nearly half of all mothers with severe preeclampsia delivering preterm. Point also to be noted is that out of total preterm births, about 10% are the preterm births resulting from hypertensive disorders of pregnancy
- Birth asphyxia
- Intrauterine deaths
- Stillbirths are an important problem with hypertensive disorders being the foremost identifiable cause of stillbirths⁴⁴.

AIM OF THE STUDY

1. To study the demographic profile; risk factors, prevalence and epidemiological parameters of antenatal mothers presenting with hypertension during pregnancy
2. To determine the incidence of the different hypertensive disorders of pregnancy and study its pattern of occurrence
3. To assess the maternal and fetal outcome in pregnancies complicated with hypertension
4. To study the maternal and perinatal mortality and morbidity

MATERIALS AND METHODS

STUDY DESIGN

Descriptive study

STUDY SETTING

Department of Obstetrics and Gynaecology, Chengalpattu Medical College, Chengalpattu

STUDY PERIOD

October 2016 - September 2017

SAMPLING SIZE

Hypertensive pregnant women attending hospital during study period.

INCLUSION CRITERIA

- All pregnant women diagnosed with pregnancy induced hypertension based on the criteria defined by the working of the NHBPEP (National high blood pressure education programme)
- Gestational hypertension or pregnancy induced hypertension.
- Pre-eclampsia- eclampsia
- Chronic hypertension
- Pre-eclampsia superimposed on chronic hypertension

EXCLUSION CRITERIA

- Multiple pregnancy

- Major fetal anomaly, incompatible with survival
- Patients who were diagnosed with other causes of convulsions in pregnancy like cerebral malaria and epilepsy.
- Pregnancies complicated with diabetes mellitus, primary renal disease and collagen vascular diseases.

CASE DEFINITION

All clinically diagnosed cases of gestational hypertension with further classification based on the biochemical investigations

ETHICAL CONSIDERATIONS

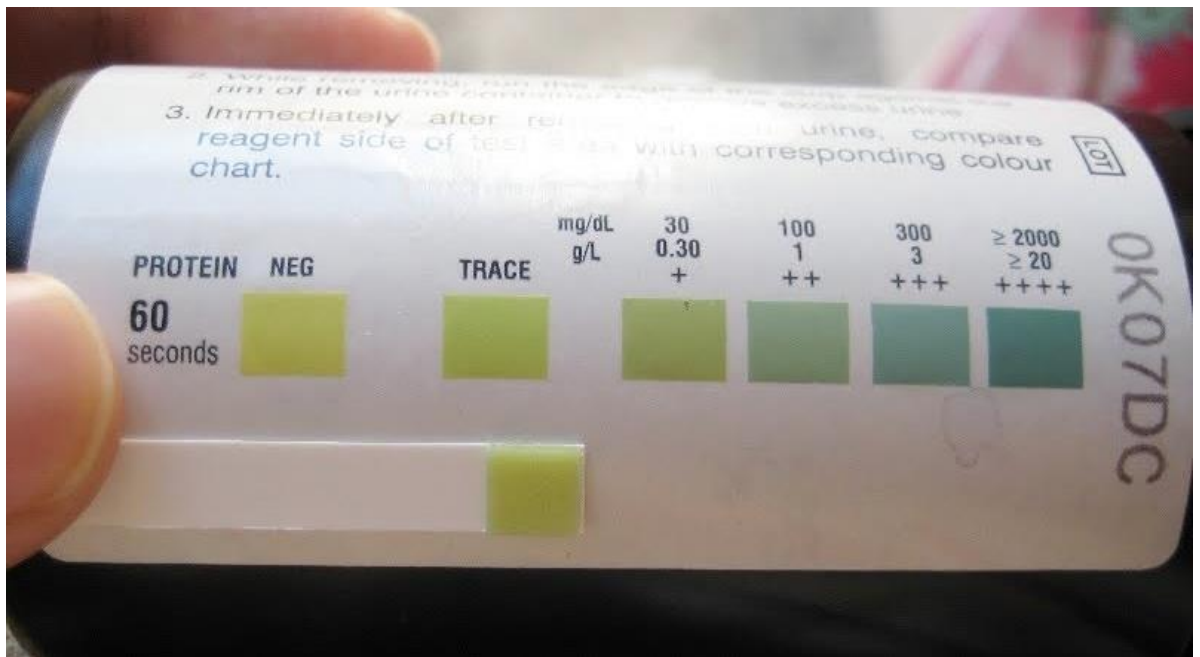
Ethical clearance was obtained from the Institutional Ethical Committee. Informed written consent was obtained from the patients of study group. Strict confidentiality was maintained throughout the study

STUDY MANOUEVRE

- All the pregnant women coming to the Antenatal OPD and Obstetric casualty were screened for hypertension and its complications. Blood pressure was measured in the sitting position in the right upper limb using mercury sphygmomanometer with arm at the level of heart. An appropriate bladder cuff was used which encircles two-thirds of the arm. Blood pressure value of more than 140/90 mmHg was followed up with another reading at an interval of 6 hours. Diagnosis was based on NHBPEP guidelines⁴.

- After carefully considering the inclusion and exclusion criteria, 450 women were selected for the study and were followed up throughout their gestation and until 2 weeks post delivery.
- Though patients were hospitalized in majority of cases, in selected cases (gestational hypertension, mild pre-eclampsia, chronic hypertension with regular blood pressure measurements) at the request of the patient, ambulatory control was allowed
- Demographic data needed for the study such as age, parity, socioeconomic status, booking status and residential area were collected by careful history taking. These along with the clinical data, laboratory results, delivery details and maternal and fetal outcomes was made note of in the pre-tested check list proforma.
- On admission, a thorough clinical examination was made including general physical examination, build, nutritional status, height, weight, BMI, blood pressure and pulse along with presence or absence of pallor and pedal edema. Weight gain during pregnancy was noted. CVS and RS were examined.
- Abdominal examination was done to determine the uterine height (in weeks), fetal lie, presentation, and fetal heart rate.

- Blood pressure monitoring was done twice daily for all patients with more frequent measurements (4th hourly) for those with elevated blood pressure.
- Daily urine albumin was monitored with the help of dipsticks supported by laboratory urine analyses.



- The patients were watched for the presence of symptoms of severe preeclampsia such as severe headache, blurring of vision, oliguria and epigastric pain. Based on all these data, patients were classified into one of the following five classes of hypertension based on NHBPEP guidelines:

Hypertensive disorders of pregnancy	
Chronic hypertension	<ul style="list-style-type: none"> • Systolic pressure ≥ 140 mm Hg &/or diastolic pressure ≥ 90 mm Hg prior to conception or 20 weeks gestation
Gestational hypertension	<ul style="list-style-type: none"> • New-onset elevated blood pressure at ≥ 20 weeks gestation • No proteinuria or end-organ damage
Preeclampsia	<ul style="list-style-type: none"> • New-onset elevated blood pressure at ≥ 20 weeks gestation AND • Proteinuria OR signs of end-organ damage
Eclampsia	<ul style="list-style-type: none"> • Preeclampsia AND • New-onset grand mal seizures
Chronic hypertension with superimposed preeclampsia	<p>Chronic hypertension AND 1 of the following:</p> <ul style="list-style-type: none"> • New-onset proteinuria or worsening of existing proteinuria at ≥ 20 weeks gestation • Sudden worsening of hypertension • Signs of end-organ damage

- Blood was collected and sent for investigations such as Complete Blood Hemogram, Liver Function tests, Renal Function tests and Serum uric acid. Urine was sent for urine albumin and sugar determination for all patients.
- Other investigations include coagulation profile, bleeding and clotting time, ECG, 24 hour urinary protein. The normal values for these investigations include:

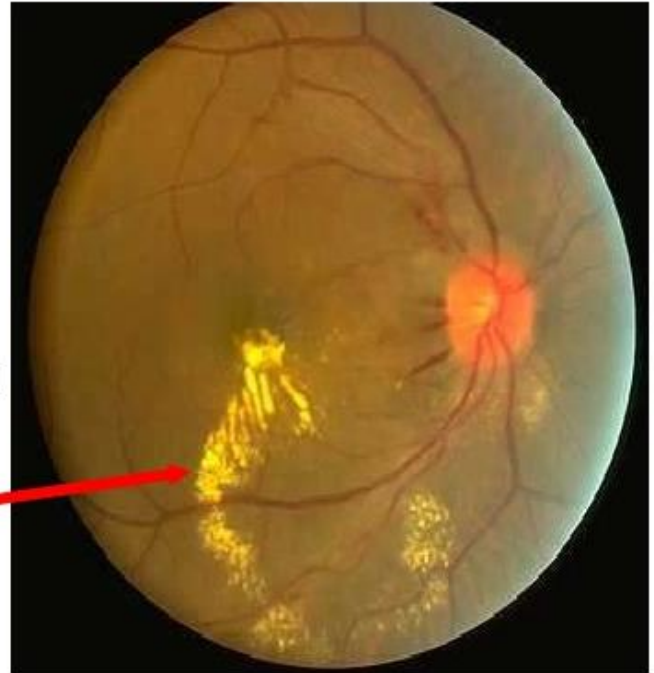
PARAMETER	NORMAL VALUES
Hemoglobin	12-16 g/dl
Hematocrit	32-36%
Platelet count	1.5-4 lakhs/cu.mm
Total Bilirubin	0.2-1 mg/dl
Indirect Bilirubin	0.1-1.0 mg/dl
Direct Bilirubin	0-0.2 mg/dl
AST/SGOT	5-43 U/L
ALT/SGPT	5-56 U/L
LDH	<600 U/L
Serum uric acid	3-7 mg/dl
Blood urea	10-15 mg/dl
Creatinine	0.7-0.9 mg/dl
PT	12-16 sec
Fibrinogen	300-600 mg%
Bleeding Time	1-3 min
Clotting Time	3-7 min

- All patients were sent for an Ophthalmic examination to study the fundus and to screen for hypertensive retinopathy



Hypertensive Retinopathy:

- **Grade I** – Thickening of arterioles.
- **Grade II** – Focal Arteriolar spasms. Vein constriction. (AV nipping)
- **Grade III** – Hemorrhages (Flame shape), dot-blot and Cotton wool (ischemia) and hard waxy exudates (lipid deposition).
- **Grade IV** – Papilloedema



- Fetal status was evaluated with daily fetal kick chart, biweekly Non Stress Test and fortnightly USG evaluation along with Uterine Artery Doppler (to rule out IUGR).
- Decision regarding the line of management of the patient was undertaken taking into consideration all of the above factors.
- Further, the patient was followed up until 2 weeks after delivery to determine the time of return of blood pressure to baseline in addition to following up the neonate for its outcome.

STATSTICAL ANALYSIS

- Primary data was entered in MS Excel and analyzed using SPSS 20v.
- The results were presented in terms of tables and graphs.
- The descriptive statistics frequency and percentage were calculated.
- The association between the categorical variables were analyzed by chi square test with 5% level of significance.

RESULTS AND ANALYSIS

According to our study, the distribution of various types of hypertensive disorders complicating pregnancy in Chengalpattu Medical College Hospital during our study period of one year from October 2016 to September 2017 is:

TABLE 1
TYPES OF HYPERTENSION

TYPE OF HYPERTENSION	NO OF PATIENTS	PERCENTAGE
Gestational HT	78	17.3%
Mild Preeclampsia	125	27.8%
Severe Preeclampsia	178	39.6%
AP Eclampsia	52	11.6%
IP Eclampsia	1	0.2%
PP Eclampsia	11	2.4%
Chronic HT	5	1.1%
Total	450	100%

Out of 450 hypertensive pregnancies, the majority were Severe Preeclampsia (39.6%). There were 78 women with Gestational Hypertension (17.3%), 125 women with Mild preeclampsia (27.8%), 178 women with Severe Preeclampsia (39.6%), 64 women with Eclampsia out of which 52 were AP Eclampsia (11.6%), 1 case was IP Eclampsia (0.2%) and 11 women were PP Eclampsia (2.4%). 5 women were Chronic hypertensives (1.1%)

FIGURE 1

TYPES OF HYPERTENSION

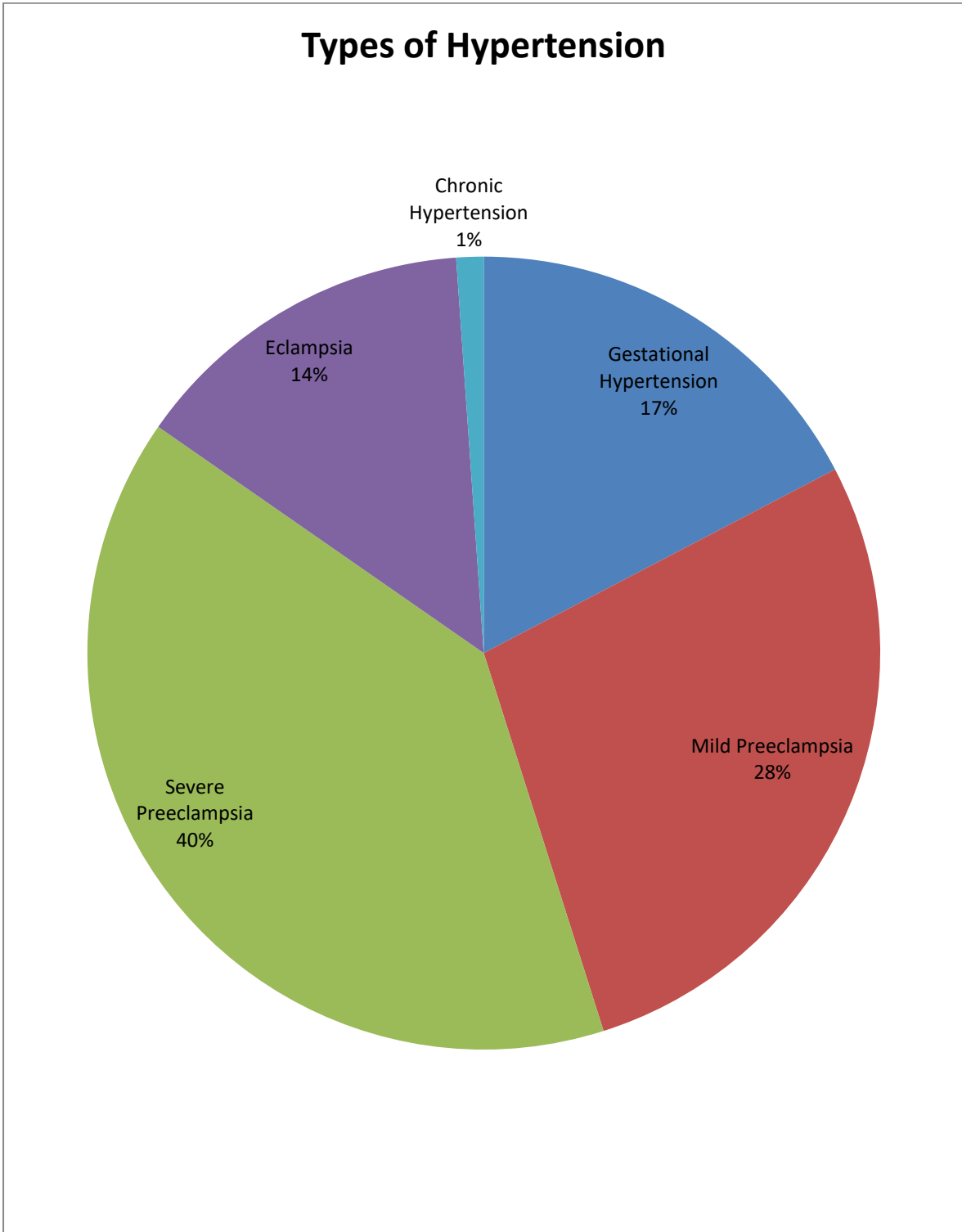


TABLE 2
AGE DISTRIBUTION

Age	No of Patients	Percentage
up to 20 yrs	95	21.1
21-25 Years	233	51.8
26 - 30 Years	103	22.9
31 - 35 Years	16	3.6
> 36 years	3	0.6
Total	450	100

FIGURE 2
AGE DISTRIBUTION

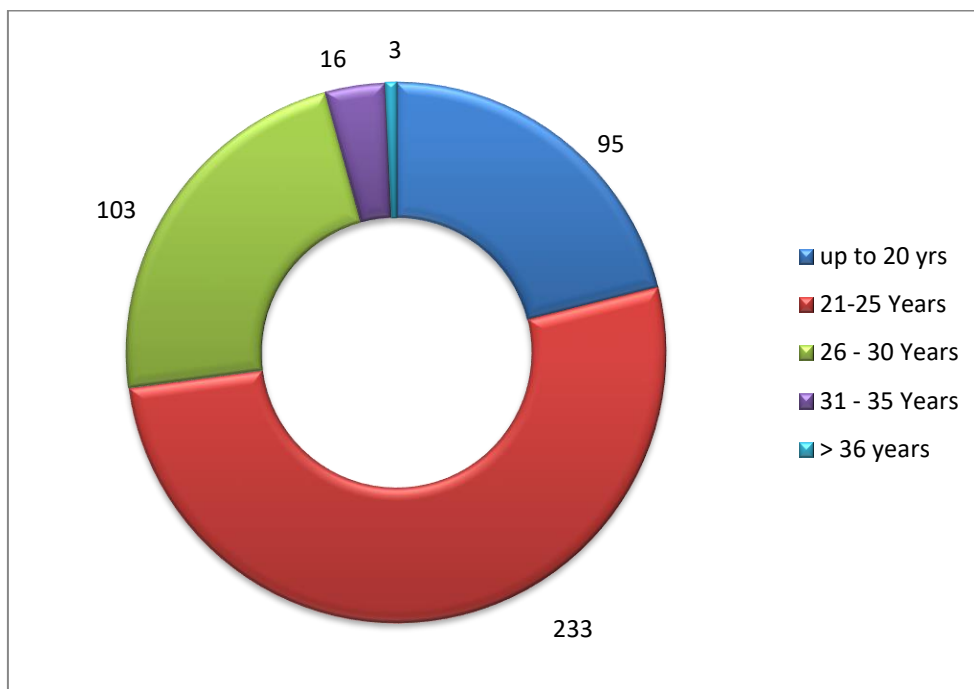


TABLE 3
AGE DISTRIBUTION IN TYPES OF HYPERTENSION

Age in years	Gestational HT	Mild Preeclampsia	Severe Preeclampsia	Eclampsia	Chronic HT	Total
<=20	13	24	38	20	0	95 (21.1%)
21-25	46	67	88	32	0	233 (51.7%)
26-30	16	30	44	11	2	103 (22.9%)
31-35	3	2	7	1	3	16 (3.6%)
>=36	0	2	1	0	0	3 (0.7%)
Total	78	125	178	64	5	450

Chi- Square value : 62.64

P value : 0.001

Statistically significant

Out of 450 patients, maximum patients (233) fall under the age group of 21-25 years (51.7%). All the forms of hypertension except Chronic hypertension most commonly occur in the 21-25 years group. Chronic hypertensives are more common in the 31-35 age group. The next common age group is that between 26-30 years with 103 patients (22.9%). The least common age group is above 36 years (0.7%).

