LOWERING PLATELET COUNT ,A PROGNOSTIC INDEX IN PREECLAMPSIA

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MAY - 2020

BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled **"LOWERING PLATELET COUNT ,A PROGNOSTIC INDEX IN PREECLAMPSIA "** is a bonafide record of original work done by **Dr. SHAMRATH BANU.C** under the guidance of **Dr. K.KANMANI, M.D.,O.G,** Professor of Obstetrics and Gynecology in Institute of social obstetrics and Kasturba Gandhi hospital ISO KGH, Chennai in partial fulfillment of the requirements for MS Degree in Obstetrics and Gynecology branch II examination of the Tamil Nadu Dr.MGR Medical university to be held in MAY 2020. The period of post graduate study and training from MAY 2017 to MAY 2020**.**

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DECLARATION

I,**Dr**. **SHAMRATH BANU.C**, Post Graduate, Department of Obstetrics and Gynaecology, Madras Medical College solemnly declare that this dissertation" **LOWERING PLATELET COUNT , A PROGNOSTIC INDEX IN PREECLAMPSIA"** was prepared by me at Department of Obstetrics and Gynecology, Madras medical college, Chennai, under the guidance of professor **DR.K.KANMANI MDOG**,institute of social obstetrics and Kasturba Gandhi hospital, Chennai

This dissertation is submitted to **The Tamil Nadu Dr.M.G.R. Medical University, Chennai** in partial fulfillment of the University regulations for the award of the degree of **M.S. (Obstetrics and Gynaecology).**

Place: Chennai
Date:

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(SHAMRATH BANU.C)

CONTENTS

INTRODUCTION

Hypertensive disorders complicating pregnancy are common and form one of the deadly triad along with hemorrhage and infections that result in much of the maternal morbidity and mortality related to pregnancy. Hypertensive disorders complicate 5-10 % of all pregnancies . Recent advances in research about pregnancy induced hypertension have facilitated a better general understanding of the pathophysiology of the disease.Incidence is influenced by genetic predisposition. The incidence of pregnancy induced hypertension is between 12% to 15% in Primigravidas and 2 to 4% in Multigravidas. The incidence of preeclampsia is 5 to 11 % in primi and 1.4 to 4% in multi and incidence of eclampsia is 0.5 to 2% of all pregnancies. Maternal mortality rate due to hypertensive disorders is 16%.This percentage is greater than other three leading causes:

Hemorrhage - 13%

Abortion - 8%,

Sepsis – 2%

The condition is more frequent in obese women and in women with multiple gestation, diabetes, chronic hypertension and previous history of preeclampsia. A number of classifications and definitions of various hypertensive disorders of pregnancy exist and new ones are being put forward constantly.

The overwhelming majority of cases can be included into 5 well defined groups: , Gestational hypertension, Preeclampsia, Eclampsia, preeclampsia superimposed on chronic hypertension,chronic hypertension .

CLASSIFICATION OF HYPERTENSIVE DISORDERS IN PREGNANCY

According to NHBPEP

I. GESTATIONAL HYPERTENSION

Systolic BP $\geq$ 140 or diastolic Bp $\geq$ 90 mmHg for the first time during pregnancy.

No proteinuria

Bp returns to normal before 12 weeks post partum.

May have imminent signs and symptoms of pre-eclampsia, like blurring of vision,headache

2.**PRE ECLAMPSIA**

Diagnosed when there is

Bp $\geq$ 140/90 mmHg after 20 weeks of gestation.

Proteinuria $\geq$ 300 mg/24 hours or $\geq$ 1 + in dipstick

SEVERE PRE ECLAMPSIA

Bp $\geq$ 160/110 mmHg for the first time during pregnancy.

Proteinuria 2.0 g/24 hours or $\geq$ 2 + dipstick

Serum creatinine > 1.2 mg/dl unless known to be previously elevated

Platelets < 1,00,000 / cu mm

Microangiopathic hemolysis

Elevated serum transaminase levels – ALT or AST

Severe headache , blurring of vision, vomiting

Persistant discomfort in epigastric region

Pulmonary edema

Oliguria < 500ml/ 24 hrs

Fetal growth restriction

3.ECLAMPSIA

Generalised tonic clonic seizures occurring in an antenatal women with preeclampsia which cannot be contributed to other causes

4.. PRE-ECLAMPSIA SUPERIMPOSED ON CHRONIC HYPERTENSION

New onset proteinuria $\geq$ 300 mg/24 hours in hypertensive women but no proteinuria before 20 weeks gestation

A sudden increase in proteinuria or blood pressure or

platelet count < 1,00,000/microlitre in women with chronic hypertension

5.CHRONIC HYPERTENSION

Bp > 140/90 mmHg before conception or identified before 20 weeks gestation not including gestational trophoblastic disease, twin gestation

(or)

Hypertension first identified after 20 weeks gestation and persisting even after 12 weeks postpartum.

HELLP SYNDROME

It is characterized by Hemolysis, Elevated liver enzymes and Low Platelet count. It is a life-threatening condition complicating pregnancy. In patients with preeclampsia, it may lead to fetal and maternal death.The term 'HELLP syndrome' was coined by Louis Weinstein in 1982.

Weinstein identified around 30 cases of severe preeclampsia–eclampsia complicated by thrombocytopenia, abnormal peripheral blood smear and abnormal LFT - liver function test. He identified that these abnormalities constituted a separate entity from the general observed eclampsia and hence gave the name HELLP syndrome (H - hemolysis; EL - elevated liver enzymes; and LP - low platelets)

The pathophysiology of HELLP syndrome is unclear. The findings of this multi system disease are attributed to abnormal vascular tone, vasospasm and coagulation defects. No precipitating factor has been found till date. This syndrome occurs as final manifestation of a series of endothelial damage and intravascular platelet activation. With platelet activation thromboxane A and serotonin are released, causing vasospasm, platelet agglutination and aggregation, and further endothelial damage. This begins a cascade that is only terminated after delivery

Some authors describe HELLP as a variant of preeclampsia, as both seem to have a common pathophysiology.Various factors such as soluble vascular endothelial growth factor receptor-1 (sVEGFR-1), are released which then binds vascular endothelial growth factor (VEGF) and placental growth factor (PGF). This causes dysfunction of endothelial cell along with placental dysfunction. It thereby prevents the binding of endothelial cell receptors with the placenta, resulting in hypertension, proteinuria, and increased platelet activation and aggregation.