FIGURE 3
AGE DISTRIBUTION IN TYPES OF HYPERTENSION

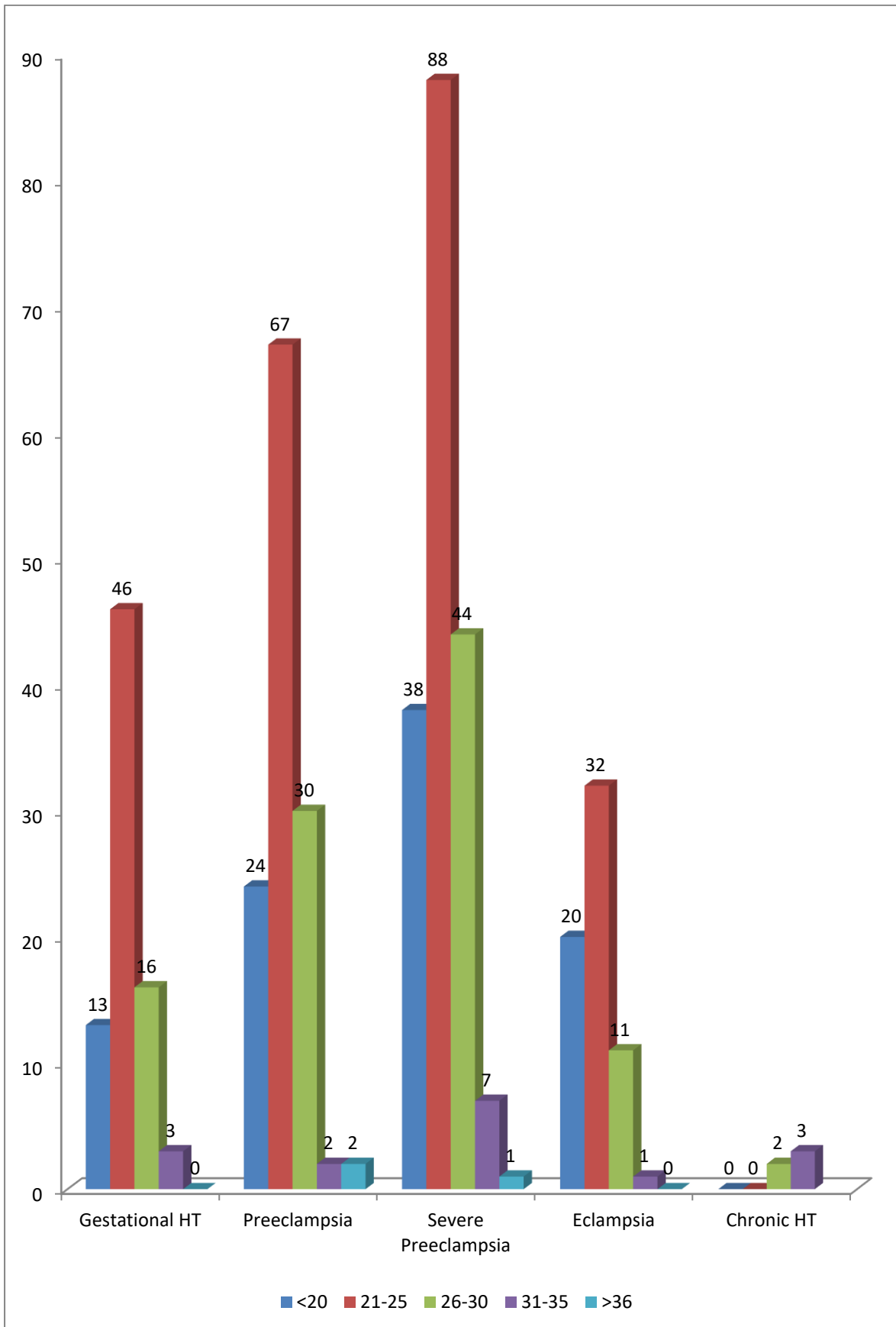


TABLE 4
GRAVIDA STATUS

Gravida	Number	Percentage
Primi	273	60.7%
G2	123	27.3%
G3 & above	54	12.0%
Total	450	100%

FIGURE 4
GRAVIDA STATUS

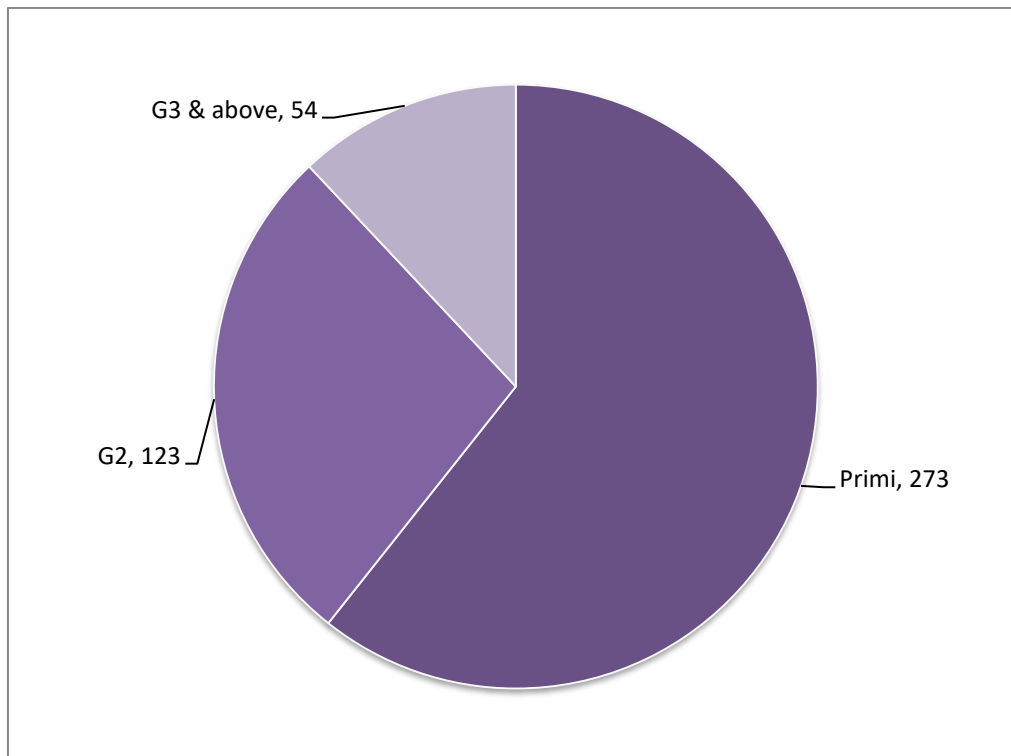


TABLE 5
GRAVIDA STATUS IN HYPERTENSIVE TYPES

Gravida	Gestational HT	Preeclampsia	Severe PE	Eclampsia	Chronic HT	Total
Primi	43 (55.1%)	71 (56.8%)	113 (63.5%)	45 (70.3%)	1 (20%)	273 (60.7%)
G2	25 (32.1%)	39 (31.2%)	42 (23.6%)	15 (23.4%)	2 (40%)	123 (27.3%)
G3 & above	10 (12.8%)	15 (12%)	23 (12.9%)	4 (6.3%)	2 (40%)	54 (12%)
Total	78	125	178	64	5	450

Chi-Square value: 21.32

P value: 0.02

Statistically significant

Out of 450 patients, primigravida were the most common (60.7%) in all forms of hypertension except in chronic hypertension. The rest are the multigravida (39.3%) of which the second gravida are most common.

In Gestational hypertension, the most common were primigravida (55.1%) followed by second gravida (32.1%). In Preeclampsia, the most common were primigravida (56.8%) followed by second gravida (31.2%). In Severe Preeclampsia also the most common are primigravida (63.5%). In Eclampsia, the most common were primigravida (70.3%) followed by second gravida (23.4%) and the least common were third gravida and more (6.3%). However in Chronic hypertension, second gravida and higher gravida are equally common (40% each) with primi being the least common (20%).

FIGURE 5

GRAVIDA STATUS IN HYPERTENSION TYPES

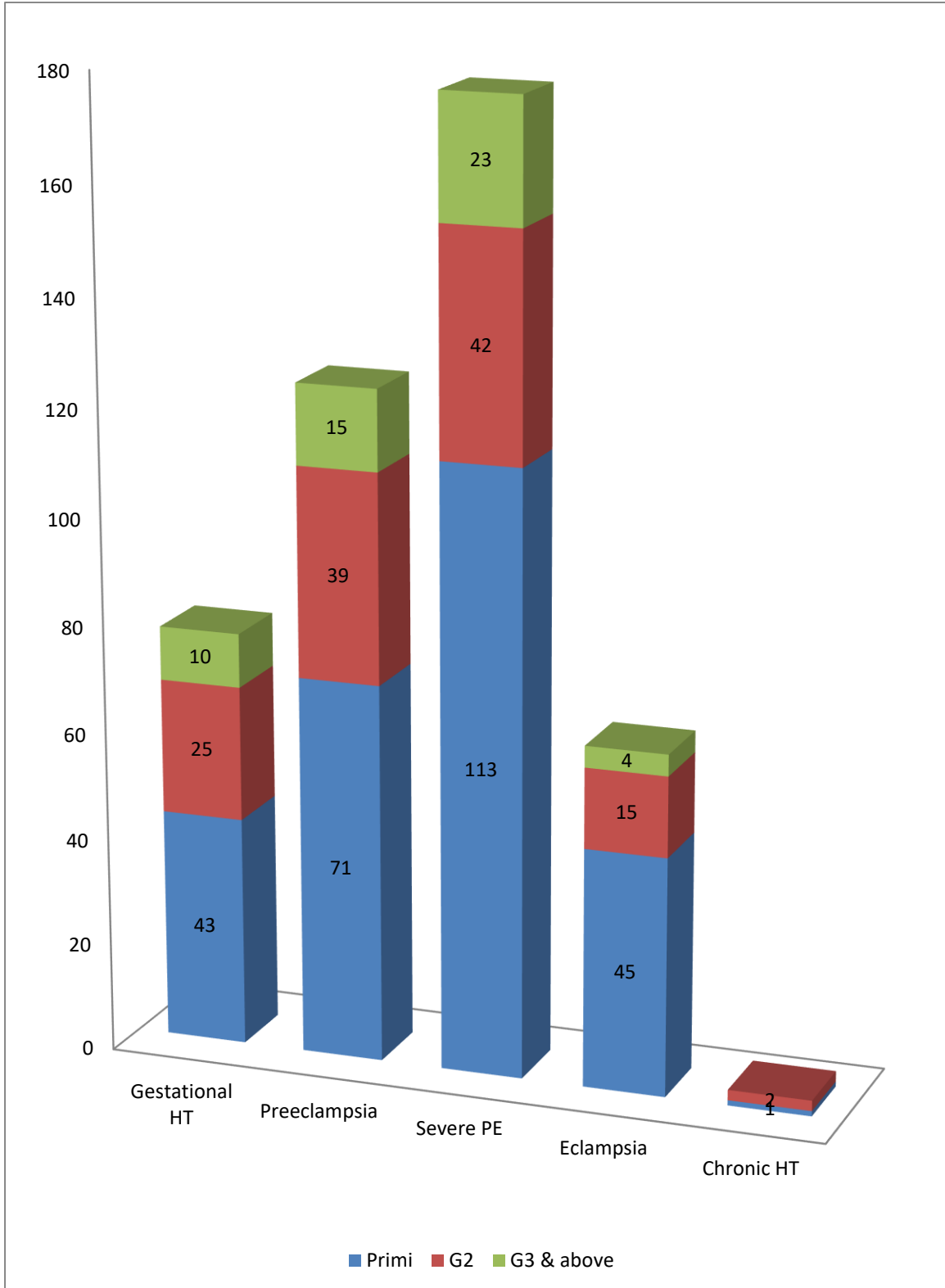


TABLE 6
SOCIOECONOMIC STATUS

Socioeconomic Status	Number	Percentage
I	Nil	Nil
II	Nil	Nil
III	Nil	Nil
IV	83	18.4
V	367	81.6
Total	450	100

Out of 450 patients studied, the majority belonged to the Socioeconomic Class V (81.6%) while the rest belonged to Class IV (18.4%). No patients belonging to Socioeconomic Class I, II and III took part in the study

FIGURE 6
SOCIOECONOMIC STATUS

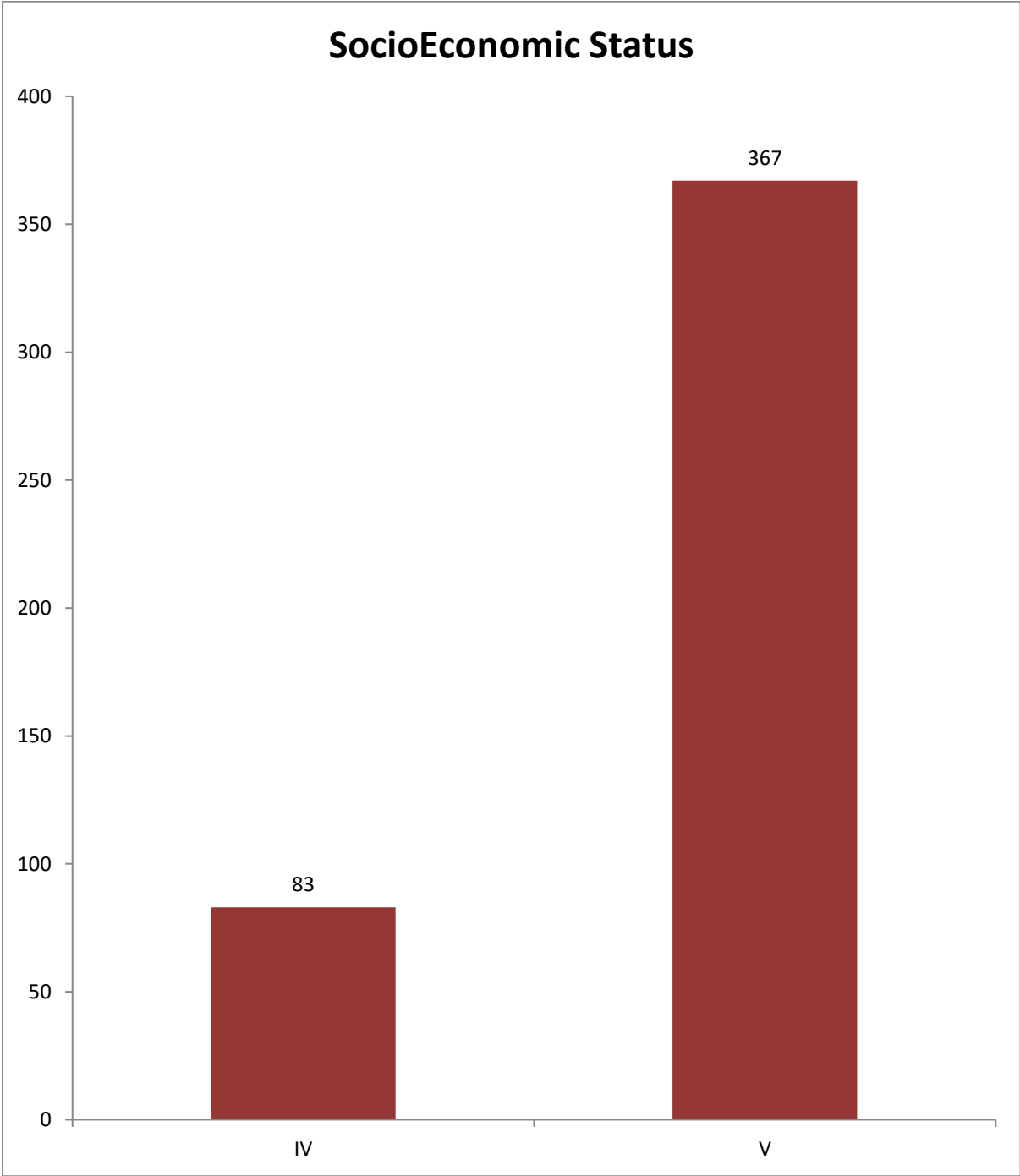


TABLE 7
GESTATIONAL AGE AT DELIVERY

Gestational Age	Gestational HT	Mild Preeclampsia	Severe PE	AP Eclampsia	PP & IP Eclampsia	Chronic HT	Total
<=28	0	0	21 (11.8%)	3 (5.8%)	0	0	24 (53.3%)
29-34	0	0	31 (17.4%)	17 (32.7%)	0	0	46 (10.2%)
35-37	6 (7.7%)	19 (15.2%)	84 (47.2%)	18 (34.6%)	6 (50%)	3 (60%)	91 (20.2%)
>37	72 (92.3%)	106 (84.8%)	42 (23.6%)	14 (26.9%)	6 (50%)	2 (40%)	289 (64.2%)
Total	78	125	178	52	12	5	450

In both Gestational hypertension and Mild Preeclampsia, majority were terminated at term (92.3% and 84.8% respectively); while the rest were terminated between 35-37 weeks.

However the figure is reversed in case of Severe Preeclampsia and Eclampsia where majority were terminated before term. In case of Severe Preeclampsia, majority were terminated between 35 and 37 weeks of gestation (47.2%). Next commonly women delivered at term (23.6%).

In case of Antepartum Eclampsia, majority of women delivered between 35 and 37 weeks (34.6%) while next commonly, women delivered between 29 and 34 weeks of gestation (32.7%). AP Eclamptic women delivering at term

were less common (26.9%). In case of intrapartum and postpartum eclampsia, delivery around 29-34 weeks and 35-37 weeks were equally common.

Of the chronic hypertensives, 3 delivered between 29 and 34 weeks and 2 delivered between 35 and 37 weeks.

FIGURE 7
GESTATIONAL AGE AT DELIVERY

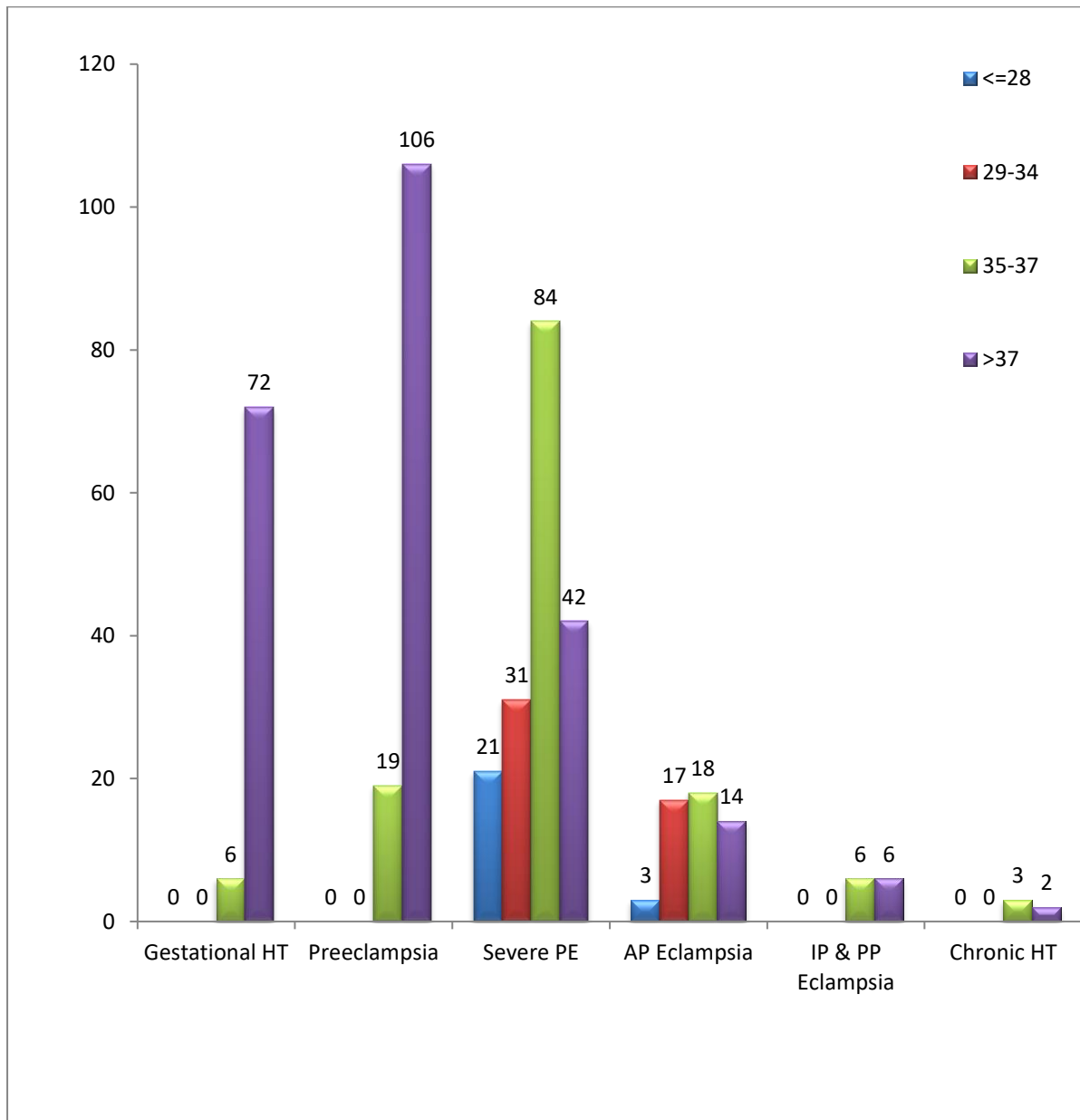


TABLE 8
MODE OF DELIVERY

Mode of Delivery	No of patients	Percentage
Labour Natural	177	39.3%
Assisted delivery	20	4.4%
Hysterotomy	4	0.9%
Spontaneous expulsion	24	5.4%
LSCS	225	50%
Total	450	100%

FIGURE 8
MODE OF DELIVERY

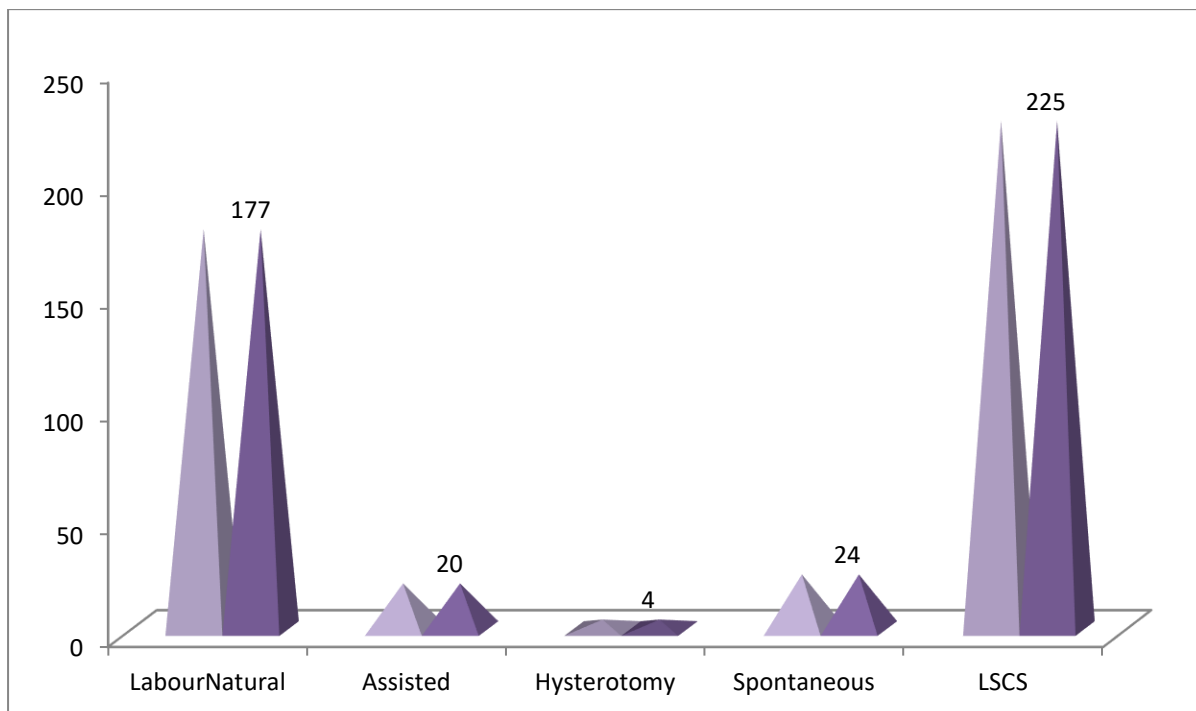


TABLE 9
MODE OF DELIVERY IN HYPERTENSION TYPES

Mode of Delivery	Gestational HT	Mild Preeclampsia	Severe PE	Eclampsia	Chronic HT	Total
Labour Natural	37 (47.4%)	61 (48.8%)	63 (35.3%)	15 (23.4%)	1 (20%)	177 (39.3%)
Assisted delivery	9 (11.5%)	11 (8.8%)	0	0	0	20 (4.4%)
Hysterotomy	0	0	0	4 (6.2%)	0	4 (0.9%)
Spontaneous expulsion	0	0	21 (11.8%)	3 (4.7%)	0	24 (5.4%)
LSCS	32 (41.1%)	53 (42.4%)	94 (52.9%)	42 (65.7%)	4 (80%)	225 (50%)
Total	78	125	178	64	5	450

Out of 450 patients, about half delivered by LSCS method (50%) while the next common method of delivery was by Labour natural (39.3%). There were only four cases of hysterotomy (0.9%) which was the least common.