The endothelial cells, when activated may release von Willebrand factor multimers. They are highly reactive with platelets. HELLP syndrome shows increased amount of active VWF leading to thrombocytopenic and thrombotic microangiopathy.

In recent studies have shown that fetal complications of mitochondrial fatty acid oxidation is assosiated with obstetric complications like placental bed infarct, pre-eclampsia, acute fatty liver of pregnancy and also the HELLP syndrome. Among some patients who are heterozygous with a defect in the long chain hydroxylacyl – co- A dehydrogenase (LCHAD)

enzyme and who also have a defect in fetus homozygous, these disorders have shown to have occured.The pathogenesis of HELLP syndrome is still unknown.

Pregnancy in fourth decade i.e, after 30 years of age and multiparity are considered to be the high-risk factors of HELLP syndrome. Statistics show that HELLP syndrome occurs in 0.5% of all pregnancies and among 8% of preeclampsia cases. This syndrome has shown to have occurred typically between third trimester of pregnancy and delivery.

PROTEINURIA :-

It can be expressed as excretion of 300 mg/24 hrs urine or a urine protein/creatinine ratio of 30 mg/mmol or, urine dipstick ≥ 1 + on dipstick or more in at least two random urine samples collected at least 4 to 6 hours apart. Urinary tract infection should be excluded. This Dipstick testing for proteinuria can be used as a screening test. But the test has very high false positive and more important false negative rates

RISK FACTORS FOR PRE-ECLAMPSIA

Couple related risk factors

Primipaternity

Limited sperm exposure

Pregnancies after Donor insemination, Oocyte donation, embryo donation

Maternal or Pregnancy – related risk factors

Extremes of maternal age

Multifetal pregnancy

Any form of hypertension in prior pregnancy

Chronic hypertension and/or renal disease

Maternal low birth weight

Obesity and Insulin resistance (with and without PCOS ; the risk proportionate to BMI)

Overt diabetes mellitus

Patients of thrombophilias getting conceived

Maternal susceptibility genes

Family history of pre-eclampsia

Smoking

LOWERING PLATELET COUNT A PROGNOSTIC INDEX IN PREECLAMPSIA

Objective: Objective of my study is to know variation of platelet count in normal pregnancy and hypertensive disorders of pregnancy and correlate fetomaternal outcome with platelet count .Early detection of platelet count abnormalities in preeclampsia and eclampsia, which is a sign of worsening disease facilitates early detection of maternal and fetal complications, thereby its role as prognostic tool in management .

Study Centre : institute of social obstetrics kasturba Gandhi hospital, Madras medical college,Chennai.

Duration of Study: 1 year (may 2018 to may 2019)

Study Design: prospective comparative study(cohort study)

The study group included 150 women with gestational hypertension, pre eclampsia and eclampsia with varying severity and duration of pregnancy. The control group included 150 women with similar demographic features and no associated complications. All the cases were selected from antenatal clinic, labor room and ward of obstetrics and gynecology department. A demographic sheet and laboratory tests were used in this study. The demographic sheet included information about the name,

age, parity of patient, duration of amenorrhea, last menstrual period, expected date of delivery, obstetric formula, compliant of the patient if any, blood pressure, systemic examination, abdominal examination, per speculum and per vaginal examination, mode of delivery, complications if any, baby details, investigations mainly platelet count done at interval along with other PIH investigations and USG**.**

After completing the demographic sheet, all the patients taken up for study were subjected to laboratory investigations. Consent is obtained.Blood is taken every week from third trimester for study group and every 4 weeks for control group .5ml of venous blood sample were aspirated from the participants ante cubital vein and mixed with EDTA (Ethylene diamine triacetic acid). The blood is mixed well and placed on a rack in an analyzer. The instrument has flow cells, photometers and apertures that analyses different elements in the blood. The cell counting components counts the number and types of different cells in the blood. The results are printed.

In patients with very low platelet count, the counts are rechecked using manual method. In manual method, whole blood is diluted with 1% ammonium oxalate solution. The isotonic balance of the diluents is such that all the erythrocytes are destroyed while platelets and leucocytes remain

intact. The standard dilution for platelet is 1:100. The dilution is prepared using leucocyte/platelet unopette system. The dilution is mixed well and incubated to permit the lysis of the erythrocytes following incubation period. The dilution is mounted on a hemocytometer chamber under the microscope. The cells are allowed to settle and then are counted in a specific area of hemocytometer under the microscope. The number of platelet is calculated per micro liter**.**

- **Platelet values.**
- **Normal 1.5 to 4 Lakhs /mm3**
- **Thrombocytopenia <1.5 lakhs/mm3.**
- **Critical count <50,000/mm3.**

Inclusion criteria women with hypertensive disorders according to ACOG guidelines .diagnostic criteria as follows

Gestational hypertension	Bp>140/90mmhg after 20weeks in previously normotensive without proteinuria
Preeclampsia hypertension and Proteinuria	≥ 300 mg/24h, or Protein: creatinine ratio ≥ 0.3 or dipstick 1+ persistant
Thrombocytopenia	platelets < 100,000/μL
Renal insufficiency	creatinine > 1.1 mg/dL or doubling of baseline
Liver involvement	Serum transaminase levels twice normal
Cerebral symptoms	Headache visual disturbamces,convulsions
Pulmonary edema	

- **Exclusion Criteria:**
- Women with hemorrhagic disorder
- Thromboembolic episode
- Epilepsy
- Hepatic diseases
- Renal diseases
- Drugs which alter platelet count and function
- **Sample size**: 150

From comparing the platelet count , increasing and decreasing trend mean value for each group is determined from which p value is calculated

to specify relationship, a comparison was made between number of cases in control and study group with normal ,low, and very low platelet count. in the present study, fetal and maternal outcome was compared between control group women and in women with pre eclampsia and eclampsia associated with thrombocytopenia. further, the association of thrombocytopenia with fetal and maternal outcome was studied. frequency distribution with class intervals was carried out for selected variables. chi-square test of association was carried out to study the association of thrombocytopenia with the maternal and fetal outcome. confidence interval will be calculated for individual parameters.p values< 0.05 will be considered as significant the data thus collected were analyzed using appropriate statistical method

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REVIEW OF LITERATURE

Evaluation of platelet count as a prognostic index in eclampsia and pre eclampsia

By Dr. R. Vinodhini and 2 Dr. Lavanya Kumari et al

In test group patients with significant thrombocytopenia the mean duration of pregnancy was reduced with higher incidences of either still birth, low birth weight babies, with an increase in operative intervention

Feroza Sultana1, Raja Parthiban2, Shameem Shariff3 et al

Platelet count alone cannot be relied upon to assess the severity of PIH. The search for a simple cost effective test for prompt management and prevention of maternal and neonatal morbidity performable in a rural hospital set up still continues

Evaluation of platelet count and its significance in toxemia of pregnancy

T Praveen, Raj Kumar Srivastava, Shirin Jahan, Sanjay Nigam et al

There was significant difference between platelet counts of eclampsia (<0.0001), severe preeclampsia (0.0002), mild preeclampsia (P=0.0004) when compared to control group. Platelet count may be considered as an

early, economical and quick method to estimate the severity of PIH cases. It can also be a useful screening test for early recognition and to assess the prognosis of the disease and outcome in pregnant women.