In Gestational hypertension, majority delivered by Labour natural (47.4%) while LSCS was the next common option of delivery (41.1%). In Mild Preeclampsia also, Labour Natural remained the most common mode of delivery (48.8%) while the next in line was LSCS (42.4%)

In Severe Preeclampsia however, the common mode of delivery was LSCS (52.9%) which is followed by Labour Natural (35.3%). In Eclampsia too,

LSCS was the common method to deliver (65.7%) and Labour Natural the next common method (23.4%).

In Chronic Hypertensives, 80% delivered by LSCS & 20% by Labour Natural.

FIGURE 9
MODE OF DELIVERY IN HYPERTENSION TYPES

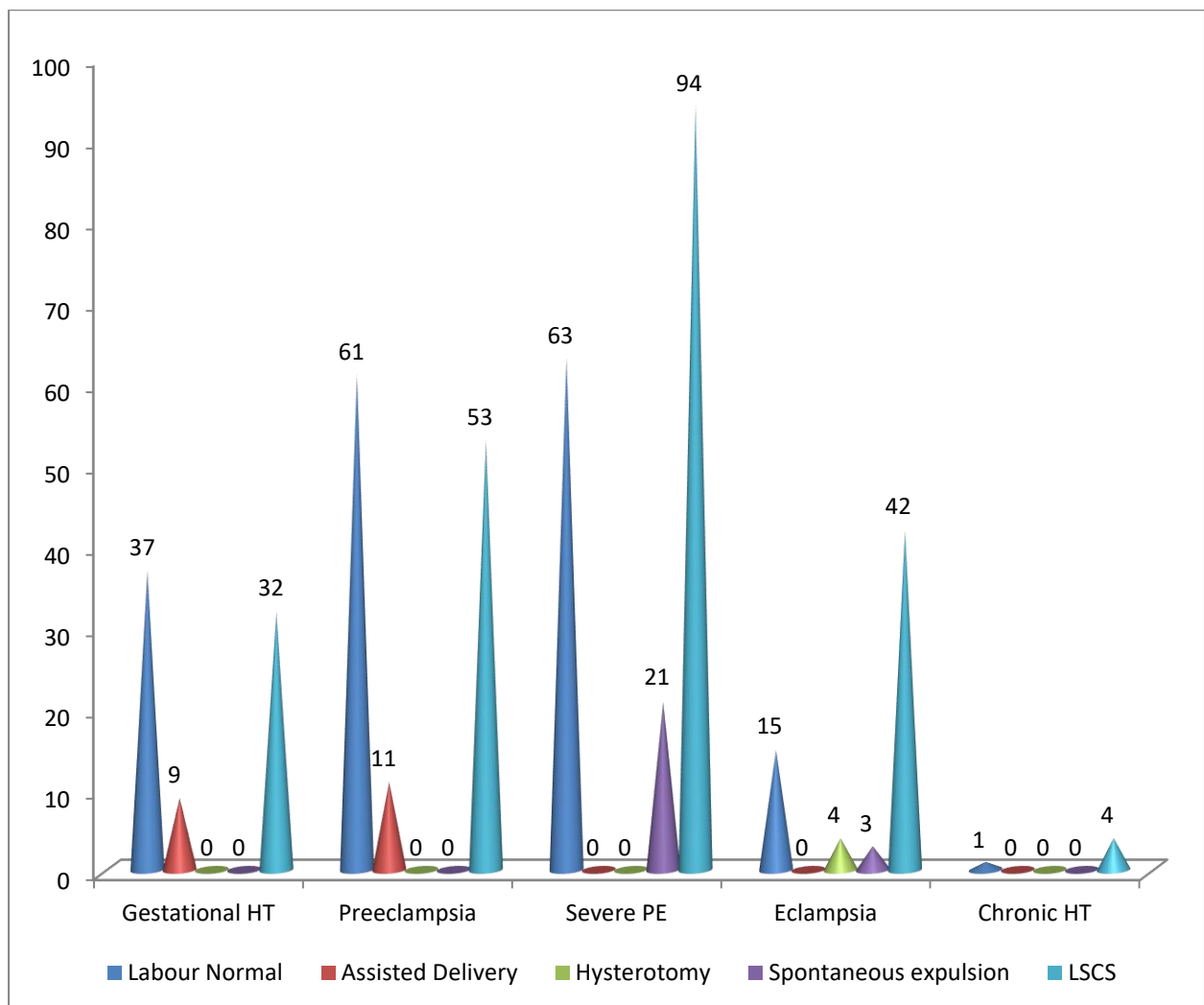


TABLE 10
MATERNAL COMPLICATIONS

Complication	Number	Percentage
Eclampsia	64	14.2%
Abruptio placenta	51	11.3%
HELLP	8	1.8%
PPH	27	6%
DIC	7	1.6%
ARF	6	1.3%
CVA	9	2%
Pulmonary edema	2	0.4%
Retinopathy	1	0.2%
Maternal Death	3	0.6%

FIGURE 10
MATERNAL COMPLICATIONS

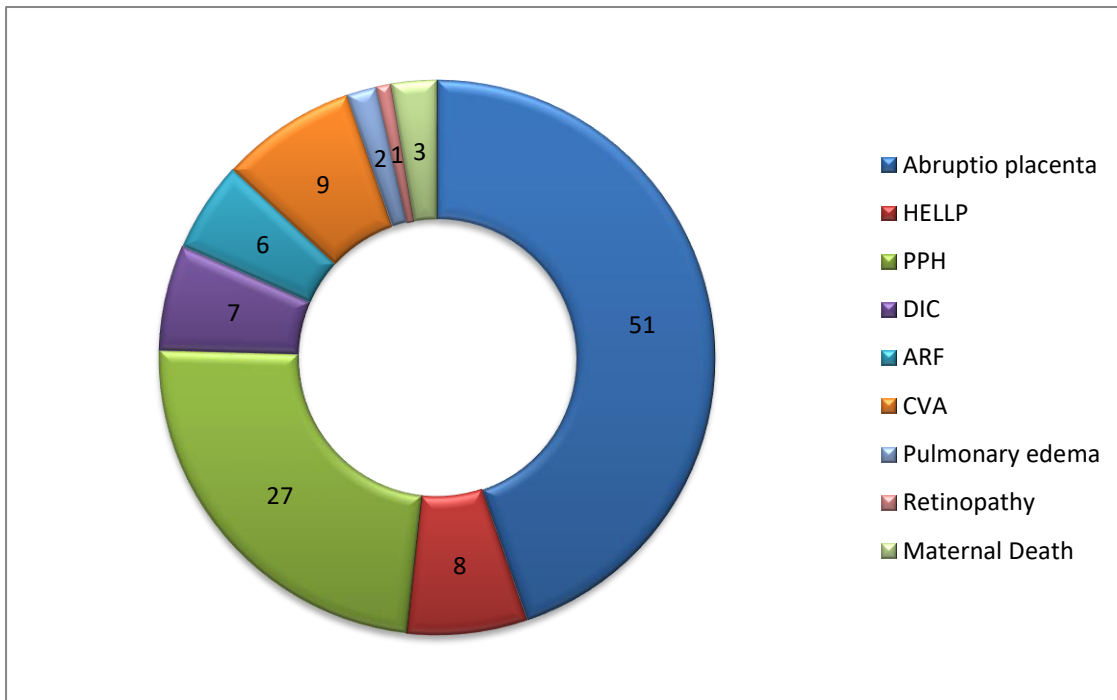


TABLE 11
MATERNAL COMPLICATIONS IN HYPERTENSION TYPES

Maternal Complications	Gestational HT	Mild Preeclampsia	Severe PE	Eclampsia	Chronic HT	Total
Maternal Death	0	0	1	2	0	3 (0.6%)
ARF	0	0	2	4	0	6 (1.3%)
Abruptio placenta	0	9	37	5	0	51 (11.3%)
DIC	0	0	6	1	0	7 (1.6%)
CVA	0	0	0	9	0	9 (2%)
HELLP	0	0	6	2	0	8 (1.8%)
PPH	1	6	19	1	0	27 (6%)
Pulmonary edema	0	0	1	1	0	2 (0.4%)
Retinopathy	0	0	1	0	0	1 (0.2%)
Total	1	15	73	25	0	114

Out of the total complications, majority occurred in patients with Severe Preeclampsia and Eclampsia. About 353 patients sustained an uneventful pregnancy without any complications (78.4%).

Out of 450 patients, 51 developed Abruptio placenta (11.3%) and 9 developed Cerebrovascular accidents (2%) which includes cases with CT features suggestive of PRES. 8 mothers developed HELLP syndrome (1.8%) and 6 women developed Acute Renal Failure (1.3%). PPH occurred in 27 women (6%).

FIGURE 11
MATERNAL COMPLICATIONS IN HYPERTENSION TYPES

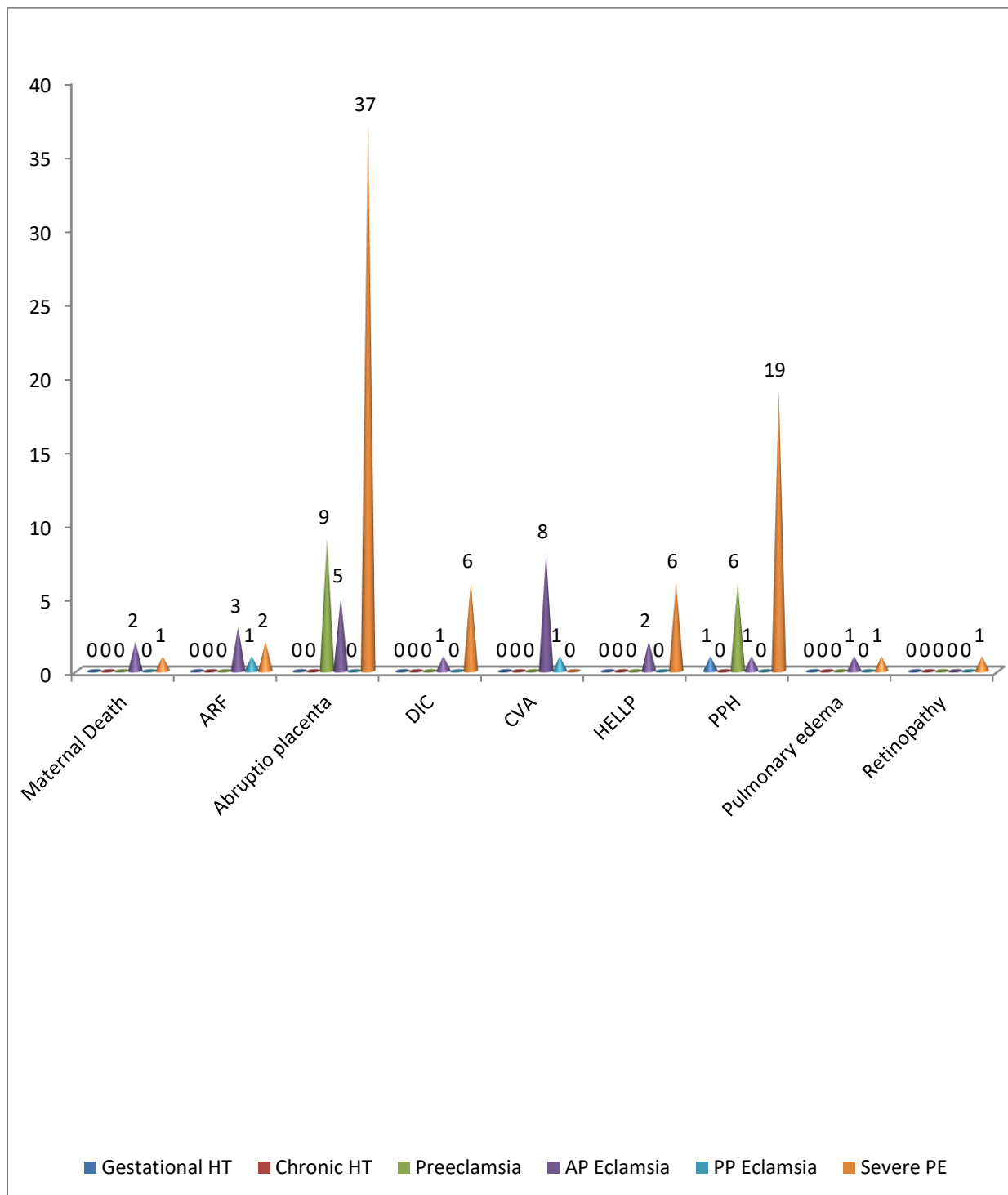


TABLE 12**POSTPARTUM RETURN OF BLOOD PRESSURE TO BASELINE**

HYPERTENSION TYPE	0-2 DAYS	3-13 DAYS	2 WEEKS AND ABOVE
GESTATIONAL HT	75	3	-
PREECLAMPSIA	102	14	7
SEVERE PREECLAMPSIA	8	92	57
ECLAMPSIA	-	29	27
TOTAL	185	138	91
PERCENTAGE	41.1	20.7	20.2

Out of 450 women studied, 33 women did not achieve postpartum return of Blood Pressure value to baseline within the period followed up and were discharged with antihypertensives. These 33 women also include the 5 chronic hypertensives. Excluding the 3 maternal deaths, the rest 414 women had their pressure values returned to baseline.

Majority of these women became normotensive within the first two days of delivery (41.1%). The next major group had normal pressure values within 13 days (20.7%) closely followed by those who took 2 weeks and more to return to the normal blood pressure range (20.2%)

FIGURE 12
POSTPARTUM RETURN OF BLOOD PRESSURE TO BASELINE

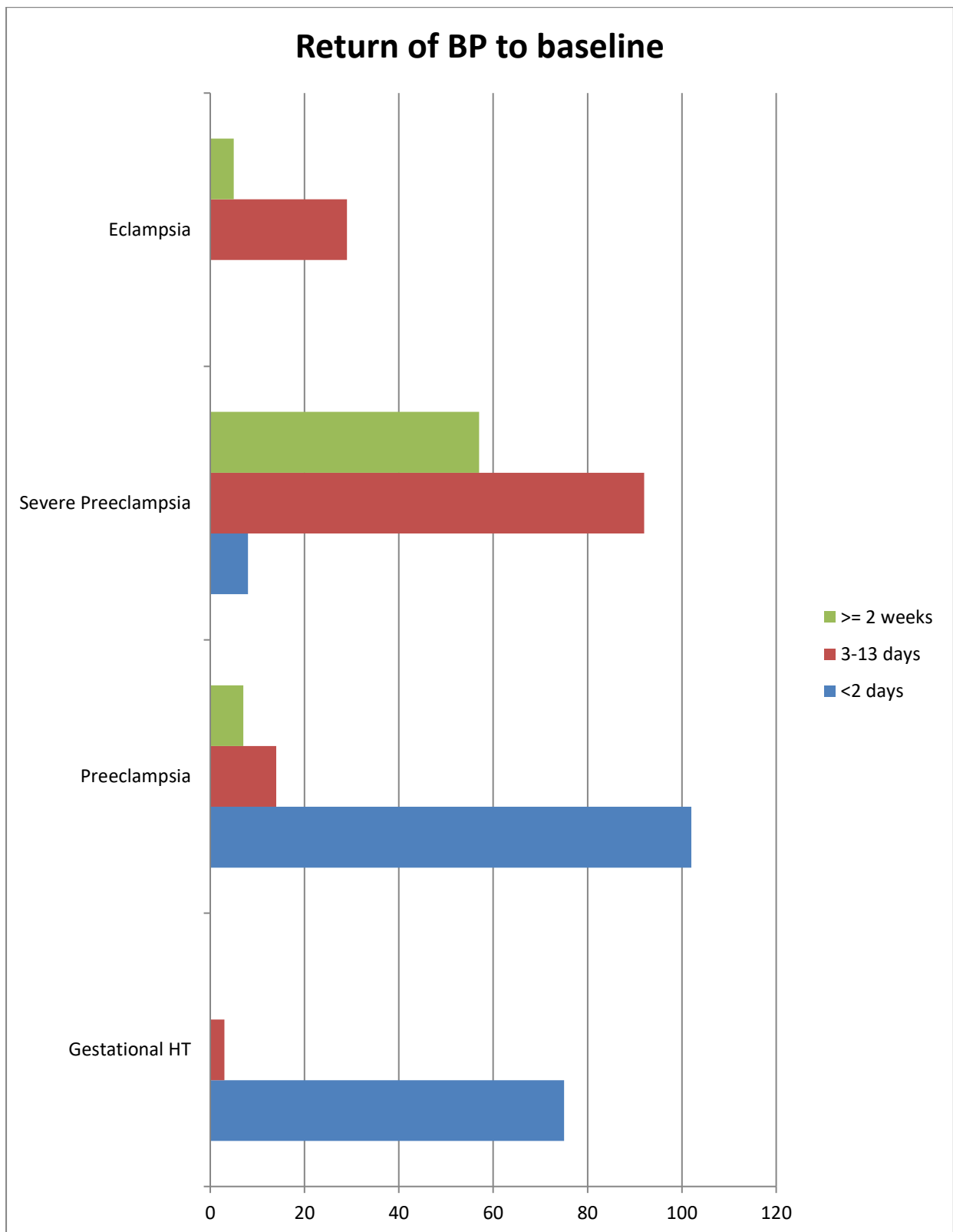


TABLE 13
FETAL OUTCOME
BIRTHWEIGHT OF THE BABIES

Birth Weight (kgs)	No of babies	Percentage
<2	123	27.3%
2.1-2.5	137	30.5%
2.6-3	141	31.4%
>3.1	49	10.8%
Total	450	100%

FIGURE 13
BIRTHWEIGHT OF BABIES

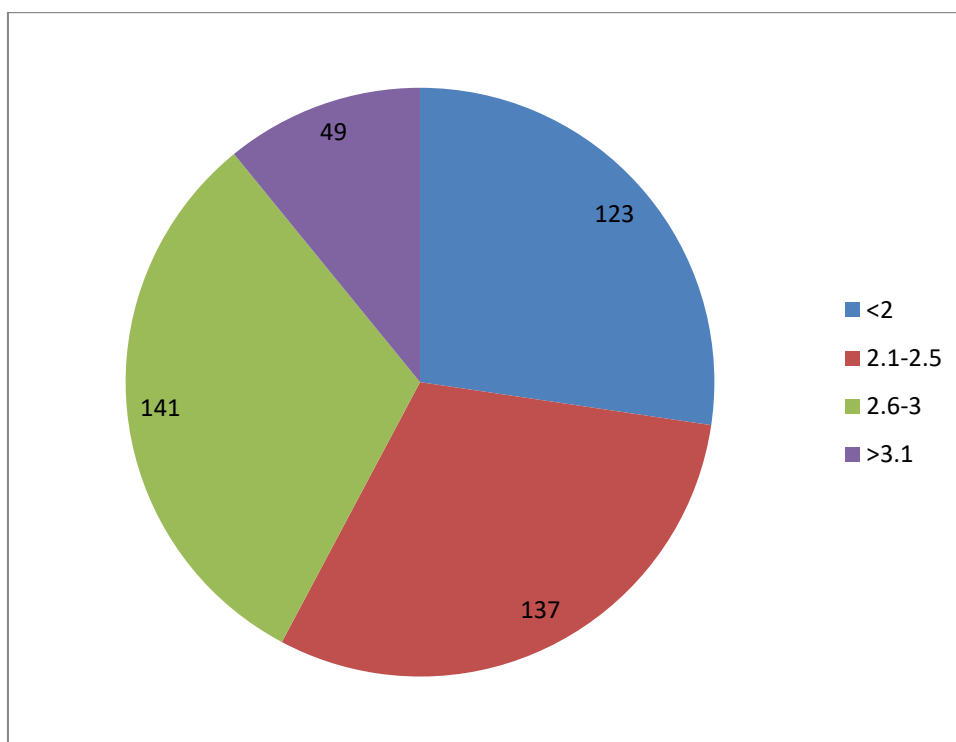


TABLE 14**BIRTHWEIGHT ACCORDING TO TYPES OF HYPERTENSION**

Birth Weight (kgs)	Gestational HT	Preeclampsia	Severe PE	Eclampsia	Chronic HT	Total
<2	0	6 (4.8%)	78 (43.8%)	39 (60.9%)	0	123 (27.4%)
2.1-2.5	17 (21.8%)	45 (36%)	59 (33.2%)	16 (25%)	0	137 (30.4%)
2.6-3	41 (52.6%)	56 (44.8%)	32 (18%)	9 (14.1%)	3 (60%)	141 (31.3%)
>3.1	20 (25.6%)	18 (14.4%)	9 (5%)	0	2 (40%)	49 (10.9%)
Total	78	125	178	64	5	450

Chi-Square value: 166.2

P value: 0.001

Statistically significant

Most of the babies born to hypertensive mothers weighed between 2.6 to 3 kgs (31.3%). This was closely followed by babies weighing between 2.1 to 2.5 kgs (30.4%) which was almost similar to the previous category. Only 10.9% of babies weighed more than 3 kgs while 27.4% of babies were below 2 kgs.

While in Gestational Hypertension and Mild Preeclampsia, the majority of the babies weighed between 2.6 to 3 kgs, in Severe Preeclampsia and Eclampsia group, the majority of the babies weighed below 2 kg.

FIGURE 14
BIRTHWEIGHT OF BABIES ACCORDING TO TYPES OF
HYPERTENSION

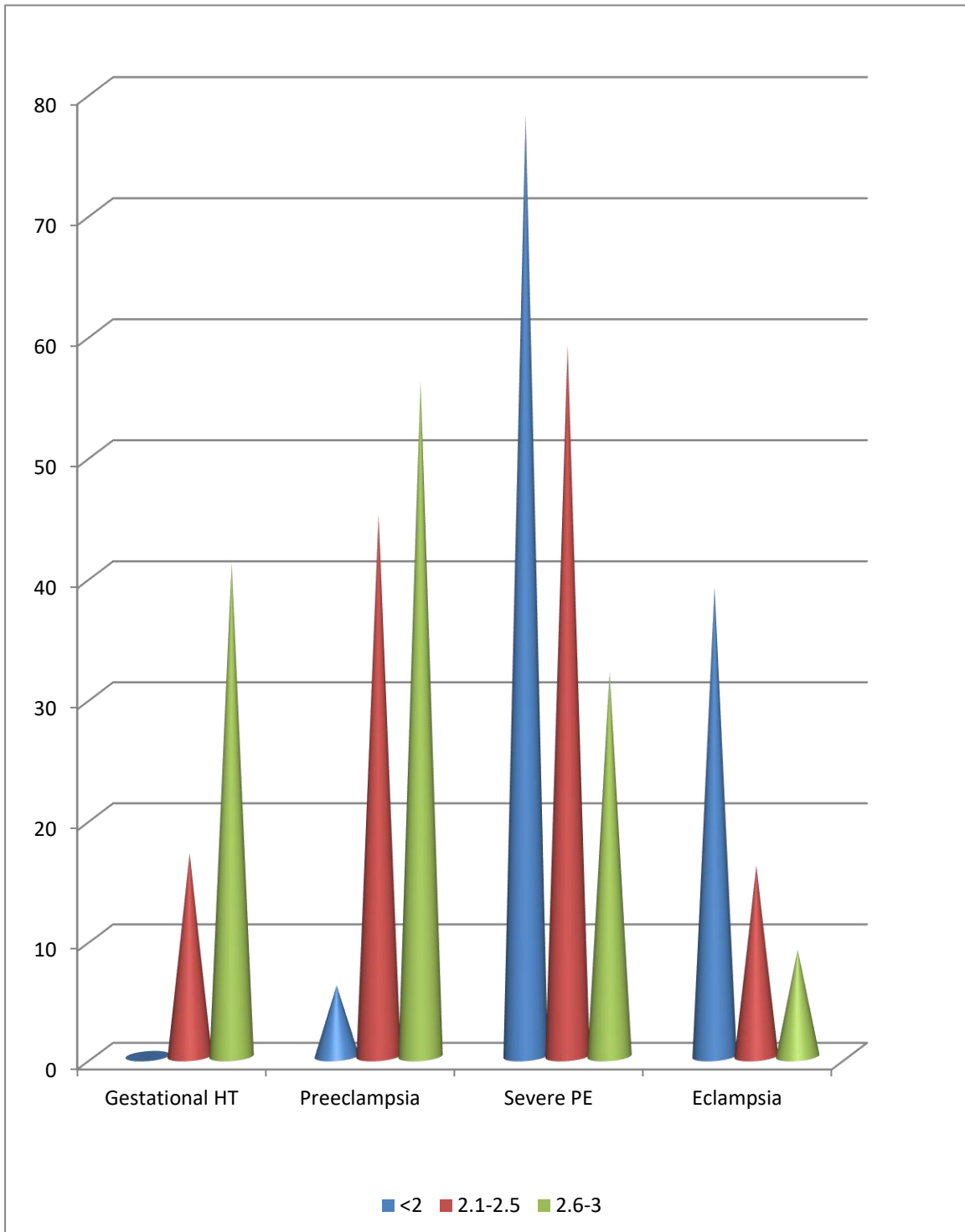


TABLE 15
FETAL COMPLICATIONS

Fetal Complications	Number of babies	Percentage
Birth asphyxia	17	3.8%
IUGR	72	16%
Neonatal Death	16	3.5%
Pre maturity	139	30.9%
Still Birth	2	0.4%
IUD	25	5.5%

FIGURE 15
FETAL COMPLICATIONS

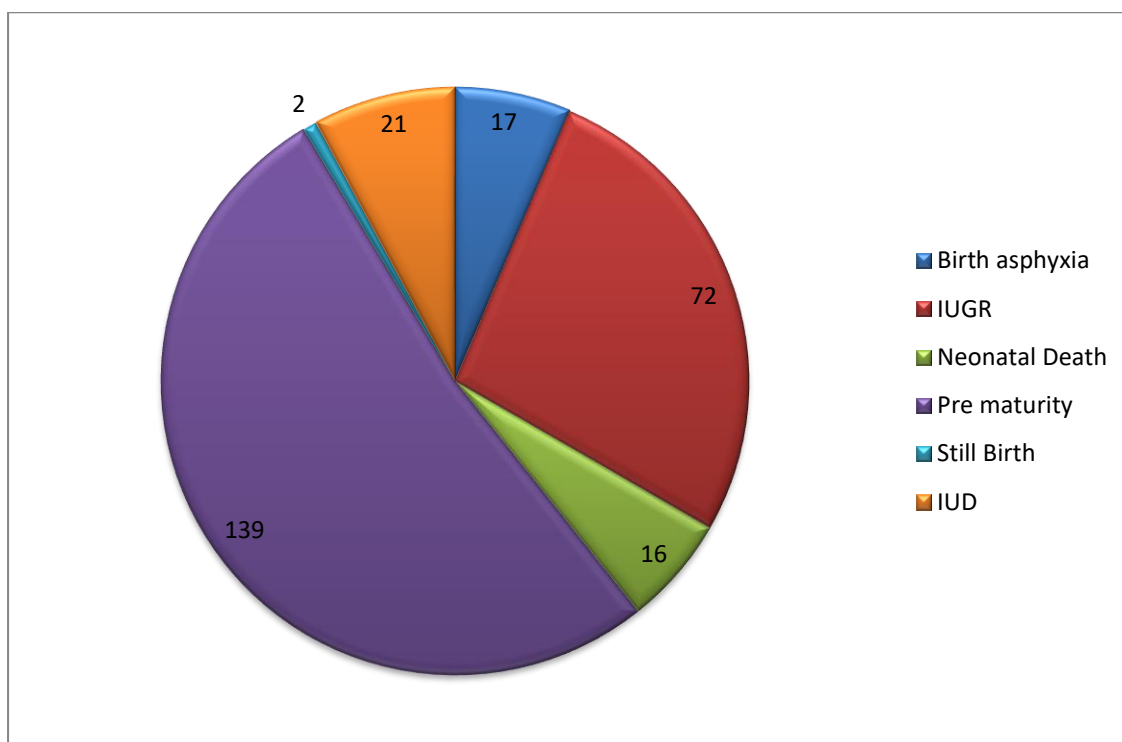


TABLE 16
FETAL COMPLICATIONS IN HYPERTENSION TYPES

Fetal Complications	Gestational HT	Preeclampsia	Severe PE	Eclampsia	Chronic HT	Total
Neonatal Death	0	2	9	5	0	16 (3.5%)
Stillbirth	0	0	2	0	0	2 (0.4%)
Prematurity	6	19	93	20	1	139 (30.9%)
IUD	0	0	21	4	0	25 (5.5%)
IUGR	4	22	37	9	0	72 (16%)
Birth asphyxia	0	3	9	5	0	17 (3.8%)
Total	10	46	171	43	1	271

Out of all the babies born to mothers with hypertension, 231 babies were born without any complication (51.3%). 219 babies developed some form of complication of which few babies had overlapping (more than one) complication.

In total, there were 25 Intrauterine Deaths (5.5%) of which 21 IUDs occurred in the Severe Preeclampsia group and 4 IUDs occurred in Eclamptic women. There were only 2 stillbirths in the study group (0.4%) and both of these occurred in the Severe Preeclampsia group.

Totally there were 16 Early Neonatal deaths (3.5%) of which the majority were in the Severe Preeclampsia group (9 deaths) and the next common in the Eclampsia group (5 deaths).

The most common complication however is Prematurity with an incidence of 30.9% and the next common complication was IUGR with an incidence of 16%. Birth Asphyxia occurred in 3.8% of babies.

FIGURE 16
FETAL COMPLICATIONS

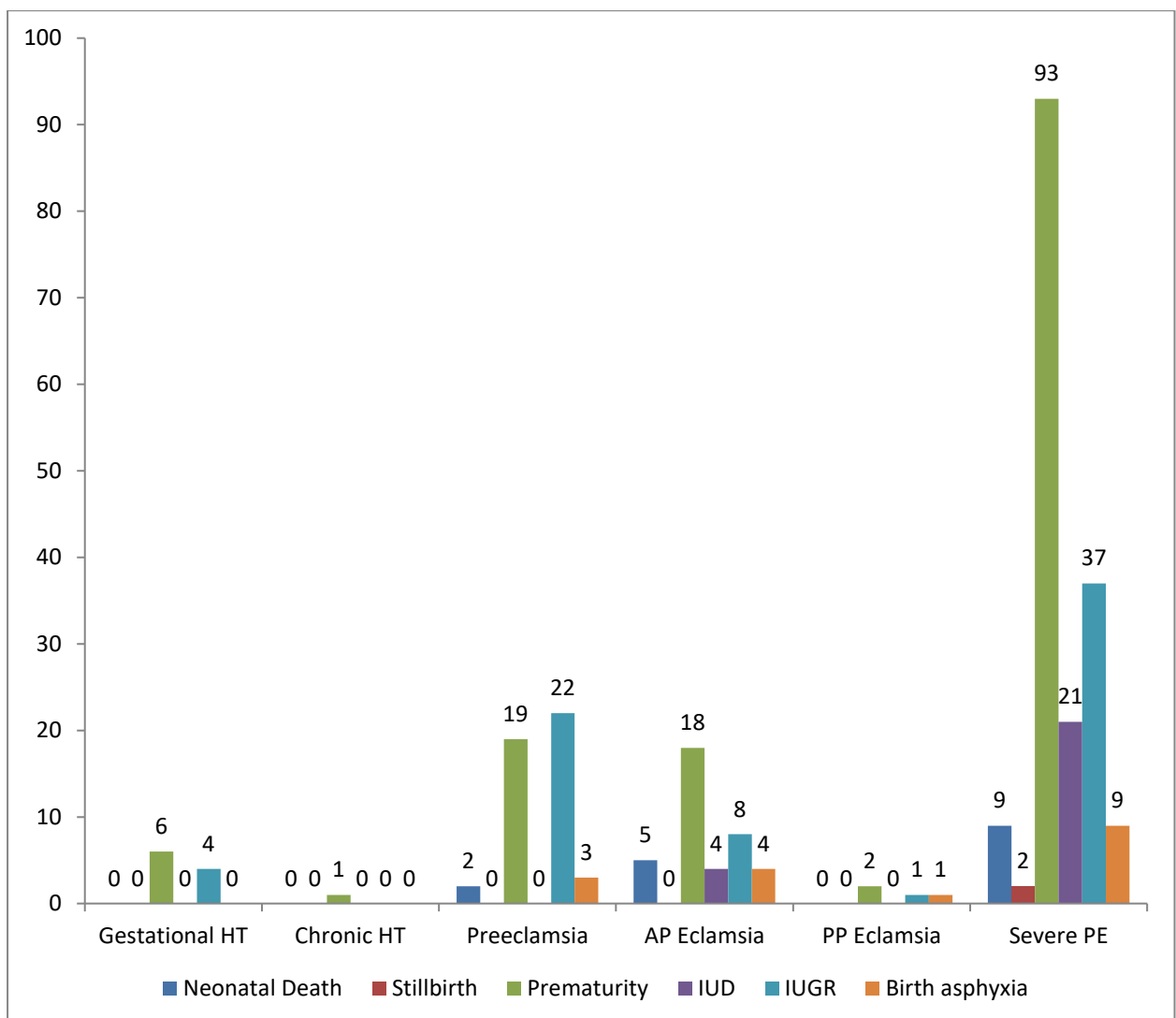


TABLE 17
FETAL MORTALITY IN HYPERTENSION TYPES

Fetal outcome	Gestational HT	Preeclampsia	Severe PE	Eclampsia	Chronic HT	Total
Perinatal Deaths	0	2 (1.6%)	11 (6.1%)	5 (7.8%)	0	18 (4%)
IUD	0	0	21 (11.8%)	4 (6.3%)	0	25 (5.5%)
Live births	78 (100%)	123 (98.4%)	146 (82.1%)	55 (85.9%)	5 (100%)	407 (90.5%)
Total	78	125	178	64	5	450

Chi-Square value: 40.52

P value: 0.001

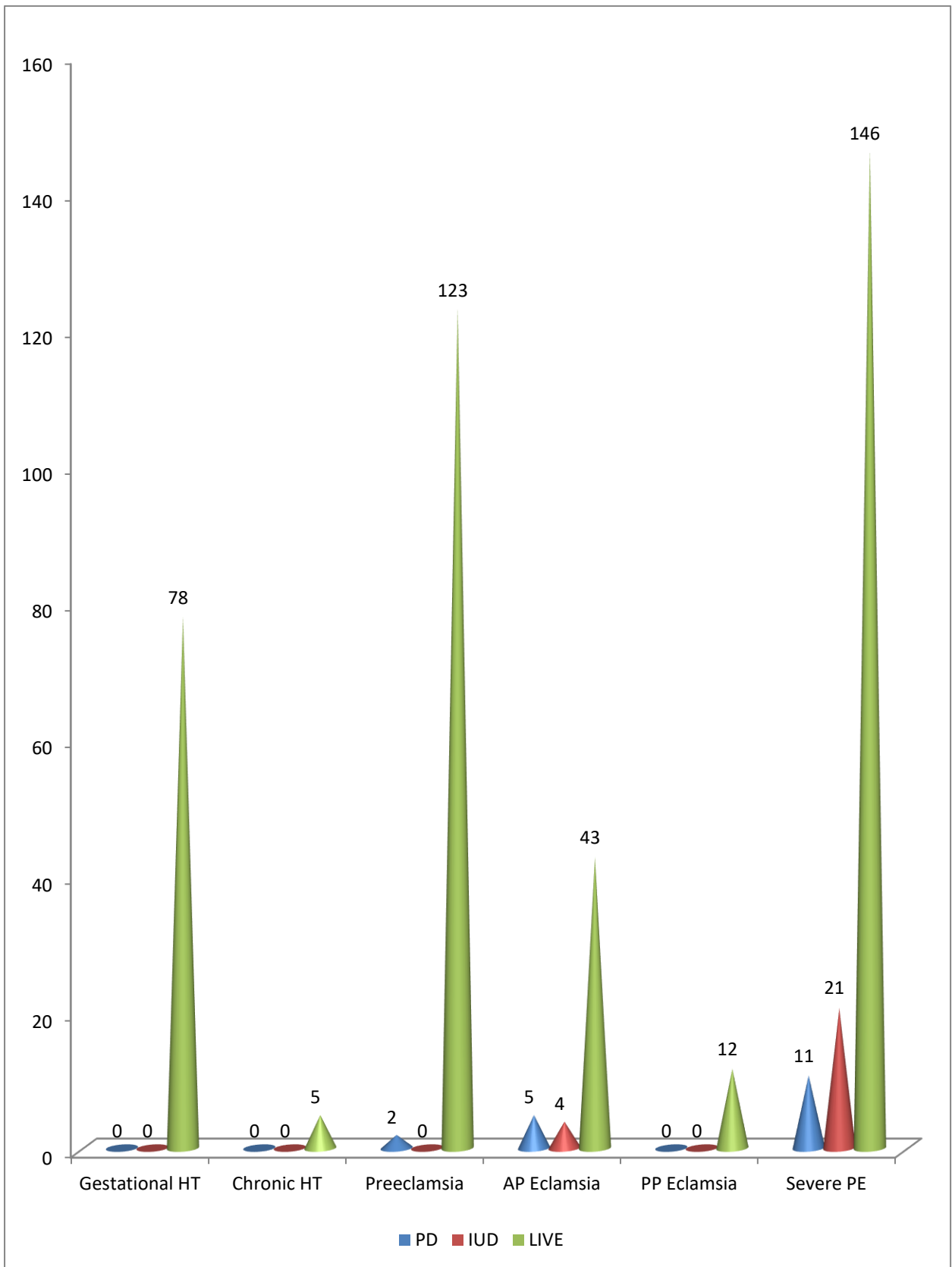
Statistically significant

Of all the results of hypertensive pregnancies, 90.5% were successful resulting in live births. IUDs constituted about 5.5% while perinatal deaths made up 4% of all hypertensive pregnancies.

Of all IUDs, the majority occurred in Severe Preeclampsia (21 IUDs) while 4 deaths occurred in Eclampsia. Majority of perinatal deaths also occurred in Severe Preeclampsia (11 deaths) with Eclampsia being the next common (5 deaths).

There was no mortality in Gestational hypertension, Mild Preeclampsia and Chronic Hypertension groups.

FIGURE 17
FETAL MORTALITY IN HYPERTENSION TYPES



DISCUSSION

450 antenatal women with hypertensive disorder of pregnancy were studied after taking into consideration the inclusion and exclusion criteria for a period of one year. These women were studied for their demographic characters such as age, parity and socioeconomic class. Gestational age at delivery, mode of termination, maternal complications and fetal outcomes were also studied.

INCIDENCE AND DISTRIBUTION OF HYPERTENSION IN PREGNANCIES

The exact incidence of hypertensive disorders complicating pregnancy could not be determined by the study since the exclusion criteria of the study ruled out multiple pregnancies, pregnancies with other comorbidities such as diabetes mellitus, renal disorders and so on.