Howarth S *et al* (1999) conducted a study in US involving 349 normal pregnancies at various gestational stages, and in 30 cases of preeclampsia Platelet count and mean platelet volume (MPV) were estimated. A probability plot was constructed from these data using discriminant analysis of MPV versus platelet count for the preeclamptic versus normal pregnancies. The study found that the sensitivity of MPV was 90%and specificity 83.3% for the prediction of preeclampsia development

A study done in Stanford University northern California stated that platelets appear to play an important role in prediction of preeclampsia. Enhanced platelet activation, as determined by whole blood analyzed using flow cytometry and increased levels of platelet endothelial cell adhesion molecule-1 (PCAM-1) also occur in women who develop preeclampsia as demonstrated by **Taylor *et al*, and Roberts's *et al*,**

The platelet count decreased significantly with the severity of preeclampsia as demonstrated by **Dadhich S *et al*.** (2012) in southern Asia, they also noted that the decrease in platelet count was antedating significant increase in blood pressure by 4 to 6 weeks. As a result, the authors concluded that this Plateleta parameter can be used to predict development of progressive hypertension in at risk patients.

A comparison of platelet count in severe preeclampsia, mild preeclampsia and normal pregnancy

Amit Gupta*, Bindu S. Gaur, K. B. Mishra, Ishan Dubey

The frequency of thrombocytopenia was found to be directly related with the severity of disease, so platelet count can be used as a simple and cost effective tool to monitor the progression of preeclampsia, thereby preventing complications to develop during the gestational period.

Platelet Count In Preeclampsia

Razia Sultana, S. M. Fazlul Karim, Farhana Atia, Shahnila Ferdousi, Selina Ahmed

Among them 50 diagnosed cases of preeclampsia were selected as cases and 50 normal healthy pregnant women as controls. Platelet count was measured in all study subjects. The mean platelet count in cases and controls were 1,44,260±96,472 and 1,98,100±51,219 respectively. The present study showed significant difference of mean platelet count between cases and controls. The study revealed that low platelets count is associated with preeclampsia

Estimation of Platelet Count As A Prognostic Marker for Feto-Maternal Outcome in Preeclampsia & Eclampsia

Purnima Pachori, Dr. Neelam Saini, Dr. Mukesh Kumar Saini,
Dr. Swati

Platelet Count was found to be significantly lower in preeclampsia cases as compared to control group. The feto-maternal outcome was observed to be poor in thrombocytopenic cases as compared to their normal platelet count counterpart subjects.

Abdul Rahim Gari-Bai et al in their study of THROMBOCYTOPENIA DURING PREGNANCY which was a prospective surveillance study of 1357 healthy women, who presented at the end of normal pregnancies in Hamilton, Ontario, Canada, reported thrombocytopenia in 112 (8.3%) of those women.The platelet counts were only very mildly reduced(mean $135x10^{9}/L$).

Keith R. McCrae et al in their study of Thrombocytopenia in Pregnancy showed that Thrombocytopenia affects 6% to 10% of all pregnant women and other than anemia is the most common hematologic disorder in pregnancy. Gestational thrombocytopenia, also known as incidental thrombocytopenia of pregnancy, is the most common cause of thrombocytopenia in pregnant women, accounting for approximately 75% of all cases

Sarah L. Janes et al in their study of Thrombocytopenia in pregnancy showed that Normal pregnancy is generally thought not to affect the platelet count but it has been suggested that the normal range is lower in pregnancy, and that the count falls in the third trimester.

Levy JA et al in their study of Thrombocytopenia in pregnancy showed that Thrombocytopenia is the second most common hematologic abnormality during pregnancy and is usually a benign condition.

Michal Parnas et al in their study of Moderate to severe thrombocytopenia during pregnancy comparing 199 pregnant women with moderate to severe thrombocytopenia (platelet count below 100 _ 109/l) with 201 pregnant women without thrombocytopenia found that the main causes of thrombocytopenia were gestational thrombocytopenia (GT) (59.3%), immune thrombocytopenic purpura (ITP) (11.05%), preeclampsia (10.05%), and HELLP (Hemolysis, elevated liver enzymes and low platelet count) syndrome (12.06%) and concluded as Moderate to severe maternal thrombocytopenia points to a higher degree of severity of the primary disease, which increases perinatal complications. However, the adverse outcome is specifically attributed to preeclampsia, HELLP syndrome, and rare causes, while the perinatal outcome of GT and ITP is basically favorable.

Nadine Ajzenberg et al Pregnancy-Associated Thrombocytopenia Revisited:Assessment and Follow-Up of 50 Cases Whatever the severity of thrombocytopenia, it was found that biological features of an autoimmune disorder in 48% of the women, and chronic thrombocytopenia in 55%. A familial thrombocytopenia was evidenced in 1 case. These 50 women gave birth to 63 neonates, among whom 24 were thrombocytopenic, either at birth or during the first week of life.

Hisanori Minakami et al in their study of gestational thrombocytopenia showed that Although the development of thrombocytopenia in women with pre-eclampsia is well documented, as acknowledged by George, changes in platelet count in an apparently normal pregnancy has not been adequately considered.

Sainio S, et al in their study of Maternal thrombocytopenia at term: a population-based study showed that Women with gestational thrombocytopenia do not require alteration of their treatment. Fetal blood sampling is not considered necessary when thrombocytopenia is discovered unexpectedly at term

Prognostic significance of platelet count in Pregnancy Induced Hypertension Vinod Prabhushetty T1,*, Ramachandra G Latti

The platelet counts were lower in pre-eclampsia and eclampsia as compared to control group (p<0.01)

AETIOPATHOGENESIS

The most common basic abnormalities consistently seen are Incomplete invasion of endovascular cytotrophoblasts into spiral arteries

Endothelial cell dysfunction,

Inflammation acute or chronic

Immunologic intolerance.