Of the 450 women in our study, the most common hypertension type was Severe Preeclampsia with an incidence of 39.6% while the next common was Mild Preeclampsia with an incidence of 27.8%. Similar findings were reported by Seyom et al⁴⁵ in whose study Severe Preeclampsia was the most common with an incidence of 35.5%. However this was followed by Eclampsia (19%) and then by Mild Preeclampsia (14.9%). The proportion of Eclampsia in our study is 14.2%.

The proportion of Chronic Hypertension was noted to be around 4% in the Indian study by Yadav and Saxena⁴⁶. However in our study, chronic

hypertension constituted only 1.1% of all cases. This discrepancy might be due to the fact that the presence of other comorbidities has been already ruled out and hence the figure of women with pure hypertension alone dips.

AGE DISTRIBUTION

The maximum incidence of hypertension during pregnancy in our study was noted in the age group between 21 to 25 years (51.8%) which was followed by the age group 26-30 years (22.9%). This only goes to prove the point that child bearing age is earlier in our developing country when compared with developed nations. Almost analogous findings were reported by Seyom et al⁴⁵ with 82.6% of hypertensive mothers falling within the broad age group of 18-34 years. Also, Aabidha et al reported an incidence of 46.23% in the age group 21-25 yrs and 23.65% in the age group 26-30 yrs⁴⁷.

PARITY

It has been traditionally said that Preeclampsia is a disease of primiparity. Conforming to the same, primigravida constituted 60.7% of all hypertensive mothers in our study. The next majority were the second gravida making up 27.3% while higher parity make up the rest (12%). These results are similar to those noted by Aabidha et al in a study done in a secondary care center in TamilNadu where primigravida constituted 61.2% and second gravida made up 21.5%⁴⁷. Also the study by Bhattacharya⁴⁹ showed that primigravida constituted 65.6% of total cases.

SOCIOECONOMIC STATUS

Our study shows 81.6% of women belonging to Socioeconomic class V and the rest (18.4%) belonging to Socioeconomic class V. This data goes to prove the type of population the tertiary care center caters to.

GESTATIONAL AGE AT DELIVERY

In both Gestational hypertension and mild preeclampsia, majority of mothers delivered at term (>37 weeks). But in both severe preeclampsia and AP Eclampsia, most women delivered as late preterm (between 35 and 37 weeks).

MODE OF DELIVERY

In our study, LSCS was the common mode of termination of the pregnancy (in 50% of cases) closely followed by Labour Natural (in 39.3%). Assisted delivery in the form of forceps, vacuum etc was resorted to in 4.4% of cases. This correlates well with the Indian study conducted by Meshram et al⁴⁸ where abdominal route (including both LSCS and hysterotomy) was the common method of terminating hypertensive pregnancies with a slightly higher incidence than in our study (65.6%). Vaginal delivery constituted about 34.4% in their study while this was slightly higher (39.3%) in our study.

BP RETURN TO BASELINE

Majority of patients have become normotensive within 2 days postpartum (41.1%) while 20.7% had their BP returned to baseline within 3-13 days and 20.2% became normotensive after 2 weeks.

MATERNAL COMPLICATIONS

Maternal complications occurred in 21.6% of cases in our study as opposed to 18% in the study by Seyom et al⁴⁵. The most common maternal complication noted in our study was that of Placental abruption (11.3%) followed by PPH (6%). This is more or less alike to the South Indian study conducted by Aabidha et al⁴⁷ where APH constituted 13.97% and PPH constituted 10.75% of pregnancies. Study by Seyom et al⁴⁵ reported a 7.4% incidence of PPH in pregnancy induced hypertension. Excepting preterm labour, Abruptio Placenta was the most common complication (19.04%) in the study by Bangal et al⁵⁰ also.

The incidence of HELLP syndrome in our study was 1.8% as opposed to the incidence of 2.2% in the study by Yucesoy et al⁵¹. The incidence of Retinopathy was 0.2% as opposed to incidence of 1.4% in the study by Nankali et al⁵².

Maternal mortality was noted in 0.6% in our study as opposed to 1.2% in the study by Ahmed et al⁵³.

FETAL COMPLICATIONS

Prematurity is the most common complication occurring in 30.9% of deliveries. Similar findings were reported by Bangal et al⁵⁰ where incidence of prematurity were 17.99%, 47.62% and 52.63% in Mild Preeclampsia, Severe Preeclampsia and Eclampsia respectively. The next common complication was IUGR (16%) which is also in accordance with the study by Bangal et al⁵⁰

wherein the incidence were 7.69%, 26.19% and 31.58% respectively in the above categories.

Our study revealed 42.2%, 30.4% and 27.4% of babies with birth weights >2.5 kg, between 2 and 2.5 kg and <2 kg respectively. Similar findings were reported by Ahmed et al⁵³ where the percentages in the respective classes were 45.2%, 26% and 28.8% respectively.

CONCLUSION

From the above demographic and other data presented, it becomes evident that Pregnancy Induced Hypertension is one of the major causes of admissions to antenatal wards apart from being a significant contribution towards maternal morbidity, mortality and complications. Complications can affect all maternal systems including Central nervous system, renal system and hematological systems. Apart from these, the fetus also faces the brunt of insult. Fetal complications include prematurity, low birth weight, still births, intrauterine deaths and intrauterine growth restrictions. However such vast complications can be prevented by simple blood pressure monitoring and proper antenatal mother followup. Regular antenatal monitoring along with intrapartum fetal surveillance can help us in deciding about the mode of termination of the pregnancy. In addition to the timely diagnosis of the various complications, prompt referral to higher centers is also essential for expert opinion and other specialty opinion. Cases which were promptly followed up and managed on a regular basis were found to be associated with lower rates of morbidity.

LIMITATIONS

The major limitation of the study is its exclusion criteria. Since the exclusion criteria excluded other co-morbidities such as diabetes mellitus, renal disorders and so on, the exact incidence and figures of Pregnancy Induced Hypertension could not be determined. However since the other high risk conditions have been ruled out, the advantage is that the study showed the potentiality of complications arising out of pure hypertensive etiology alone.

The other limitation of the study is that of the few selected patients who were managed on an outpatient basis, a small number were lost in follow up reducing an insignificant number of patients in the process.

BIBLIOGRAPHY

1. James PR, Nelson-Piercy C. Management of Hypertension before, during and after pregnancy. *Heart*. 2004;90:1499-1504.
2. Khan KS et al. WHO analysis of causes of maternal death: a systematic review. *Lancet*. 2006 Apr 1;367(9516):1066-74
3. Manjusha Sajith et al. Incidence of pregnancy induced hypertension and prescription pattern of antihypertensive drugs in pregnancy. *IJPSR* 2014 Apr Vol5 No4
4. NHBPEP (National High Blood Pressure Education Program) Working Group on High Blood Pressure. Report of the National High Blood Pressure Education Program Working Group in High Blood Pressure in Pregnancy. *Am J Obstet Gynecol*. 2000;183:S1-22
5. Levine RJ, Ewell MG, Hauth JC et al: should the definition of preeclampsia include a rise in diastolic Bp > 15 mm Hg to a level <90mm Hg in association and preeclampsia? *Am J obstet Gynb* 183:787,2000
6. Berg CJ et al. Pregnancy related mortality in the United States, 1991-1997. *Obstet Gynecol*. 2003 Feb;101(2)..289-96
7. Cantwell R et al. Saving Mothers' life: Reviewing maternal death to make motherhood safer: 2006-2008. The Eighth report of the

- Confidential enquiries into maternal death in the United Kingdom. *Brit J Obstet Gynecol.* 2011;118, Suppl 1:1-203
8. Lamminpa R et al. Preeclampsia complicated by advanced maternal age: A registry-based study on primiparous women in Finland. *BMC Pregnancy Childbirth* 2012, Jan 11; 12(1):47
 9. Robillard P.Y & Hulsey, T.C. (1996) Association of PIH, Preeclampsia and Eclampsia with duration of sexual cohabitation before conception, *Lancet* 347, 619.
 10. MacGillivray I. (1959) Some observations on the incidence of preeclampsia *Journal of obstet Gynaecol of British. Common wealth* 65, 536 – 539.
 11. Chesley LC. Diagnosis of Preeclampsia. *Obstet Gynecol* 1985: 65: 423–425
 12. Cincotta RB, Brennecke SP, Family History of Preeclampsia in Primigravida. *Int J Gynaecol Obstet* 1998: 60: 23 – 27.
 13. *Progress in Obstetrics and Gynecology- John Studd 18th edition*
 14. Bouthoornsh et al. Ethnic differences in blood pressure and hypertensive complication during pregnancy: The generation R study. *Hypertension* 2012 Jul;60(1):198-205
 15. *Deswriet Textbook of Obstetrics 2004*
 16. Sibai et al. Hypertensive disorders in twin vs singleton gestations. *Am J Obstet Gynecol* 2000;182:938

17. Shigeru Saito et al. A review of the mechanism of poor placentation in early onset preeclampsia: The role of autophagy in trophoblast invasion and vascular remodeling. *Journal of Reproductive Immunology*. Mar 2014; Vol 101-102; 80-88
18. Jeffcoate and Scott J.S. 1959. Some observations of placental Factor in pregnancy toxemia – *American Journal of Obstetrics and Gynecology* 72: 475 – 489.
19. Majumdar S et al. A study of placenta in normal and hypertensive pregnancies. *J Anat Soc India* 2005;54(2): 1-9
20. Magee LA et al. Hypertension Guideline Committee; Strategic Training Initiative in Research in the Reproductive Health Sciences (STIRRHS) Scholars. Diagnosis, evaluation and management of the hypertensive disorders of pregnancy. *J Obstet Gynecol Can* 2008 Mar; 30(3 Suppl): S1-48
21. Chesley LC. Diagnosis of Preeclampsia. *Obstet Gynecol* 1985; 65: 423–425
22. Gant N.F. Chand S. Worley R.J., Macdonald P.C (1974) *American Journal of Obstetrics and Gynecology* 120, 1.
23. Baker PN et al. The use of hand-grip test for predicting pregnancy-induced hypertension. *Eur J Obstet Gynecol Reprod Biol*. 1994 Sep;56(3):169-72

24. Chhabra S, Gandhi D, Prediction of Pregnancy induced hypertension/ Preeclampsia by detecting Microalbuminuria Journal of Obstetrics and Gynecology of India 2002. Jan – Feb; 52(1): 56 – 60.
25. Maikarnaz P, Holley JL, et al 1989; Gestational Hypercalciuria Causes Pathological Urine Calcium Oxalate Supersaturations. Kidney Int 36:108 –113.
26. Saudan et al. Urinary calcium/creatinine ratio as a predictor of Preeclampsia. AJH 1998;11:839-843
27. Ballegeer V et al 1989. Predictive Value of Increase Plasma Levels of Fibronectin in Gestational Hypertension AmJ Obstet Gynaecol 161:432 – 436.
28. Millar JG, Kyle PM et al Br J Obstet Gyneacol 1996 Oct; 103(10): 981 – 7
29. Begam S et al 1996. Urinary levels of metabolites of nitric oxide in normal pregnancy of preeclampsia J Obstet Gynaecol 22(6):552-559.
30. Oney T Et al. Relationship between Serum Prolactin Concentration, Vascular Angiotensin Sensitivity and Arterial blood pressure during third trimester pregnancy. Arch Gynecol Obstet:1988;243(2);83 – 90.
31. Kleinrouweler CE et al. The added value of second trimester uterine artery Doppler to patient characteristics in the identification of

nulliparous women at increased risk for Preeclampsia: An individual patient data Metaanalysis. *Ultrasound Obstet Gynecol* 2013 Feb 15

32. Kuc S et al. Evaluation of 7 serum biomarkers and uterine artery Doppler ultrasound for first trimester prediction of preeclampsia: A systemic review. *Obstet Gynecol Surv* 2011 Apr; 66(4): 225-39
33. Hofmeyr et al. Calcium supplementation during pregnancy for preventing hypertensive disorders and related problems. *Cochrane database Syst Rev* 2010 Aug 4;(8):CD001059
34. Osterdal ML et al. Does leisure time physical activity in early pregnancy protect against preeclampsia? Prospective cohort in Danish women. *BJOG* 2009 Jan; 116(1):98-107
35. Management of Hypertensive disorders during pregnancy. NICE Clinical Guideline 107, Jan 2011 National Institute of Clinical Excellence.
36. CLASP: a randomized trial of low dose aspirin for prevention and treatment of preeclampsia among 9364 pregnant women. CLASP (Collaborative Low Dose Aspirin Study in Pregnancy) Collaborative Group *Lancet* 1994 Mar 12; 343(8898):619-29
37. Bujold E et al. Acetylsalicylic acid for the prevention of Preeclampsia and intrauterine growth restriction in women with abnormal uterine artery Doppler. A systemic review and Metaanalysis. *J Obstet Gynecol Can* 2009 31:818-26

38. Magee LA et al. Hydralazine for treatment of severe hypertension in pregnancy: Metaanalysis. *BMJ* 2003 Oct25;327(7421):955-60
39. Duley L. Preeclampsia and Hypertensive disorders of pregnancy. *BR Med J* 2003;67;161
40. Wagner LK. Diagnosis and Management of Preeclampsia. *Am Fam Physician* 2004 Dec15;70(12):2317-24
41. Begum R et al. A low dose Magnesium sulphate regime for eclampsia. *ACTA Obstet Gynecol Scand* 2001 Nov;80(11):998-1002
42. Devi GU et al. Unusual accompaniments of pregnancy induced hypertension. *J Obstet Gynecol India* 2001;51(6):9-10
43. Magann Ef, Bass D, Chauhan SP, Sullivan DL, Martin RW, Martin JN Jr, ANtepartam. Corticosteroids: Disease Stabilization in Patients with the Syndrome of Hemolysis, elevated liver enzymes, and low platelets (HELLP). *An J Obstet Gynecol* 1994: 171:1148-53.
44. Fiona M et al. The pre-eclampsia community guideline (PRECOG): How to screen for and detect onset of pre-eclampsia in the community. *BMJ* 2005;30:576-580
45. Seyom et al. Maternal and fetal outcome of pregnancy related hypertension in Mettu Karl Referral Hospital, Ethiopia. *J Ovarian Res.* 2015; 8:10
46. Yadav et al. Hypertensive disorders of pregnancy and maternal and fetal outcomes. *J Indian Med Assoc.* 1997 Oct;95(10):548-51

47. Aabidha et al. Maternal and fetal outcome in pre-eclampsia in a secondary care hospital in South India. J Family Med Prim Care. 2015 Apr-Jun 4(2): 257-260
48. Meshram et al. Maternal and fetal outcomes in Pregnancy Induced Hypertension-A hospital based study. IJPSI. 2014 Apr 3(4): 23-26
49. Bhattacharya Sudhindra Mohan: Pregnancy induced hypertension and prior trophoblastic exposure. J Obstet Gynecol India 2004; 54(6): 568-70
50. Bangal et al. Maternal and fetal outcome in Pregnancy Induced Hypertension: A study from Rural tertiary care teaching hospital in India. IJBR 2(12); 2011:595-599
51. Yucesoy et al. Maternal and perinatal outcome in pregnancies complicated with hypertensive disorder of pregnancy: a seven year experience of a tertiary care centre. Arch Gynecol Obstet. 2005;273:43-49
52. Nankali et al. Maternal Complications associated with Severe Preeclampsia. J Obstet Gynecol India. 2013 Apr; 63(2): 112-115
53. Ahmed et al. Study of Feto-Maternal Outcome in Pregnancy Induced Hyperrrtension. Global Journal of Medical Research. Apr 2104;14(1)

ABBREVIATIONS

GHT – Gestational Hypertension

AP Eclampsia – Antepartum Eclampsia

IP Eclampsia – Intrapartum Eclampsia

PP Eclampsia – Postpartum Eclampsia

Severe PE – Severe Preeclampsia

VEGF – Vascular Endothelial Growth Factor

PIGF – Placental Growth Factor

HELLP – Hemolysis, Low platelets, Low platelets

ARF – Acute Renal Failure

NST – Non Stress Test

CTG – Cardiotocography

LN – Labour Natural

LSCS – Lower Section Caesarean Section

LDH – Lactate Dehydrogenase

AST – Aspartate Transaminase

ALT – Alanine Transaminase

PT – Prothrombin Time

DIC – Disseminated Intravascular Coagulation

PROFORMA

Study No : **Hospital No.** :

Name : **Occupation** :

Age :

Address :

SOCIO- ECONOMIC STATUS:

1. CHIEF COMPLAINTS

2. HISTORY OF PRESENTING COMPLAINTS

H/o months of amenorrhea

C/o pain abdomen yes/no

C/o leaking/bleeding PV

C/o headache/blurring of vision/pedal edema

C/o oliguria / epigastric pain

3. OBSTETRIC HISTORY

Married life

Consanguinity

Obstetric score: Gravida

Para

Living

Abortion

Dead

Booked/ Unbooked

Details of each pregnancy:

No of pregnancies	Period of gestation	Associated complications	Outcome NVD/LSCS	Baby details

Present pregnancy: booked at

Trimester history:

I trimester: H/O excessive vomiting/fever/UTI

Folic acid supplementation

Dating scan

II trimester: Quickening felt at

Anomaly scan

C/O headache/blurring of vision/pedal edema

III trimester: Perceiving fetal movement

C/O headache/pedal edema/blurring of vision

4. MENSTRUAL HISTORY

Age of Menarche:

Flow:

Clots:

Dysmenorrhoea:

LMP: Wks. Days

EDD: Wks. Days

5. PAST HISTORY

TB / Bronchial asthma/ RHD/Blood transfusion / Any operations

6. FAMILY HISTORY

TB / Bronchial Asthma / Diabetes mellitus / Hypertension / Any cancer /

Bleeding disorders / Thyroid disorders

7. PERSONAL HISTORY

Diet :

Appetite :

Bowels :

Micturition :

Sleep :

Excessive intake of coffee :

8. EXAMINATION OF PATIENT:

On examination

1. Height in cms :

2. Pre pregnant Weight :

Present weight :

Weight gain :

BMI :

3. Temperature in degree

4. BP in mm of Hg

5. Respiratory rate per minute

6. Pulse rate

7. Pallor

8. Edema

9. Icterus

Pre pregnant Weight :

Present weight :

Weight gain :

BMI :

Systemic examination

a. CVS

b. RS

c. Per abdomen :

Inspection: abdomen-normal/over distended

Striae gravidarum :

Linea nigra :

Dilated veins/sinuses :

Palpation: Fundal height:

Fundal grip :

Lateral grip :

Pelvic grip :

Liquor: adequate/scanty

Auscultation: FHS (/min)

EFW

Per vagina: cervix dilatation:

Length :

Effacement :

Position :

Consistency :

Vertex station :

Membranes :

Pelvis: adequate/not:

DIAGNOSIS:

Investigations

Hb

RBS

PCV

Blood group

Urine routine: albumin/ Sugar

BT

CT

HIV

HBsAg

VDRL

PIH PROFILE:

Blood urea

Serum Creatinine

Serum Uric acid

LFT

Platelet count:

Lipid profile

Fundoscopy

USG

Electrocardiogram

Echocardiogram

Pregnancy outcome:

- Gestational age at delivery
- Vaginal delivery: assisted/ spontaneous
- Caesarean section
- Maternal complications

Perinatal outcome:

- Live/Still birth/IUD
- Male/female
- Birth weight
- Fetal distress during labor
- APGAR score less than 7
- NICU admission
- Neonatal Deaths
- Neonatal complications

PATIENT INFORMATION SHEET

Place of Study: Chengalpattu Medical College Hospital

Name of the Investigator: Dr. Suganthi P

Name of the Participant: **Age:** **Hospital No:**

Title: “CLINICAL STUDY ON DEMOGRAPHIC PROFILE, MATERNAL AND PERINATAL OUTCOME IN PREGNANT WOMEN WITH HYPERTENSIVE DISORDER”

- You are invited to take part in this study. The information in the document is meant to help you decide whether or not to take part. Please feel free to ask if you have any queries or concerns.

Purpose of the research:

- We are conducting a study on “Demographic profile, Maternal and perinatal outcome in pregnant women with hypertensive disorder”
- The purpose of the study is to find the incidence of hypertensive disorder and fetomaternal morbidity and mortality among these groups.
- The privacy of the patient in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

- Taking part in the study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time. Your decision will not result in any loss of benefits to which you are otherwise entitled
- The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.
- We have obtained permission from the Institutional Ethical Committee.