I. ABNORMAL PLACENTATION

In normal conception, the spiral arterioles of the uterus, undergo extensive remodeling as they are invaded by endovascular cytotrophoblasts. They invade the deciduas and extend into the walls of the spiral arteriole to replace the endothelium and muscular wall. This remodeling creates a dilated low resistance vessel. The process commences around 10 – 12 weeks of Gestation and is completed by 18-20 weeks of pregnancy. .This trophoblastic invasion extends from the decidua to inner third of the myometrium.

In pre-eclampsia, there is incomplete trophoblastic invasion. With such shallow invasion, only decidual vessels become lined with endovascular trophoblasts. Vessels of myometrium are not involved.The deeper myometrial arterioles do not lose their endothelial lining and musculoelastic tissue and their mean external diameter is only half that of vessels in normal placentas.Thus, it has been clearly dealt that abnormal invasion of this spiral arterioles, decrease the uteroplacental blood flow. Diminished perfusion and a hypoxic environment eventually lead to release of placental debris that initiates a systemic inflammatory response.

Using electron microscopy, Dewolf and co-workers examined arteries at the implantation site, which were found to be occluded by fibrinoid material and there was medial necrosis Lipid accumulated first in myo-intimal cells and then with macrophages. Such lipid laden cells along with the fibrinoid necrosis termed as Atherosis ·Acute atherosis and associated thrombosis are the cause of placental infarctions, which are more common in pre-eclampsia ·

Placental abruption in pre-eclamptic patients probably results from due to thrombosis in the placental vessels, leading to decidual separation and bleeding which could be revealed or concealed.

II. **INFLAMMATION :-**

Pregnancy implies a substantial systemic inflammatory stress on all pregnant women. The two stage model of pre-eclampsia, originally proposed more than a decade, envisages that pre-eclampsia occurs as a result of placental ischemia – reperfusion injury. This may be due to consequence of inadequate placentation

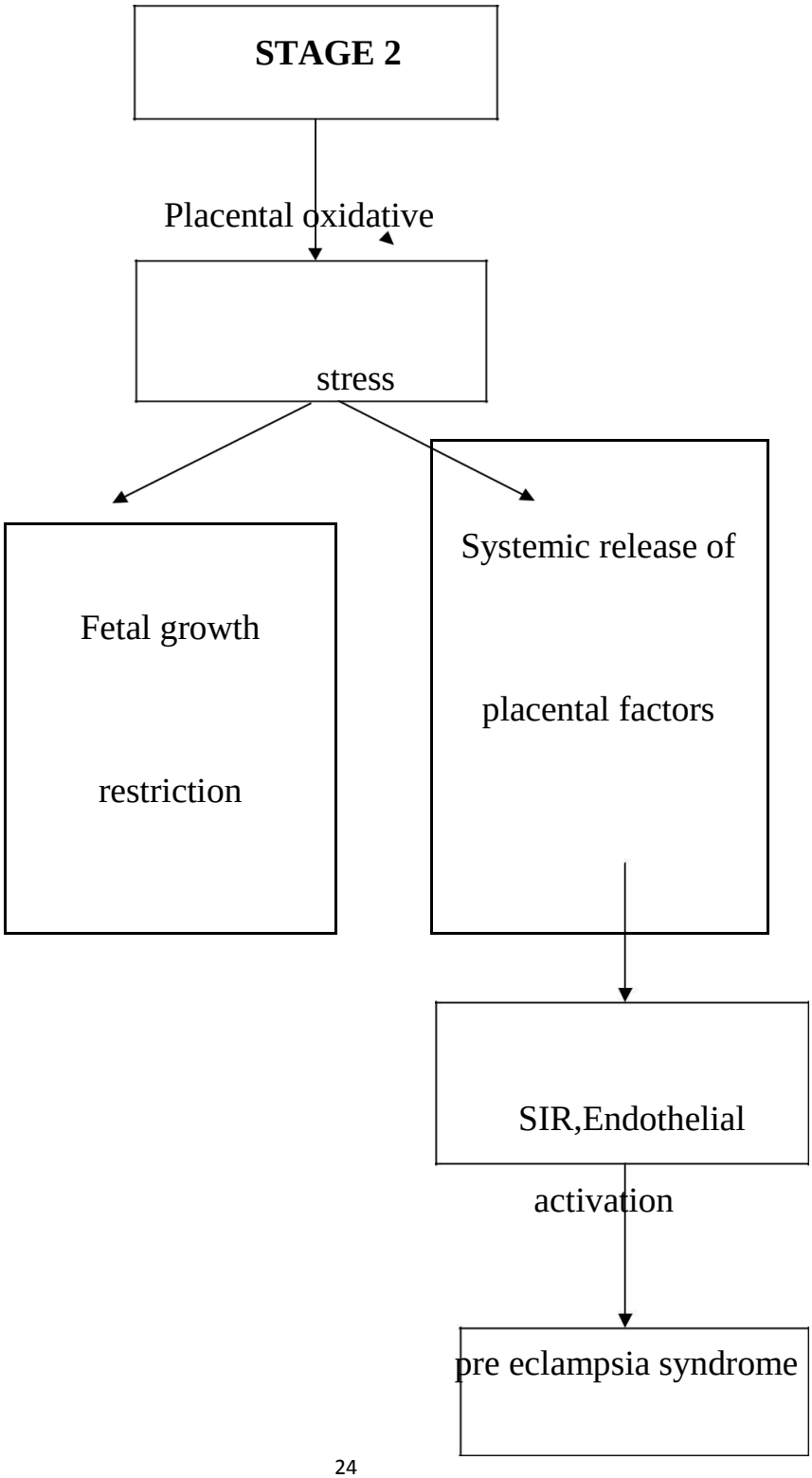
According to Redman and colleagues (2009)

Stage I is preclinical caused by faulty endovascular trophoblastic remodeling, that is followed by stage of vasospasm (stage 2) which manifests clinically.

Stage 2 is caused by release of placental factors into the maternal circulation causing systemic inflammatory response and endothelial activation. Importantly stage 2 is susceptible to modification by pre-existing maternal conditions that include cardiac or renal disease or hereditary influences. Thus pregnancy induced hypertension is a leads to of series of maternal inflammatory responses, exaggerated by the conception itself

STAGE 1

POOR PLACENTATION



III. ENDOTHELIAL CELL DYSFUNCTION

Endothelial cell dysfunction occurs as a complication of an extreme activated state of leukocytes in the maternal circulation. Briefly cytokines such as TNF alpha and Interleukins (IL), may contribute to the oxidative stress associated with pre-eclampsia . This is characterized by Reactive oxygen species and free radicals that leads to the occurence of self-propagating lipid peroxides

The Endothelium is one of the key organs involved in the pathophysiology of pre-eclampsia, as evidenced by the prostacyclin (PGI 2)/ TXA2 imbalance, impairment of the Nitric oxide-cyclic Guanosine monophosphate (No – CGMP) pathway and a series of markers indicating endothelial activation

A key pathophysiological finding in women with pre-eclampsia is generalized intense vasospasm which results in decreased perfusion to virtually all body organs . Although the exact mechanism is not clear, this may be a result of decreased production of endogenous vasodilators such as Nitric Oxide (No)

and prostacyclins, increased generation of vasoconstrictors such as endothelin and thromboxane

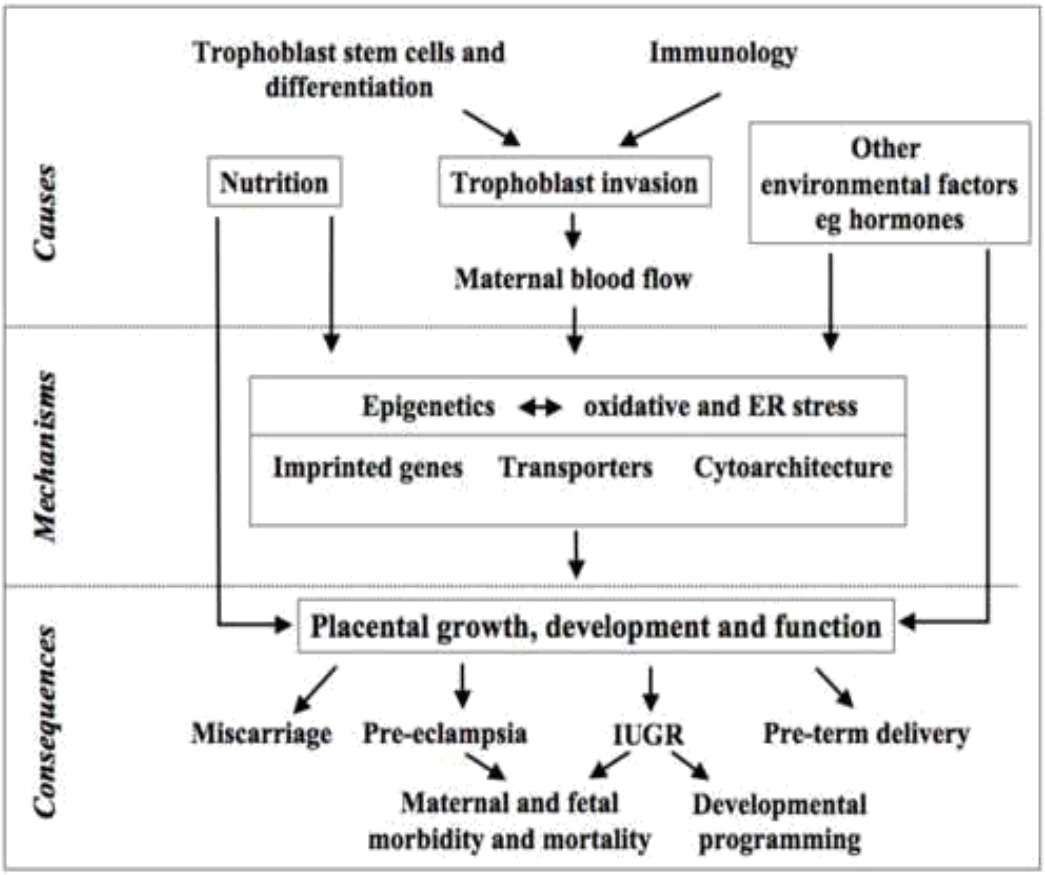
(TXA2) exaggerated response to Angiotensin II

IV. IMMUNOLOGIC INTOLERANCE

The immunologic theory finds support in the observations that pre-eclampsia is most commonly a disease of first conception, increased in primipara and multiparas with a new spouse or undergoing donor insemination and also more common in immuno compromised women. It has been shown that a prior normal pregnancy and even a previous abortion can be considered as a safe measure against this disease.

The placenta being fetal in origin has both maternal and paternal haplotypes and genetic determinants. Of the histocompatibilitity antigens, only HLA-G is expressed on the surface of the trophoblast, the placental cell most intimate to the maternal system. Differences in expression of HLA –G has been noted in trophoblasts from pre-eclampsia than from normal pregnant patients Mediators of the immune maladaptation in pre-

eclampsia include cytokines, particularly tissue necrosis factor (TNF – alpha) and interleukins IL – 2 and Interleukins IL-6