Principal investigator

DR.SUGANTHI.P

II year MS O & G,

Department of Obstetrics and Gynaecology

Chengalpattu Medical College Hospital,

Chengalpattu

Place:

Date:

Signature of patient

$$\gamma \leftrightarrow \zeta \Phi \downarrow E \lambda \quad \wp \equiv \zeta \neg \wp \rightarrow \dots \kappa \zeta \uparrow \zeta \spadesuit > \uparrow \kappa \wp \uparrow \kappa \Delta$$

$$\gamma \leftrightarrow \zeta \Phi \downarrow E \Sigma f \uparrow \uparrow > \heartsuit \wp \uparrow \Delta \sqrt{f} \Delta \quad : \partial \leftrightarrow \bullet \neg \equiv \uparrow \wp \textcircled{R} \uparrow \therefore \uparrow \uparrow \mu \kappa \Re \uparrow _ \wedge \uparrow \\ \therefore \uparrow \uparrow \mu \kappa \therefore \uparrow \spadesuit \neg \otimes \equiv \\ \uparrow _ \wp \textcircled{R} \uparrow .$$

$$\xi > \uparrow \therefore \gamma \leftrightarrow \zeta \Phi \downarrow E B \zeta \uparrow \uparrow \quad : \therefore \uparrow . \wp . \bullet \uparrow \subseteq \uparrow$$

$$\xi > \uparrow \therefore \kappa \alpha \uparrow \zeta \textcircled{R} \uparrow \quad : \therefore \uparrow . \bullet . \otimes \Delta \wp \uparrow \uparrow \zeta \therefore \zeta \uparrow$$

$$\gamma \Phi \sigma \uparrow > \uparrow \uparrow \heartsuit A \quad : \chi B \uparrow \sqrt{\leftrightarrow} \uparrow \uparrow > \partial \uparrow \uparrow > \mu f \uparrow \partial \angle \therefore \uparrow \Re \uparrow \heartsuit \wp \uparrow \\ \Delta \quad \uparrow \uparrow \div M \neg \wp \uparrow \uparrow \uparrow \therefore \uparrow \rightarrow \Delta \dots$$

$$\otimes \Phi \Re \zeta \textcircled{\circ} \wp \wp \uparrow \Delta \quad \wp \zeta \uparrow \heartsuit A \uparrow \uparrow \\ \wp \wp \uparrow B \gamma \Phi \Upsilon$$

செங்கல்பட்டு அரசு பொது மருத்துவமனையில் மகப்பேறு மற்றும் மகளிர் நல துறையில் ஆராய்ச்சி நடைபெற்றுவருகின்றது.

நீங்களும் இந்த ஆராய்ச்சியில் பங்கேற்க நாங்கள் விரும்புகிறோம் .இதனால் உங்களுக்கு எந்த பாதிப்பும் ஏற்படாது என்பதை தெரிவித்துக் கொள்கிறோம்.

முடிவுகளை அல்லது கருத்துகளை வெளியிடும் போது அல்லது ஆராய்ச்சியின் போது உங்களது பெயரையோ அல்லது அடையாளங்களையோ வெளியிடமாட்டோம் என்பதையும் தெரிவித்துக்கொள்கிறோம்.

இந்த ஆராய்ச்சியில் பங்கேற்பது தங்களுடைய விருப்பத்தின் பேரில் தான் இருக்கிறது. மேலும் நீங்கள் எந்த நேரமும் இந்த ஆராய்ச்சியிலிருந்து பின்வாங்கலாம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

இந்த சிறப்பு பரிசோதனைகளின் முடிவுகளை ஆராய்ச்சியின் முடிவின் போது தங்களுக்கு அறிவிப்போம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

...ΣζB≠λ[¬φυ...⊕ζΓ / φζμ|ζκΓΓ

¬φBΓ:.....
.....

||¬Bζ♥φΔ:.....

...>]:

γ↔ζΦ↓EBζ<Γ

¬φBΓ:.....
.....

||¬Bζ♥φΔ:.....

...>]:

⊗ζ®E 1.

¬φBΓ:.....
.....

||¬Bζ♥φΔ:.....

...>]:

⊗ζ®E 2.

→ ϕB [:.....

|| → Bϕ♥ ϕΔ :

.....

...>]:

MASTER CHART

S. NO	NAME	AGE	PARITY	SE CLASS	HT CLASS	MODE OF DELIVERY	GA AT DELIVERY	MATERNAL COMPLICATIONS	POSTPARTUM RETURN TO BASELINE	BIRTH WEIGHT	FETAL COMPLICATIONS
1	Kowsalya Mani	19	Primi	V	Preeclampsia	LSCS	37 weeks		1 day	2.4 kg	Prematurity
2	Manimegalai	24	Primi	V	Gestational HT	Assisted delivery	38 weeks		1 day	2.3 kg	
3	Revathy	23	Primi	IV	Preeclampsia	LSCS	38 weeks		1 day	2.3 kg	IUGR
4	Poongavanam	33	G2A1	V	Severe Preeclampsia	LSCS	39 weeks		2 weeks	3.3 kg	
5	Hemavathy	26	Primi	V	Preeclampsia	LN	39 weeks		1 day	2.7 kg	
6	Surya Banu	20	Primi	IV	Preeclampsia	LN	36 weeks		2 days	2.8 kg	Prematurity
7	Ragini	26	Primi	V	AP Eclampsia	LSCS	38 weeks		2 weeks	2.4 kg	
8	Sujatha	31	Primi	IV	AP Eclampsia	LSCS	33 weeks	CVA	10 days	1.5 kg	Prematurity
9	Bhuvanawari	21	G3P1L1 A1	IV	Gestational HT	LN	39 weeks		1 day	3.1 kg	
10	Seetha	21	Primi	V	Severe Preeclampsia	Spontaneous expulsion	29 weeks	Abruptio placenta		IUD	IUD
11	Abirami	28	Primi	V	Preeclampsia	LSCS	38 weeks	Abruptio placenta	2 days	2.9 kg	
12	Sujatha	31	Primi	IV	Gestational HT	LN	40 weeks		2 days	2.7 kg	
13	Nandhini	25	G2P1L1	V	Severe Preeclampsia	LSCS	39 weeks		10 days	2.4 kg	
14	Amala	22	G2P1L1	IV	Preeclampsia	LSCS	40 weeks		10 days	2.3 kg	
15	Dhanalakshmi	25	Primi	IV	AP Eclampsia	LN	30 weeks		3 weeks	1.4 kg	Prematurity
16	Janani	27	Primi	V	Severe Preeclampsia	LSCS	39 weeks		1 week	3.2 kg	
17	Ponmari	20	Primi	V	Severe Preeclampsia	LSCS	36 weeks	Abruptio placenta		2.3 kg	Prematurity & IUGR
18	Valli	25	G4P3L3	IV	Severe Preeclampsia	LSCS	32 weeks		1 week	1.4 kg	Prematurity
19	Mahalakshmi	22	Primi	V	Gestational HT	LSCS	40 weeks		2 days	2.8 kg	
20	Anjugam	26	Primi	V	Gestational HT	LSCS	40 weeks		2 days	3.4 kg	
21	Suseela	24	G2P1L1	V	Severe Preeclampsia	LSCS	37 weeks	PPH	2 weeks	2.4 kg	Birth asphyxia

22	Gowthami	25	P1L1	IV	Preeclampsia	LN	39 weeks		2 days	3.1 kg	
23	Valli	25	P4L4	V	Preeclampsia	Assisted delivery	37 weeks		1 day	2.6 kg	Prematurity
24	Lakshmi	24	G2P1L1	V	Gestational HT	LN	39 weeks		1 day	2.6 kg	
25	Subha	21	Primi	IV	Severe Preeclampsia	LSCS	39 weeks		10 days	2.7 kg	
26	Vimala	24	Primi	V	Severe Preeclampsia	LSCS	35 weeks		2 days	1.6 kg	Prematurity & IUGR
27	Silambarasi	30	Primi	V	Gestational HT	LSCS	38 weeks		1 day	2.4 kg	
28	Kavitha	27	G2P1L1	IV	Chronic HT	LSCS	37 weeks			2.7 kg	
29	Sangeetha	26	G2P1L1	IV	Preeclampsia	LSCS	39 weeks		1 day	2.8 kg	
30	Ramya	27	G4P2L2 A1	IV	Severe Preeclampsia	LSCS	35 weeks		2 weeks	1.5 kg	Prematurity & IUGR
31	Mercy	30	G5P2L2 A2	V	Severe Preeclampsia	Spontaneous expulsion	33 weeks	Abruptio placenta	3 weeks	IUD	IUD
32	Anitha	22	G3A2	V	Gestational HT	LN	39 weeks		1 day	2.7 kg	
33	Poongodi	32	G2A1	V	Preeclampsia	LSCS	37 weeks		2 days	2.3 kg	Prematurity & IUGR
34	Tamilselvi	24	Primi	IV	Gestational HT	LSCS	40 weeks		2 days	3.7 kg	
35	Jothi	33	G2P1L1	V	Chronic HT	LSCS	39 weeks			3.2 kg	
36	Vasantha	21	Primi	IV	Severe Preeclampsia	LSCS	36 weeks		2 weeks	2.2 kg	Prematurity
37	Anjali	29	G4P2L1 A1	V	Gestational HT	LSCS	39 weeks		2 days	2.3 kg	
38	Ambika	28	Primi	IV	Severe Preeclampsia	LSCS	39 weeks	Abruptio placenta	10 days	2.8 kg	
39	Hemavathy	22	Primi	IV	Preeclampsia	LSCS	38 weeks		2 days	2.8 kg	
40	Sudha	30	Primi	V	Severe Preeclampsia	LSCS	32 weeks		1 week	1.6 kg	Prematurity
41	Rekha	26	G3P1L1 D1	V	Preeclampsia	LN	39 weeks		2 days	2.9 kg	
42	Gnanasoundh ari	25	Primi	V	Severe Preeclampsia	LSCS	35 weeks		2 weeks	2.2 kg	Prematurity
43	Devaki	20	Primi	IV	Preeclampsia	LSCS	40 weeks	Abruptio placenta	2 days	2.6 kg	
44	Prema	34	G2P1L1	IV	Gestational HT	LN	39 weeks		2 days	2.9 kg	
45	Kabhija	34	G2P1L1	V	Severe Preeclampsia	LSCS	35 weeks		1 week	1.8 kg	Prematurity & IUGR
46	Saranya	25	Primi	V	Preeclampsia	Assisted delivery	36 weeks		2 days	2.2 kg	Prematurity
47	UdhayaPaapa	32	G2P1L1	IV	Severe Preeclampsia	LSCS	38 weeks		10 days	2.1 kg	IUGR
48	Nandhini	24	G4P1L1 A2	V	Preeclampsia	LSCS	38 weeks		1 week	3.3 kg	
49	Revathy	23	Primi	V	Severe Preeclampsia	LSCS	30 weeks			1.6 kg	Prematurity
50	Parimala	24	Primi	V	Preeclampsia	LN	39 weeks		2 days	2.6 kg	
51	Selvi	26	G2A1	IV	Severe Preeclampsia	LSCS	39 weeks	Abruptio placenta	3 weeks	2.4 kg	
52	Thiripurasundh ari	20	Primi	IV	Gestational HT	LN	39 weeks		2 days	2.6 kg	
53	Leena	37	G2P1L1	IV	Preeclampsia	LSCS	39 weeks		1 day	2.7 kg	
54	Shobana	27	G2P1L1	V	Gestational HT	LSCS	38 weeks		2 days	2.7 kg	
55	Ponnamal	26	Primi	V	AP Eclampsia	LSCS	30 weeks		2 weeks	1.4 kg	Prematurity
56	Brindha	21	Primi	IV	Preeclampsia	LN	37 weeks	PPH	2 days	2.6 kg	Prematurity
57	Suganthi	26	G2P1L1	IV	Severe Preeclampsia	LSCS	36 weeks		10 days	1.6 kg	Prematurity & IUGR
58	Geetha	29	Primi	V	Gestational HT		38 weeks		2 days	2.5 kg	
59	Ushaselvi	41	Primi	V	Severe Preeclampsia	LN	38 weeks	PPH		2.7 kg	Birth asphyxia
60	Geetha	25	G2P1L1	IV	Severe Preeclampsia	LSCS	30 weeks	Abruptio placenta	10 days	1.3 kg	Prematurity
61	Punitha	35	Primi	IV	Preeclampsia	LN	37 weeks		2 days	2.4 kg	Prematurity
62	Geetha	30	G2P1L1	V	Severe Preeclampsia	LSCS	39 weeks		2 weeks	2.2 kg	IUGR
63	Anuja	26	P1L1	V	Preeclampsia	LSCS	40 weeks		2 days	2.8 kg	
64	Meenatchi	25	G2P1L1	IV	Severe Preeclampsia	LN	36 weeks	Abruptio placenta	3 weeks	2.2 kg	Prematurity
65	Nandhini	24	P1L1	IV	Preeclampsia	LN	38 weeks		2 days	3.2 kg	
66	Yamunadevi	26	Primi	V	Gestational HT	LSCS	38 weeks		1 day	2.3 kg	

67	Prabhavathi	27	G2A1	V	Severe Preeclampsia	LSCS	32 weeks		1 week	1.4 kg	Prematurity
68	Arulvizhi	24	G4P3L2 D1	IV	Preeclampsia	LN	39 weeks		1 day	2.2 kg	IUGR
69	Suseela	32	G2P1L1	V	Gestational HT	LN	38 weeks		2 days	3.2 kg	
70	Sridevi	30	G4A3	V	Preeclampsia	LN	40 weeks		1 day	3.2 kg	
71	Durga	24	Primi	IV	AP Eclampsia	Spontaneous expulsion	27 weeks	CVA	2 weeks	IUD	IUD
72	Malathi	22	Primi	V	Severe Preeclampsia	LN	35 weeks	PPH	2 weeks	2.1 kg	Prematurity
73	Jothika	24	Primi	IV	Severe Preeclampsia	LSCS	38 weeks			2.9 kg	
74	Banupriya	24	G5P3L2 A1	V	Severe Preeclampsia	LN	39 weeks		10 days	3.3 kg	
75	Suganya	21	P1L1	IV	PP Eclampsia	LN	35 weeks		2 weeks	1.3 kg	Prematurity
76	Divya	21	P1L1	V	PP Eclampsia	LSCS	39 weeks		3 weeks	2.9 kg	
77	Vanaja	24	G2P1L1	IV	AP Eclampsia	LSCS	36 weeks	ARF	2 weeks	2.3 kg	
78	Porkodi	26	Primi	V	AP Eclampsia	LSCS	33 weeks	CVA	1 week	1.6 kg	Prematurity
79	Sridevi	30	G4A3	V	Gestational HT	Assisted delivery	39 weeks		1 day	3.2 kg	
80	Angujarathi	21	Primi	IV	Severe Preeclampsia	LSCS	39 weeks	ARF	3 weeks	2.1 kg	IUGR
81	Priyanka	21	Primi	V	Preeclampsia	Assisted delivery	39 weeks		2 days	2.7 kg	Birth asphyxia
82	Vasanthi	22	Primi	V	AP Eclampsia	LSCS	30 weeks	Pulmonary edema		1.6 kg	Birth asphyxia
83	Merlin	22	G2P1L1	IV	Severe Preeclampsia	LN	36 weeks		2 days	2.6 kg	Prematurity
84	Nivedha	20	Primi	V	Severe Preeclampsia	LN	35 weeks		1 week	1.9 kg	Birth asphyxia & IUGR
85	Saraswathi	21	Primi	V	Gestational HT	LN	39 weeks		1 day	2.9 kg	
86	Nancy	24	G2P1L1	V	Preeclampsia	LN	40 weeks		2 days	2.8 kg	
87	Ramy	23	G2A1	IV	Preeclampsia	LSCS	40 weeks	Abruptio placenta	10 days	2.6 kg	
88	Priyanka	25	P1L1	V	Preeclampsia	LSCS	39 weeks		2 days	2.6 kg	
89	Sherena Banu	25	G3P2L2	IV	Gestational HT	LSCS	40 weeks		2 days	2.8 kg	
90	Jayalakshmi	25	G2P1L1	V	AP Eclampsia	LSCS	37 weeks		1 week	2.7 kg	
91	Malathi	21	Primi	V	Severe Preeclampsia	LN	39 weeks		10 days	2.1 kg	IUGR
92	Bhuvanawari	26	Primi	V	Preeclampsia	LSCS	38 weeks	PPH	2 days	2.3 kg	Prematurity
93	Vijayalakshmi	23	Primi	IV	Gestational HT	LN	40 weeks		1 week	2.7 kg	
94	Jeevitha	26	Primi	IV	Gestational HT	LN	40 weeks		2 days	2.7 kg	
95	Divya	21	Primi	V	AP Eclampsia	LSCS	40 weeks		2 weeks	2.3 kg	
96	Nandhini	23	G2P1L1	IV	Severe Preeclampsia	LSCS	33 weeks	Abruptio placenta	1 week	1.4 kg	Prematurity
97	Rasathi	22	Primi	V	Preeclampsia	LSCS	39 weeks		2 days	2.8 kg	
98	Shanthalakshmi	24	Primi	V	Preeclampsia	LN	40 weeks		1 day	2.7 kg	
99	Jeeva	26	G2P1L1	IV	Gestational HT	LSCS	39 weeks		1 day	2.6 kg	
100	Kalaivani	26	Primi	V	AP Eclampsia	LSCS	38 weeks	Abruptio placenta	1 week	2.4 kg	
101	Sasikala	22	Primi	V	Severe Preeclampsia	LN	38 weeks		10 days	2.8 kg	
102	Shalini	25	Primi	IV	Severe Preeclampsia	Spontaneous expulsion	30 weeks	Abruptio placenta	2 weeks	IUD	IUD
103	Sangeetha	19	Primi	V	Preeclampsia	LN	39 weeks		1 day	3.5 kg	
104	Gowthami	22	G2P1L1	V	Gestational HT	LN	39 weeks		1 day	2.9 kg	
105	Karpagavalli	23	Primi	V	Preeclampsia	LSCS	40 weeks		2 days	2.6 kg	
106	Revathy	19	Primi	IV	Preeclampsia	LN	39 weeks		2 days	2.2 kg	IUGR
107	Kasthuri	21	Primi	V	Gestational HT	Assisted delivery	37 weeks		1 day	2.3 kg	Prematurity
108	Ammu	20	Primi	V	Severe Preeclampsia	LN	37 weeks		3 weeks	1.8 kg	Prematurity & IUGR
109	Fathima	19	Primi	V	Severe Preeclampsia	LSCS	39 weeks		3 weeks	2.2 kg	IUGR
110	Revathy	27	Primi	IV	Preeclampsia	LN	38 weeks		1 day	3.1 kg	
111	Muthulakshmi	23	Primi	V	Severe Preeclampsia	LN	35 weeks		1 day	1.5 kg	Prematurity & IUGR