PATHOLOGY OF PLACENTA AND UTERUS

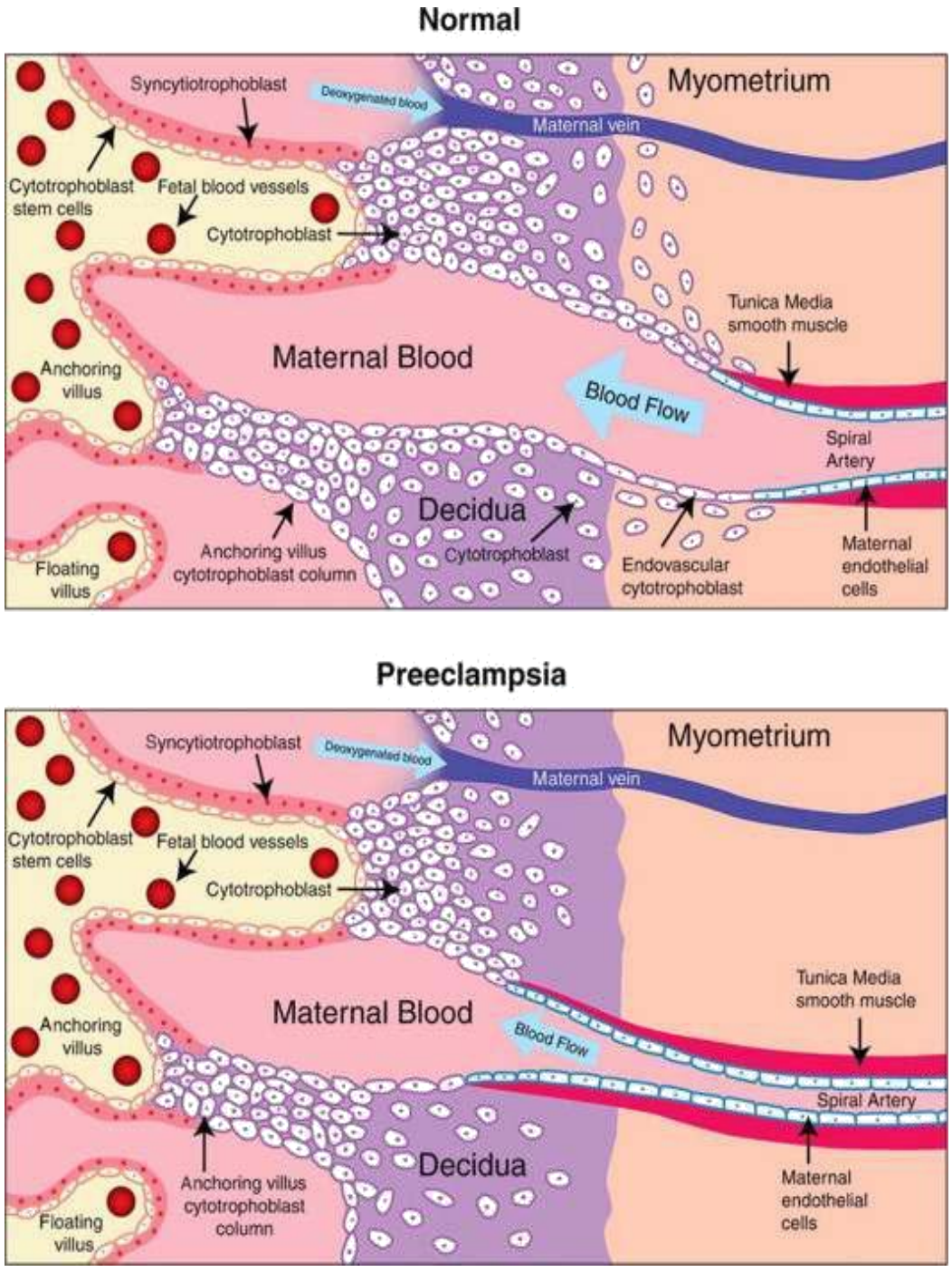
Comparisons of placenta from normal and hypertensive pregnancies have shown an increased incidence of infarcts, haematomas, congested chorionic villi, proliferative end-arteritis and degeneration in the hypertensive group. The degree of these changes is roughly proportional to the clinical severity of the disease. Microscopy reveals increased synctial knots, cytotrophoblastic cellular proliferatioin, fibrinoid necrosis endothelial proliferation and calcified and hyalinised villous spots

UTEROPLACENIAL VASCULATURE

One of the remarkable features of human placental development is the extensive modification of maternal vasculature by trophoblasts. These events occur in the first half of pregnancy. Modifications of spiral arteries are carried out by two populations of extravillous trophoblasts - interstitial trophoblasts, which surrounds the arteries and endovascular trophoblasts, which penetrates the spiral artery lumen [26].

The interstitial trophoblasts are now recognized to constitute a major portion of the placental bed, penetrating the decidua and adjacent

myometrium. They aggregate around spiral arteries and their functions may include vessel preparation for endovascular trophoblast invasion



The endovascular trophoblast enters the lumen of the spiral arteries and initially forms cellular plugs. It then destroys vascular endothelium via an apoptosis mechanism, invades, modifies vascular media. Thus fibrinoid material replaces smooth muscle and connective tissue of the vessel media. Spiral arteries later regenerate endothelium. Invading endovascular trophoblast can extend several centimeters along the vessel lumen and they must migrate against arterial flow. Invasion by trophoblasts involves only the decidual arteries, not the decidual veins

In their summary of anatomical studies of the uteroplacental vasculature, Ramsey and Donner (1980), said that these development proceeds in two waves or stages[28].

The first wave occurs before 12 weeks post-fertilisation and consists of invasion and modification of spiral arteries limited to the borders between decidua and myometrium.

The second wave is between 12 and 16 weeks and involves some invasion of the intra-myometrial segments of spiral arteries. The

remodeling by two waves converts narrow-lumen, muscular spiral arteries into dilated, low resistance uteroplacental vessels.

Molecular mechanisms of these crucial events should be learnt, since they have an important role in the aetio pathogenesis of pregnancy induced hypertensive disorders and IUGR.

Platelet are non-nucleated cellular fragments of megakaryocytes, they play a key role in hemostasis.(1) Thrombocytopenia is defined as a platelet count 150 x10^9/l, caused by accelerated platelet destruction or decreased production. The normal reference range of platelets in non-pregnant women is 150 -400*10^9/l. Due to hemodilution of plasma volume, platelet count may decrease by approximately 6%-7% occurs during 3rd trimester,though absolute platelet count remains within normal reference range in most patients.Thrombocytopenia can be classified as

mild thrombocytopenia100–150x 10^9/l

moderate thrombocytopenia 50–100 x 10^9/l

severe less than 50 x 10^9/l

In pregnancy, most cases are due to gestational thrombocytopenia, idiopathic purpura or pre-eclampsia. Other causes include such as malaria, folate deficiency, leukaemia and aplastic anemia.

Gestational thrombocytopenia(GT) is considered as the most common cause of thrombocytopenia, accounts for 75% of thrombocytopenia in pregnancy.

Its characterised by incidental identification of mild to moderate reduction in platelet count during pregnancy in healthy women with no previous history of idiopathic thrombocytopenia purpura or conditions known to be associated with thrombocytopenia. Its not an early manifestation of autoimmune disease, there is no significant fetal or maternal morbidity and normalisation of platelet count occur in vast majority of patients post partum. The incidence of thrombocytopenia in neonates born to GT patients is similar to those reported in non-GT women.Though the pathogenesis of gestational thrombocytopenia is not well understood, it may involve factors such as haemodilution or accelerated platelet clearance. Confirmation of normal platelet count

prior to pregnancy decreases the probability of underlying immune thrombocytopenia purpura

Pregnant women with thrombocytopenia have higher risk of bleeding excessively during and after childbirth, particularly they need to have caesarean section or other surgical intervention during pregnancy, labor, puerperium. Such bleeding complications are likely when platelet count is less than 150 $x10^9$/l.

Hematological abnormalities develop as a complication of preeclampsia. Out of all hematological changes that occur in preeclampsia and eclampsia, thrombocytopenia is most common hematological abnormality found followed by anemia. Thrombocytopenia is defined as platelet count less than 150 X 109 /L. Though in normal pregnancy there is no effect on platelet count, studies show that platelet counts might fall to some extent but not lesser than the normal range. This usually occurs during the third trimester and reverts back to normal immediately following delivery. Changes in platelet count is well established in preeclampsia and studies show, with evolution of severe preeclampsia there was a fall in circulating platelet count much earlier than expected.

Thrombocytopenia is attributed to two main causes. Failure of platelet production and early excessive platelet consumption, where the second cause is observed in eclampsia and pre eclampsia. Accelerated platelet activation occurs normally in pregnancy that explains the mild thrombocytopenia seen in normal patients and is termed as pregnancy associated thrombocytopenia or incidental thrombocytopenia. The incidence is 0.4 to 8.3%. In these cases the fetus is not at risk as perinatal outcome is quite satisfactory.

In preeclampsia, thrombocytopenia may occur without other evidence of coagulation disturbances. It probably occurs as a result of immunologically mediated process or more likely due to increased platelet deposition at the site of endothelial damage and activation of coagulation system in small vessels. Thus, there is an increased platelet activation and consumption and simultaneous increase in platelet production. Thus platelet life span is reduced and activation increased as evidenced by raise in beta thromboglobulin levels. It reflects the severity of the pathological process. More severe the thrombocytopenia, greater the maternal and fetal morbidity and mortality. After delivery, the platelet count will progressively increase and return to normal. Thus the treatment for progressive preeclampsia

including thrombocytopenia is immediate delivery irrespective of gestational age.

The mechanism of thrombocytopenia in pre eclampsia is variously explained as under:

- It may be due to increased consumption of platelets with increased megakaryocytic activity to compensate it. Platelets adhere to areas of damaged vascular endothelium resulting in secondary destruction of platelets (O'Brien et al).
- Prostacyclin is an important eicosanoid that exerts strong inhibition of platelet aggregation. There is continuous availability of this eicosanoid from blood vessels which keeps circulating platelets in a dispersed and disaggregated form (O'Brien et al).Deprivation of this prostacyclin makes the circulating platelets even more vulnerable to aggregation. Removal of aggregated platelets might be responsible for thrombocytopenia often observed in pregnancy induced hypertension (FitzGerald et al)
- Platelets from severely preeclamptic patients showed less response than normal to a variety of aggregating agents suggesting that platelets may have undergone previous aggregation in the microcirculation

Recent studies have documented that increased plasma levels of sFlt1-soluble vascular endothelial cell growth factor (VEGF) receptor type 1 as well as endoglin, an endothelial cell-derived member of the tumor growth factor-2 (TGF-2) receptor family (Venkatesha et al), are present in patients intended to develop preeclampsia as early as the late first trimester.30 Increased levels of soluble fms-like tyrosine kinase-1 (sFlt1) and endoglin mRNA is present in preeclamptic placentae, suggesting this is the source of these proteins (Kita et al).31 sFlt1 binds and neutralizes VEGF and placental growth factor (PLGF), another important VEGF and placental growth factor (PLGF), another important VEGF family member whose levels normally increase during pregnancy, whereas endoglin blocks the binding of TGF-2 to endothelial cells. (Young et al).32 These types of pregnancies are also associated with qualitative alterations suggesting increased platelet turnover.

PREVENTION OF PRE ECLAMPSIA

A variety of strategies used to prevent / modify the severity of preeclampsia.

METHODS PROPOSED TO PREVENT PREECLAMPSIA

1. Diet and exercise
2. Salt restriction
3. Fish oil supplementation
4. Heparin
5. Low dose aspirin
6. Calcium supplementation
7. Anti oxidants
8. Anti Hypertensive medication in chronic hypertension

RESULTS

Table 1: Age distribution of study participants

Age (Years)	Frequency	Percent
16-20	53	17.7
21-30	210	70
31-40	37	12.3
Total	300	100

The study was conducted in 300 female study participants among them majority were in the age group of 21 years to 30 years (70%). Their mean age was 24.96 $\pm$ 4.59 years.

Table 2: Association of age of patients with status of Eclampsia

Age	Nil	GHT	Mild preeclampsia	Eclampsia	Severe Eclampsia	Total	Chi sq	p
16-20	26	10	7	2	8	53	1.87	0.9
21-30	105	41	34	7	23	210		
31-40	19	9	5	1	3	37		
Total	150	60	46	10	34	300		