112	Roobini	20	Primi	V	Severe Preeclampsia	LSCS	34 weeks	Abruptio placenta, HELLP & DIC	3 weeks	1.7 kg	Prematurity
113	Sumathy	24	Primi	V	Preeclampsia	LSCS	39 weeks		3 weeks	2.9 kg	
114	Kavitha	23	G2P1L1	IV	PP Eclampsia	LN	35 weeks		2 weeks	1.5 kg	Prematurity
115	Sindhu	21	Primi	V	Preeclampsia	LN	38 weeks		1 day	2.6 kg	Prematurity
116	Jayasree	21	G2P1L1	V	Gestational HT	LSCS	38 weeks		1 day	3.4 kg	
117	Karpagam	27	Primi	V	Gestational HT	LN	38 weeks		2 days	2.6 kg	
118	Mohanapriya	25	Primi	IV	Severe Preeclampsia	LN	35 weeks	PPH	1 week	1.8 kg	Prematurity & IUGR
119	Vanaja	24	G2P1L1	V	AP Eclampsia	Spontaneous expulsion	26 weeks		10 days	IUD	IUD
120	Sumathy	24	Primi	V	Severe Preeclampsia	LN	40 weeks		2 weeks	2.4 kg	
121	Chitra	32	G2P1L1	V	Severe Preeclampsia	LSCS	39 weeks			3.4 kg	
122	Divya	24	Primi	IV	Preeclampsia	LSCS	39 weeks		1 day	2.3 kg	IUGR
123	Panchali	28	G2P1L1	V	Preeclampsia	LN	40 weeks		1 day	2.7 kg	
124	Sowmya	25	Primi	V	Preeclampsia	LSCS	40 weeks		10 days	2.6 kg	
125	Jayanthi	26	Primi	V	AP Eclampsia	LSCS	30 weeks	HELLP	3 weeks	1.7 kg	Prematurity
126	Anjali	25	Primi	V	Severe Preeclampsia	LSCS	35 weeks		1 week	2.2 kg	Prematurity
127	Saranya	23	Primi	IV	Severe Preeclampsia	LN	38 weeks		1 week	2.7 kg	
128	Kalaiselvi	26	Primi	V	Severe Preeclampsia	LN	35 weeks	Abruptio placenta	10 days	1.6 kg	IUGR & Neonatal death
129	Blessy	27	Primi	V	Severe Preeclampsia	LSCS	34 weeks	Abruptio placenta	3 weeks	1.8 kg	Stillbirth
130	Mohanapriya	26	G2P1L1	V	Preeclampsia	LN	39 weeks		1 day	2.9 kg	
131	Dilshandh	25	G2P1D1	V	Preeclampsia	LSCS	38 weeks		2 days	3.4 kg	
132	Sushmitha	23	Primi	V	Severe Preeclampsia	Spontaneous expulsion	27 weeks		3 weeks	IUD	IUD
133	Sundhari	25	Primi	IV	Severe Preeclampsia	LN	36 weeks		10 days	2.4 kg	
134	Sarasu	23	Primi	V	Preeclampsia	LSCS	38 weeks		1 day	2.3 kg	
135	Dhanakoteeswari	26	Primi	V	Preeclampsia	LSCS	39 weeks	Abruptio placenta	2 days	2.3 kg	
136	Parvarthi	27	G2A1	V	Preeclampsia	LN	38 weeks		2 days	1.7 kg	IUGR
137	Radha	23	G3A1	IV	Severe Preeclampsia	Spontaneous expulsion	36 weeks		10 days	IUD	IUD
138	Vasuki	23	Primi	V	Severe Preeclampsia	LSCS	35 weeks		1 week	1.7 kg	Prematurity & IUGR
139	Manjula	20	P1L1	V	PP Eclampsia	LSCS	39 weeks	CVA	2 weeks	2.3 kg	
140	Selvi	24	Primi	IV	Preeclampsia	LN	37 weeks		2 days	2.4 kg	Prematurity
141	Jayalakshmi	29	G3P2L2	IV	Severe Preeclampsia	LN	32 weeks	Abruptio placenta	10 days	1.3 kg	Prematurity
142	Ammulu	25	Primi	V	Preeclampsia	LN	38 weeks		1 day	2.8 kg	
143	Priya	27	P2L2A1	IV	PP Eclampsia	LSCS	36 weeks		3 weeks	2.8 kg	
144	Shanmugavalli	31	G3P2L2	V	Severe Preeclampsia	LN	38 weeks	PPH	3 weeks	2.7 kg	Birth asphyxia
145	Karuthammal	23	Primi	V	Preeclampsia	Assisted delivery	39 weeks		2 days	2.9 kg	
146	Sheela	24	Primi	IV	AP Eclampsia	LN	37 weeks	PPH	3 weeks	1.7 kg	Birth asphyxia & IUGR
147	Kanniyammal	28	Primi	V	Severe Preeclampsia	LSCS	37 weeks		1 week	2.3 kg	Prematurity
148	Muniyammal	21	Primi	V	Gestational HT	LN	39 weeks	PPH	10 days	2.8 kg	
149	Sathyavathi	21	Primi	IV	Severe Preeclampsia	LN	38 weeks	ARF	10 days	2.2 kg	IUGR
150	Asha	22	G3P2L2	V	Severe Preeclampsia	Spontaneous expulsion	35 weeks		2 weeks	IUD	IUD
151	Rajeswari	30	Primi	IV	Preeclampsia	LN	40 weeks		1 day	2.4 kg	
152	Adhilakshmi	27	Primi	V	Severe Preeclampsia	Spontaneous expulsion	26 weeks	Abruptio placenta	3 weeks	IUD	IUD
153	Uma	21	Primi	V	Preeclampsia	LN	39 weeks		10 days	3.3 kg	
154	Geetha	28	Primi	V	Severe Preeclampsia	LSCS	36 weeks		2 days	1.8 kg	Prematurity & IUGR

155	Nithya	28	Primi	IV	Severe Preeclampsia	LSCS	35 weeks	Abruptio placenta, HELLP, DIC & Maternal Death		2.1 kg	Prematurity
156	Lavanya	23	Primi	V	Gestational HT	LSCS	39 weeks		2 days	2.4 kg	IUGR
157	Kala	24	Primi	V	Preeclampsia	LN	39 weeks		1 day	2.7 kg	
158	LoorthuMary	23	Primi	V	Severe Preeclampsia	LN	34 weeks	Abruptio placenta	3 weeks	2.3 kg	Prematurity
159	Nirmala	28	Primi	IV	Severe Preeclampsia	LN	37 weeks		10 days	2.2 kg	Prematurity
160	Maheswari	28	G2P1L1	V	Severe Preeclampsia	LSCS	37 weeks		1 week	2.8 kg	
161	Renuka	21	Primi	V	AP Eclampsia	LSCS	32 weeks	ARF	3 weeks	1.25 kg	Prematurity
162	Sumathy	34	G3A2	V	Chronic HT	LSCS	36 weeks			2.6 kg	Prematurity
163	Dhanabooshanam	21	Primi	V	Preeclampsia	Assisted delivery	40 weeks		1 day	2.9 kg	
164	Sangeetha	21	Primi	IV	Preeclampsia	LN	40 weeks		1 day	2.6 kg	
165	Pushpa	24	G2P1L1	V	Severe Preeclampsia	LN	33 weeks	Abruptio placenta	2 weeks	1.6 kg	Prematurity
166	Saranya	25	Primi	V	Severe Preeclampsia	LSCS	39 weeks	Abruptio placenta	3 weeks	2.6 kg	
167	Yasmin	26	Primi	V	Preeclampsia	LN	39 weeks		1 day	2.2 kg	IUGR
168	Soundharya	23	Primi	IV	Severe Preeclampsia	LSCS	35 weeks		1 week	1.6 kg	Prematurity & IUGR
169	Revathy	26	G2P1L1	V	Preeclampsia	LSCS	38 weeks		2 days	2.3 kg	IUGR
170	Amala	23	G2P1D1	IV	Gestational HT	Assisted delivery	36 weeks		2 days	2.2 kg	Prematurity & IUGR
171	Rajalakshmi	27	G3P2L2	V	Preeclampsia	LN	39 weeks	PPH	1 day	2.7 kg	
172	Thilakavathy	24	G3P1L1 A1	IV	Severe Preeclampsia	LN	34 weeks		10 days	1.7 kg	Prematurity
173	Balamani	36	G2P1L1	V	Preeclampsia	LSCS	39 weeks		2 weeks	3.4 kg	
174	Dharani	27	Primi	IV	Severe Preeclampsia	Spontaneous expulsion	27 weeks		10 days	IUD	IUD
175	Yuvarani	20	Primi	IV	Preeclampsia	LN	38 weeks		1 day	2.8 kg	
176	Saraswathy	33	G2P1L1	IV	Severe Preeclampsia	LN	35 weeks	PPH	2 weeks	2.3 kg	Prematurity
177	Priya	26	G2A1	IV	Preeclampsia	LN	40 weeks		1 day	3.4 kg	
178	Sivaranjini	21	Primi	V	Preeclampsia	LN	40 weeks	PPH	10 days	2.9 kg	
179	Alfonsa	19	Primi	V	Severe Preeclampsia	LSCS	35 weeks		1 week	1.9 kg	Prematurity & IUGR
180	Kanchana	24	Primi	V	AP Eclampsia	LN	33 weeks		3 weeks	1.4 kg	Prematurity
181	Prema	19	Primi	IV	Gestational HT	LSCS	40 weeks		1 day	3.6 kg	
182	Lalitha	23	Primi	V	Gestational HT	LN	38 weeks		2 days	2.4 kg	
183	Prameela	27	Primi	IV	Gestational HT	Assisted delivery	38 weeks		2 days	2.3 kg	
184	Nandhini	20	Primi	V	Severe Preeclampsia	LSCS	33 weeks	Abruptio placenta, HELLP & DIC		2.2 kg	Birth asphyxia & Prematurity
185	Sofiya	21	Primi	V	Preeclampsia	LSCS	39 weeks		1 day	2.4 kg	
186	Usha	25	Primi	IV	Severe Preeclampsia	LSCS	39 weeks		3 weeks	2.7 kg	
187	Banupriya	23	Primi	V	Severe Preeclampsia	LN	39 weeks		10 days	2.9 kg	
188	Bhavani	21	Primi	IV	Severe Preeclampsia	LSCS	36 weeks		1 week	1.7 kg	IUGR & Neonatal death
189	Shalini	29	G2P1L1	V	Preeclampsia	LSCS	38 weeks		1 day	1.7 kg	Prematurity & IUGR
190	Divyabharathi	24	Primi	V	Severe Preeclampsia	LN	35 weeks		2 weeks	1.8 kg	Prematurity & IUGR
191	Revathy	24	G2P1L1	IV	Severe Preeclampsia	LN	38 weeks	Abruptio placenta	3 weeks	2.1 kg	IUGR
192	Rajapriya	26	Primi	V	Severe Preeclampsia	LN	35 weeks		10 days	2.3 kg	
193	Vasanthi	25	P1L1	V	Preeclampsia	LSCS	39 weeks		2 days	2.1 kg	IUGR
194	Geethanjali	24	Primi	V	Severe Preeclampsia	LSCS	37 weeks		1 week	2.3 kg	Prematurity
195	Geetha	19	Primi	V	Severe Preeclampsia	Spontaneous expulsion	27 weeks	Abruptio placenta	3 weeks	IUD	IUD
196	Keerthi	33	G2P1L1	IV	Severe Preeclampsia	LSCS	37 weeks	Abruptio placenta		2.2 kg	Prematurity
197	Tamilselvi	33	G3P2L2	V	Chronic HT	LN	38 weeks			2.9 kg	

198	Kaveri	20	Primi	V	Preeclampsia	LN	39 weeks		2 days	2.2 kg	IUGR
199	Lavanya	23	G2P1L1	IV	Gestational HT	LSCS	38 weeks		2 days	2.7 kg	
200	Sasikala	26	G2P1L1	V	Severe Preeclampsia	LN	32 weeks		1 week	1.5 kg	Prematurity
201	Amudha	27	G2P1L1	V	Severe Preeclampsia	LSCS	38 weeks		2 days	2.6 kg	
202	Srimathi	21	Primi	IV	AP Eclampsia	LSCS	35 weeks	CVA & Maternal death		1.4 kg	Prematurity
203	Prameela	22	Primi	V	Severe Preeclampsia	LSCS	36 weeks		1 week	1.8 kg	Prematurity & IUGR
204	Alamelumangali	23	Primi	IV	Preeclampsia	Assisted delivery	38 weeks		10 days	2.6 kg	
205	Vimala	27	G3P1L1 A1	IV	Preeclampsia	LN	40 weeks		1 day	2.9 kg	
206	Kalaiarasi	22	Primi	IV	Gestational HT	LSCS	39 weeks		1 day	2.8 kg	
207	Lakshmi	25	Primi	V	Preeclampsia	LN	40 weeks		1 day	3.2 kg	
208	Usha	29	G2P1L1	V	Preeclampsia	LSCS	38 weeks	Abruptio placenta	2 days	1.6 kg	IUGR & Neonatal death
209	Suvalakshmi	25	Primi	V	Preeclampsia	LN	38 weeks		1 day	2.2 kg	IUGR
210	Deepa	24	Primi	IV	Severe Preeclampsia	LSCS	32 weeks		3 weeks	1.7 kg	Prematurity
211	Rajeswari	24	Primi	V	Severe Preeclampsia	LN	37 weeks		1 week	3.2 kg	
212	Rekha	26	G3P2L2	V	Severe Preeclampsia	LSCS	27 weeks		2 weeks	1.3 kg	Prematurity
213	Shakila	27	Primi	IV	Severe Preeclampsia	LSCS	37 weeks		10 days	2.3 kg	IUGR
214	Bavani	24	Primi	V	Preeclampsia	LN	39 weeks		1 week	2.8 kg	
215	Dilshandh	21	Primi	V	Severe Preeclampsia	LN	37 weeks		10 days	2.1 kg	Prematurity & IUGR
216	Shakthi	21	Primi	IV	Severe Preeclampsia	LSCS	33 weeks		2 weeks	1.5 kg	Prematurity
217	Vanitha	21	Primi	V	Severe Preeclampsia	LN	40 weeks		10 days	2.7 kg	
218	Priyanka	27	G3P1L1 A1	V	Severe Preeclampsia	LSCS	36 weeks		3 weeks	2.3 kg	Prematurity
219	Hayathi	21	Primi	IV	Severe Preeclampsia	LN	36 weeks		10 days	2.8 kg	Prematurity
220	Sathyavani	25	Primi	V	Gestational HT	Assisted delivery	39 weeks		2 days	2.3 kg	IUGR
221	Selvi	25	Primi	V	Preeclampsia	LN	39 weeks	PPH	2 days	3.3 kg	Birth asphyxia
222	Maria	24	Primi	IV	Preeclampsia	LN	38 weeks		1 day	2.7 kg	
223	Parimala	18	Primi	V	Severe Preeclampsia	Spontaneous expulsion	26 weeks		2 weeks	IUD	IUD
224	Ishwari	23	Primi	V	Gestational HT	LN	38 weeks		10 days	2.9 kg	
225	Saranya	25	Primi	V	Severe Preeclampsia	LSCS	34 weeks	Abruptio placenta	10 days	2.2 kg	Prematurity
226	Bavani	20	Primi	V	Severe Preeclampsia	LSCS	35 weeks		1 week	1.6 kg	Prematurity & IUGR
227	Banupriya	26	G2P1L1	V	Preeclampsia	LN	39 weeks		3 weeks	2.4 kg	
228	Sathya	22	Primi	V	Severe Preeclampsia	LN	37 weeks	PPH	1 week	2.2 kg	Birth asphyxia
229	Kanniyammal	21	Primi	V	Severe Preeclampsia	LSCS	39 weeks		3 weeks	3.5 kg	
230	Shobana	22	Primi	V	Gestational HT	LSCS	39 weeks		2 days	3.2 kg	
231	Tamilselvi	19	Primi	V	Severe Preeclampsia	LSCS	34 weeks		10 days	1.7 kg	Prematurity
232	Sathya	26	Primi	V	Preeclampsia	LN	36 weeks		1 day	1.8 kg	Prematurity, IUGR & Neonatal Death
233	Pavithra	19	Primi	V	AP Eclampsia	LSCS	36 weeks		3 weeks	2.2 kg	IUGR
234	Suganya	23	Primi	V	Severe Preeclampsia	LN	36 weeks		10 days	2.3 kg	Prematurity
235	Monisha	24	Primi	V	Severe Preeclampsia	LSCS	38 weeks	Abruptio placenta	10 days	2.3 kg	
236	Ramya	20	Primi	V	Preeclampsia	LSCS	38 weeks		1 day	2.8 kg	
237	Hemavathy	26	Primi	V	Severe Preeclampsia	LN	27 weeks		10 days	1.4 kg	Prematurity
238	Kavitha	24	G2A1	V	Severe Preeclampsia	LSCS	36 weeks		3 weeks	2.4 kg	
239	Rajeswari	22	Primi	V	Preeclampsia	LN	39 weeks		1 day	3.1 kg	
240	Sathyavani	27	Primi	V	Severe Preeclampsia	LSCS	36 weeks		1 week	2.3 kg	
241	Saranya	28	Primi	V	Preeclampsia	LN	36 weeks		2 days	2.2 kg	Prematurity

242	Nithya	26	G4P1L1 A2	V	Severe Preeclampsia	LSCS	34 weeks		3 weeks	1.5 kg	Prematurity
243	Radha	28	G2P1L1	V	Preeclampsia	LN	39 weeks		2 days	2.2 kg	
244	Manjula	28	G3P1L1 A1	V	Gestational HT	LN	40 weeks		2 days	2.3 kg	IUGR
245	Selvi	29	G3P2L2	V	Severe Preeclampsia	LN	39 weeks		1 week	2.9 kg	
246	Chamundeeswari	19	Primi	V	Severe Preeclampsia	LN	26 weeks		10 days	1.3 kg	Prematurity & Neonatal death
247	Poosanam	23	Primi	V	AP Eclampsia	LSCS	37 weeks	CVA	10 days	2.3 kg	Birth asphyxia
248	Srisha	20	Primi	V	Severe Preeclampsia	LSCS	35 weeks	Abruptio placenta, HELLP & DIC		2.4 kg	Prematurity
249	Meena	23	G2P1L1	V	Severe Preeclampsia	LSCS	33 weeks	Pulmonary edema		1.5 kg	Prematurity, IUGR & Neonatal Death
250	Suganthi	22	Primi	V	Severe Preeclampsia	LN	37 weeks		2 weeks	2.2 kg	Prematurity & IUGR
251	Mangala	25	Primi	V	Gestational HT	LSCS	40 weeks		1 day	2.9 kg	
252	Sangeetha	19	G3P1L1 A1	V	Preeclampsia	LN	39 weeks		2 days	2.7 kg	
253	Kavitha	22	Primi	V	Severe Preeclampsia	LSCS	37 weeks		3 weeks	2.3 kg	
254	Kanchana	27	Primi	V	Severe Preeclampsia	Spontaneous expulsion	29 weeks		3 weeks	IUD	IUD
255	Abirami	20	Primi	V	Preeclampsia	LSCS	35 weeks		3 weeks	1.6 kg	Prematurity
256	Pachaiammal	23	G2P1L1	V	Preeclampsia	LN	38 weeks		1 day	2.2 kg	IUGR
257	Nalini	26	G3P2L1 D1	V	Severe Preeclampsia	LN	36 weeks	PPH	10 days	2.4 kg	Prematurity
258	Sabreena Begum	21	Primi	V	Severe Preeclampsia	Spontaneous expulsion	26 weeks		2 weeks	IUD	IUD
259	Yuvarani	29	Primi	V	Chronic HT	LSCS	36 weeks			3.4 kg	
260	Ramyia	20	Primi	V	Gestational HT	LN	40 weeks		1 day	3.1 kg	
261	Venda	23	Primi	V	AP Eclampsia	LSCS	38 weeks		10 days	2.4 kg	
262	Angujavalli	30	Primi	V	PP Eclampsia	LN	35 weeks	ARF	1 week	1.7 kg	
263	Valli	21	Primi	V	Severe Preeclampsia	LN	38 weeks		1 week	2.2 kg	IUGR
264	Keerthana	19	Primi	V	Gestational HT	LN	35 weeks		1 day	2.1 kg	Prematurity
265	Jamuna	20	Primi	V	Preeclampsia	Assisted delivery	40 weeks	PPH	10 days	2.4 kg	
266	Kavitha	25	Primi	V	Severe Preeclampsia	Spontaneous expulsion	27 weeks		3 weeks	IUD	IUD
267	Arpudhavalli	30	Primi	V	Severe Preeclampsia	LSCS	34 weeks		1 week	1.5 kg	Prematurity
268	Devi	21	Primi	V	AP Eclampsia	LN	39 weeks		3 weeks	2.4 kg	IUGR
269	Saranya	23	Primi	V	Severe Preeclampsia	LN	37 weeks	PPH	1 week	2.8 kg	
270	Sumithra	22	Primi	V	AP Eclampsia	LSCS	32 weeks		10 days	1.4 kg	Prematurity
271	Kokila	30	G2P1L1	V	Gestational HT	Assisted delivery	39 weeks		2 days	2.8 kg	
272	Valli	22	G2A1	V	Severe Preeclampsia	LSCS	37 weeks		10 days	3.5 kg	
273	Amudha	25	G2P1L1	V	AP Eclampsia	LSCS	36 weeks		3 weeks	2.8 kg	
274	Jayanthi	30	G3P2L2	V	Preeclampsia	LSCS	38 weeks		1 day	3.4 kg	
275	Bhuvanawari	24	G2P1L1	V	Gestational HT	LSCS	39 weeks		1 day	2.7 kg	
276	Brindha	28	Primi	V	Severe Preeclampsia	LSCS	27 weeks	Abruptio placenta	3 weeks	1.4 kg	Prematurity & Neonatal death
277	Hemavathy	23	Primi	V	Severe Preeclampsia	LN	36 weeks		10 days	2.2 kg	Prematurity
278	Samundeeswari	26	Primi	V	Severe Preeclampsia	LSCS	30 weeks	Abruptio placenta	3 weeks	1.7kg	Prematurity
279	Rameera	23	Primi	V	Preeclampsia	LN	39 weeks		1 day	2.4 kg	
280	Meenatchi	26	Primi	V	Preeclampsia	LSCS	40 weeks		2 days	2.3 kg	
281	Devi	28	G3P1L1 A1	V	AP Eclampsia	LN	31 weeks		1 week	1.5 kg	Prematurity
282	Parvatham	20	Primi	V	Severe Preeclampsia	LSCS	35 weeks		2 days	1.5 kg	Prematurity & IUGR
283	Karpagam	20	Primi	V	AP Eclampsia	LSCS	38 weeks			1.7 kg	

284	Dhavamani	27	G2P1D1	V	Gestational HT	LN	37 weeks		1 day	2.3 kg	Prematurity
285	Srimathi	26	G2P1L1	V	Preeclampsia	LSCS	39 weeks		2 days	2.7 kg	
286	Meena	24	G2P1L1	V	Gestational HT	LSCS	38 weeks		2 days	2.7 kg	
287	Mahalakshmi	23	G2P1L1	V	AP Eclampsia	LSCS	38 weeks		1 week	2.2 kg	IUGR
288	Dhanalakshmi	23	G2P1L1	V	Preeclampsia	LN	38 weeks		1 day	2.1 kg	IUGR
289	Vanitha	24	Primi	V	Severe Preeclampsia	LN	32 weeks		3 weeks	1.6 kg	Prematurity & Neonatal death
290	Vijayalakshmi	27	G2P1L1	V	Severe Preeclampsia	Spontaneous expulsion	27 weeks	Abruptio placenta		IUD	IUD
291	Amudha	23	Primi	V	Severe Preeclampsia	LSCS	36 weeks		10 days	2.7 kg	
292	Shalini	25	Primi	V	AP Eclampsia	LSCS	39 weeks		1 week	2.1 kg	
293	Dhanam	22	Primi	V	Severe Preeclampsia	LSCS	35 weeks		10 days	2.3 kg	Prematurity
294	Mubeena Begum	25	G2P1L1	V	Gestational HT	LN	38 weeks		1 day	2.7 kg	
295	Anuselvi	24	Primi	V	AP Eclampsia	Spontaneous expulsion	29 weeks		10 days	IUD	IUD
296	Gayathri	26	Primi	V	Severe Preeclampsia	LN	35 weeks		1 week	2.4 kg	Prematurity
297	Shobana	22	Primi	V	Severe Preeclampsia	LSCS	39 weeks	Abruptio placenta	2 weeks	2.8 kg	
298	Shankari	24	G2P1L1	V	Preeclampsia	LN	37 weeks		1 week	2.2 kg	Prematurity & IUGR
299	Sivagami	25	Primi	V	Severe Preeclampsia	LN	35 weeks	PPH	2 weeks	2.1 kg	Prematurity
300	Sameena	23	Primi	V	Severe Preeclampsia	LSCS	36 weeks		1 week	2.8 kg	
301	Anitha	18	Primi	V	Severe Preeclampsia	LSCS	33 weeks		1 week	1.5 kg	Prematurity
302	Kavitha	19	G2P1L1	V	Gestational HT	LSCS	38 weeks		2 days	2.6 kg	
303	Meena	24	G2P1L1	V	PP Eclampsia	LSCS	39 weeks		2 weeks	2.9 kg	
304	Latha	27	G3P2L2	V	Severe Preeclampsia	Spontaneous expulsion	27 weeks		1 week	IUD	IUD
305	Priya	23	Primi	V	Severe Preeclampsia	LSCS	38 weeks	Abruptio placenta	3 weeks	2.9 kg	
306	Hemavathy	20	G2A1	V	Severe Preeclampsia	LN	33 weeks		10 days	1.6 kg	Birth asphyxia & Prematurity
307	Vasantha	24	Primi	V	Preeclampsia	Assisted delivery	39 weeks		1 day	3.4 kg	
308	Kavya	27	G4P3L3	V	AP Eclampsia	LSCS	38 weeks	CVA	1 week	2.3 kg	
309	Sasikala	19	G2A1	V	Severe Preeclampsia	LSCS	36 weeks			2.3 kg	Prematurity
310	Ramya	29	G3P2L2	V	Preeclampsia	LSCS	38 weeks		3 weeks	2.3 kg	IUGR
311	Meena	21	G2A1	V	Gestational HT	LN	39 weeks		2 days	2.6 kg	
312	Saranya	20	Primi	V	Severe Preeclampsia	LN	36 weeks		10 days	2.1 kg	Prematurity
313	Vasanthi	18	G2P1L1	V	Preeclampsia	LSCS	39 weeks	Abruptio placenta	2 days	2.8 kg	
314	Devi	19	Primi	V	AP Eclampsia	LSCS	39 weeks		1 week	2.7 kg	
315	Kanchana	22	G2P1L1	V	Severe Preeclampsia	LSCS	27 weeks	PPH	3 weeks	1.3 kg	Prematurity
316	Kalaïarasi	28	G2P1L1	V	Gestational HT	LSCS	39 weeks		2 days	3.2 kg	
317	Mohanapriya	23	Primi	V	Preeclampsia	LSCS	39 weeks		2 days	2.6 kg	
318	Surya	24	G2P1L1	V	AP Eclampsia	LSCS	38 weeks	Abruptio placenta, HELLP, DIC, Pulmonary edema & Maternal Death		1.4 kg	IUGR
319	Lalitha	26	G3A2	V	Severe Preeclampsia	LN	35 weeks		1 week	1.6 kg	Prematurity & IUGR
320	Kalaivani	25	Primi	V	Preeclampsia	LN	40 weeks		2 days	2.9 kg	
321	Saranya	21	G2P1L1	V	Gestational HT	LN	38 weeks		1 day	2.3 kg	
322	Meena	28	G3P2L2	V	Preeclampsia	LN	39 weeks		2 days	2.4 kg	Birth asphyxia
323	Lakshmi	17	Primi	V	AP Eclampsia	LN	30 weeks	ARF		1.7 kg	Prematurity
324	Sarala	22	G2P1L1	V	Severe Preeclampsia	LSCS	35 weeks		1 week	1.8 kg	Prematurity & IUGR
325	Laila	18	Primi	V	Preeclampsia	Assisted delivery	40 weeks		1 week	2.9 kg	

326	Vanitha	20	G2A1	V	Severe Preeclampsia	LN	36 weeks	PPH	10 days	2.8 kg	Prematurity
327	Suryakala	23	Primi	V	Gestational HT	LN	39 weeks		1 day	2.7 kg	
328	Manopriya	24	G2P1L1	V	Preeclampsia	LSCS	40 weeks		2 days	2.6 kg	
329	Subashini	29	G4P2L2 A1	V	AP Eclampsia	LSCS	35 weeks		3 weeks	1.6 kg	Prematurity & Neonatal death
330	Laila	20	Primi	V	Severe Preeclampsia	LSCS	36 weeks		2 weeks	1.9 kg	Prematurity
331	Revathy	19	G2P1L1	V	Severe Preeclampsia	LSCS	37 weeks			2.2 kg	Prematurity
332	Gomathi	18	Primi	V	Preeclampsia	LSCS	35 weeks		2 days	2.2 kg	Prematurity
333	Sheela	25	G2A1	V	Severe Preeclampsia	LSCS	37 weeks		3 weeks	2.3 kg	
334	Rani	22	G2P1L1	V	Preeclampsia	LSCS	39 weeks		1 day	2.2 kg	
335	Lakshmi	27	G3P1L1 A1	V	Severe Preeclampsia	LN	36 weeks		10 days	1.9 kg	Prematurity & IUGR
336	Kanaga	20	Primi	V	Gestational HT	LSCS	39 weeks		2 days	3.3 kg	
337	Malini	23	G2P1L1	V	Severe Preeclampsia	LSCS	32 weeks	Abruptio placenta		1.8 kg	Prematurity
338	Manjula	18	Primi	V	Gestational HT	LN	40 weeks		1 day	2.8 kg	
339	Samithra	26	G3P2L2	V	Preeclampsia	LN	39 weeks		1 day	3.4 kg	
340	Kanaga	19	G2P1L1	V	Gestational HT	LN	36 weeks		2 days	2.9 kg	Prematurity
341	Karpagam	17	Primi	V	Severe Preeclampsia	LSCS	35 weeks		1 week	2.1 kg	Prematurity
342	Thamarai	24	Primi	V	Preeclampsia	Assisted delivery	38 weeks		10 days	2.4 kg	
343	Vaidehi	18	Primi	V	AP Eclampsia	Hysterotomy	33 weeks		2 weeks	1.5 kg	Prematurity
344	Muniyammal	20	G2P1L1	V	Severe Preeclampsia	LSCS	39 weeks		2 days	2.8 kg	
345	Kasiyammal	25	G3P2L2	V	Preeclampsia	LSCS	38 weeks	Abruptio placenta	2 days	2.3 kg	
346	Shobana	20	Primi	V	Severe Preeclampsia	LSCS	35 weeks		10 days	1.6 kg	Birth asphyxia, Prematurity & Neonatal death
347	Anisha	27	G4P3L3	V	Severe Preeclampsia	LSCS	33 weeks			1.5 kg	Prematurity
348	Devikala	25	Primi	V	Preeclampsia	LSCS	39 weeks		1 day	1.8 kg	IUGR
349	Swathi	25	G2P1L1	V	Gestational HT	LSCS	40 weeks		2 days	3.5 kg	
350	Susila	22	Primi	V	Gestational HT	LN	38 weeks		1 day	2.7 kg	
351	Meena	29	G4P3L3	V	Severe Preeclampsia	Spontaneous expulsion	27 weeks	Abruptio placenta	3 weeks	IUD	IUD
352	Chitra	20	Primi	V	Severe Preeclampsia	LSCS	36 weeks	Abruptio placenta, HELLP & DIC		2.2 kg	Prematurity
353	Ambika	19	G2A1	V	Severe Preeclampsia	LN	38 weeks		10 days	2.7 kg	
354	Priya	30	Primi	V	PP Eclampsia	LN	36 weeks		3 weeks	1.8 kg	
355	Srivathy	23	G3P2L2	V	Preeclampsia	LN	37 weeks		1 day	2.2 kg	Prematurity & IUGR
356	Sangeetha	23	Primi	V	Gestational HT	LN	39 weeks		1 day	2.9 kg	
357	Dhanalakshmi	27	G3P2L2	V	Gestational HT	LN	38 weeks		1 day	2.6 kg	
358	Kokila	28	G3P1L1 A1	V	Severe Preeclampsia	LSCS	40 weeks		10 days	3.2 kg	
359	Poornima	19	Primi	V	Severe Preeclampsia	LN	39 weeks	PPH	1 week	2.8 kg	
360	Usha	18	Primi	V	AP Eclampsia	LSCS	37 weeks	Abruptio placenta	1 week	2.3 kg	
361	Ammu	20	G2P1L1	V	Severe Preeclampsia	LN	37 weeks		1 week	1.9 kg	Prematurity & IUGR
362	Sathyapriya	24	Primi	V	Severe Preeclampsia	LSCS	38 weeks		3 weeks	2.3 kg	
363	Mercy	19	Primi	V	Severe Preeclampsia	LN	37 weeks	PPH	10 days	2.4 kg	Prematurity
364	Rooba	25	G3A2	V	Gestational HT	Assisted delivery	38 weeks		2 days	3.6 kg	
365	Fathima	18	Primi	V	Severe Preeclampsia	Spontaneous expulsion	26 weeks	Retinopathy		IUD	IUD
366	Yamuna	24	G2P1L1	V	Preeclampsia	LN	38 weeks		1 day	2.2 kg	IUGR
367	Sheela	19	G2P1L1	V	AP Eclampsia	LN	39 weeks	CVA		2.7 kg	Birth asphyxia
368	Kanmani	27	G4P1L1 A2	V	Severe Preeclampsia	LN	39 weeks		1 day	2.8 kg	

369	Vijayalakshmi	22	Primi	V	Preeclampsia	LSCS	39 weeks		1 week	2.2 kg	
370	Divyabharathi	20	G2A1	V	Severe Preeclampsia	LSCS	37 weeks	Abruptio placenta		2.1 kg	Stillbirth
371	Bharathi	19	G2P1L1	V	Preeclampsia	LN	40 weeks		2 days	2.8 kg	
372	Nithya	18	Primi	V	AP Eclampsia	LSCS	38 weeks		1 week	2.4 kg	
373	Deepa	21	G2P1L1	V	Preeclampsia	LSCS	40 weeks		2 days	2.9 kg	
374	Aarthi	28	G3P2L2	V	Severe Preeclampsia	LN	36 weeks		1 week	2.3 kg	Prematurity
375	Priya	18	Primi	V	Preeclampsia	LN	39 weeks		2 days	2.6 kg	
376	Aruna	19	Primi	V	AP Eclampsia	LSCS	39 weeks		1 week	2.9 kg	
377	Malathi	22	G2P1L1	V	Severe Preeclampsia	LSCS	38 weeks	PPH	10 days	2.6 kg	
378	Yamuna	23	G2P1L1	V	Gestational HT	LSCS	38 weeks		2 days	2.7 kg	
379	Rajakumari	24	Primi	V	Gestational HT	LN	39 weeks		1 day	2.8 kg	
380	Thenmozhi	20	G2P1L1	V	Preeclampsia	Assisted delivery	39 weeks		1 day	2.4 kg	
381	Rudra	22	G2P1L1	V	AP Eclampsia	LN	31 weeks		10 days	1.9 kg	Prematurity
382	Yashodha	23	G3A2	V	Severe Preeclampsia	LN	36 weeks	PPH	10 days	2.4 kg	Prematurity
383	Gokulapriya	19	Primi	V	Preeclampsia	LN	40 weeks		1 day	2.6 kg	
384	Kannagi	18	Primi	V	AP Eclampsia	LSCS	37 weeks		10 days	1.6 kg	IUGR
385	Muniyammal	21	G2P1L1	V	Severe Preeclampsia	LSCS	35 weeks		3 weeks	1.6 kg	Prematurity
386	Manjula	22	Primi	V	Gestational HT	LN	38 weeks		2 days	2.8 kg	
387	Kalaivani	23	G3P2L2	V	Gestational HT	LSCS	39 weeks		1 day	3.2 kg	
388	Swathi	18	Primi	V	Gestational HT	LN	39 weeks		1 day	2.9 kg	
389	Devi	20	Primi	V	AP Eclampsia	LSCS	38 weeks		2 weeks	1.7 kg	IUGR & Neonatal death
390	Rani	25	G3P2L2	V	Preeclampsia	LSCS	40 weeks	Abruptio placenta	2 days	2.8 kg	
391	Anitha	24	Primi	V	Severe Preeclampsia	LSCS	37 weeks		10 days	2.2 kg	Prematurity
392	Ambikadevi	20	Primi	V	AP Eclampsia	LN	39 weeks		10 days	2.4 kg	
393	RAni	18	Primi	V	Severe Preeclampsia	Spontaneous expulsion	25 weeks	Abruptio placenta		IUD	IUD
394	Bagyalakshmi	25	Primi	V	Preeclampsia	LSCS	40 weeks		1 day	2.9 kg	
395	Thulasi	19	Primi	V	PP Eclampsia	LSCS	35 weeks			1.7 kg	
396	Tamilselvi	25	Primi	V	Severe Preeclampsia	LN	39 weeks	PPH	1 week	2.7 kg	
397	Paanjali	24	Primi	V	Gestational HT	LSCS	40 weeks		2 days	3.5 kg	
398	Sathyavathy	23	Primi	V	Preeclampsia	LSCS	40 weeks		2 days	2.4 kg	
399	Shankari	19	Primi	V	AP Eclampsia	LSCS	36 weeks		10 days	1.9 kg	
400	Malliga	20	Primi	V	Severe Preeclampsia	Spontaneous expulsion	25 weeks		3 weeks	IUD	IUD
401	Vanitha	21	Primi	V	Preeclampsia	LSCS	40 weeks		2 days	3.6 kg	
402	Seetha	22	Primi	V	IP Eclampsia	LN	38 weeks		10 days	2.4 kg	Birth asphyxia
403	Amsa	22	Primi	V	Gestational HT	LSCS	39 weeks		2 days	3.4 kg	
404	Faridha	20	Primi	V	Preeclampsia	LN	36 weeks		3 weeks	2.8 kg	Prematurity
405	Monisha	23	Primi	V	AP Eclampsia	LSCS	39 weeks	Abruptio placenta	1 week	2.7 kg	
406	Jagadheeswari	25	Primi	V	Preeclampsia	LSCS	40 weeks		1 day	2.6 kg	
407	Kumari	25	G3P2L2	V	PP Eclampsia	LSCS	38 weeks		3 weeks	1.7 kg	IUGR
408	Sivagami	19	Primi	V	Severe Preeclampsia	LN	36 weeks		1 week	2.3 kg	Birth asphyxia
409	Lakshmi	24	G4P1L1 A2	V	Gestational HT	LN	35 weeks		2 days	2.3 kg	Prematurity
410	Radha	18	Primi	V	Preeclampsia	LSCS	39 weeks		10 days	2.3 kg	
411	Bhuvana	25	G2P1L1	V	Severe Preeclampsia	LSCS	36 weeks		10 days	2.7 kg	Prematurity
412	Ramani	19	Primi	V	AP Eclampsia	LSCS	38 weeks		3 weeks	IUD	IUD
413	Nathiya	21	Primi	V	Gestational HT	LSCS	39 weeks		1 day	2.7 kg	

414	Sridevi	20	Primi	V	Severe Preeclampsia	LN	39 weeks		1 week	2.8 kg	
415	Jhansi	22	Primi	V	Severe Preeclampsia	LSCS	36 weeks		2 weeks	2.4 kg	Prematurity
416	Renuga	23	Primi	V	AP Eclampsia	LN	37 weeks		10 days	1.8 kg	IUGR
417	Mullai	18	Primi	V	Severe Preeclampsia	LSCS	35 weeks	Abruptio placenta, HELLP & DIC		1.7 kg	Prematurity, IUGR & Neonatal Death
418	Ganga	24	Primi	V	Gestational HT	LSCS	39 weeks		1 day	3.4 kg	
419	Rubini	25	G2P1L1	V	Preeclampsia	LN	39 weeks		1 day	2.1 kg	IUGR
420	Govindhammal	19	G2A1	V	Preeclampsia	LSCS	40 weeks		1 day	2.7 kg	
421	Sreedevi	21	Primi	V	Severe Preeclampsia	LN	27 weeks	Abruptio placenta		1.4 kg	Prematurity & Neonatal death
422	Indra	21	Primi	V	AP Eclampsia	LSCS	30 weeks		10 days	1.5 kg	Prematurity & Neonatal death
423	Suganya	23	G2P1L1	V	Gestational HT	LN	39 weeks		2 days	2.8 kg	
424	Srimathi	18	Primi	V	Severe Preeclampsia	LN	38 weeks		10 days	3.4 kg	
425	Noornisha	19	Primi	V	Gestational HT	LSCS	39 weeks		1 day	2.9kg	
426	Parvathi	22	G2P1L1	V	Preeclampsia	LN	39 weeks		1 day	2.9 kg	
427	Kanniyammal	22	Primi	V	Gestational HT	LN	38 weeks		1 day	3.2 kg	
428	Niranjana	20	Primi	V	AP Eclampsia	LN	36 weeks		10 days	1.9 kg	
429	Archana	23	Primi	V	Severe Preeclampsia	Spontaneous expulsion	26 weeks	PPH	2 weeks	IUD	IUD
430	Ammu	19	Primi	V	Preeclampsia	LSCS	40 weeks		2 days	3.2 kg	
431	Valli	25	G2P1L1	V	PP Eclampsia	LSCS	39 weeks			1.9 kg	
432	Kiruba	18	Primi	V	AP Eclampsia	LSCS	36 weeks	Abruptio placenta	1 week	1.6 kg	Prematurity & Neonatal death
433	Saritha	25	G2P1L1	V	Gestational HT	LN	38 weeks		1 day	2.8 kg	
434	Vanmathi	24	G2P1L1	V	Gestational HT	LSCS	39 weeks		1 day	3.4 kg	
435	Tamilmani	19	Primi	V	AP Eclampsia	LN	35 weeks		10 days	1.6 kg	Prematurity
436	Kalpana	21	Primi	V	Preeclampsia	LN	35 weeks		3 weeks	2.1 kg	Prematurity
437	Gauthami	19	Primi	V	Gestational HT	LN	39 weeks		1 day	2.9 kg	
438	Valli	18	G2A1	V	Preeclampsia	LSCS	40 weeks	Abruptio placenta	10 days	2.6 kg	
439	Padmini	22	G2A1	V	Severe Preeclampsia	LSCS	36 weeks		1 week	2.7 kg	Prematurity
440	Vembu	23	Primi	V	AP Eclampsia	LSCS	37 weeks	CVA	3 weeks	1.9 kg	
441	Madhavi	20	Primi	V	Preeclampsia	LN	40 weeks		1 day	2.7 kg	
442	Maheswari	22	G2P1L1	V	Preeclampsia	LSCS	39 weeks		1 day	2.3 kg	
443	Gowri	18	Primi	V	Gestational HT	Assisted delivery	39 weeks		2 days	2.4 kg	
444	Rasathi	23	G2A1	V	Gestational HT	LSCS	38 weeks		2 days	2.6 kg	
445	Ammulu	23	Primi	V	Severe Preeclampsia	LN	38 weeks	PPH	3 weeks	2.8 kg	
446	Preetha	19	Primi	V	Severe Preeclampsia	LSCS	35 weeks		10 days	1.8 kg	Prematurity & IUGR
447	Vidhya	24	G2P1L1	V	Severe Preeclampsia	Spontaneous expulsion	25 weeks			IUD	IUD
448	Arivazhagi	18	Primi	V	AP Eclampsia	LN	28 weeks		1 week	0.8 kg	Prematurity & Neonatal death
449	Valarmathi	25	G2P1L1	V	Preeclampsia	LN	39 weeks		1 week	2.3 kg	
450	Deepthi	19	Primi	V	Gestational HT	LSCS	39 weeks		1 day	3.2 kg	