Graph 1: Association of age of patients with status of Eclampsia

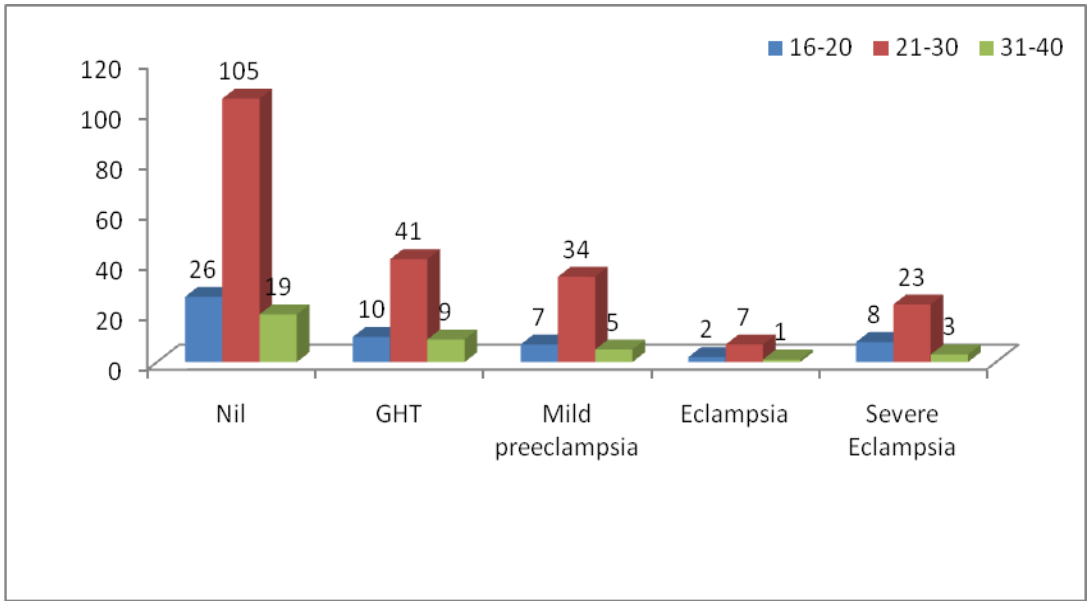
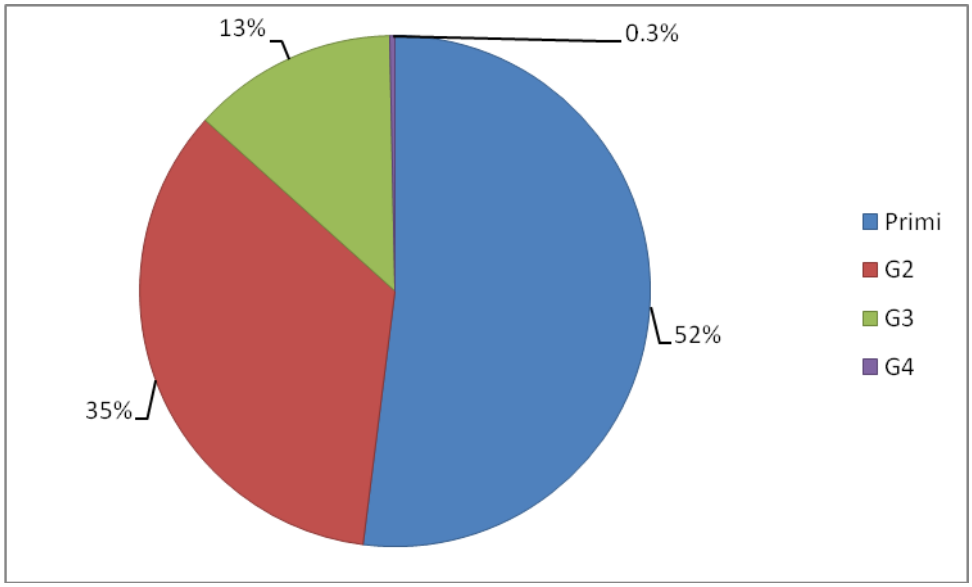
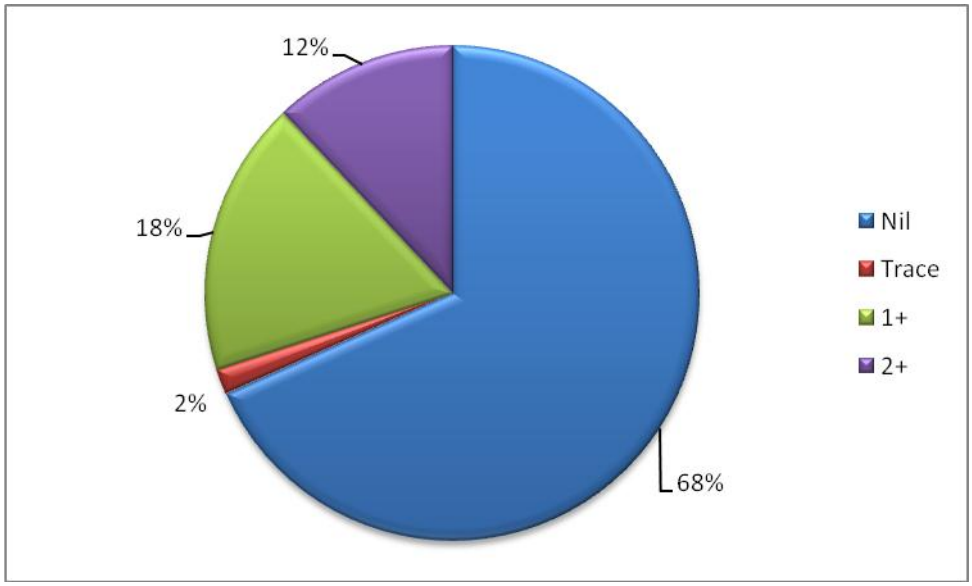


Table 2 explains the association of age of the participants with stages of Eclampsia, 34 female had severe Eclampsia and 10 had Eclampsia 46 had mild preeclampsia which was found statistically not significant (p=0.9)

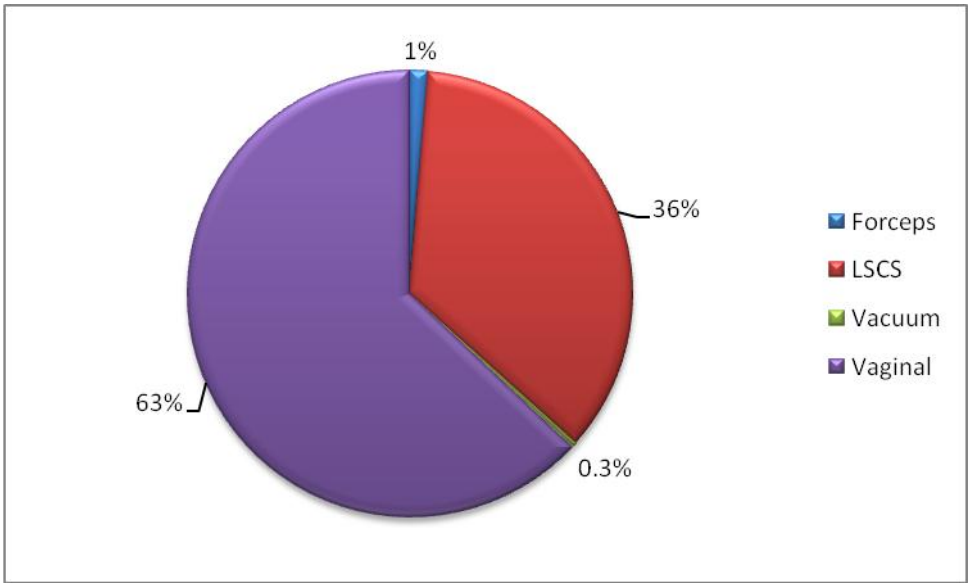
Graph 2: Distribution of Parity



Graph 3: Distribution of Urine Analysis



Graph 4: Distribution of Mode of delivery



Graph 2 explains the distribution of parity 52% were in Primi where 48% had atleast a child. Graph 3 shows that nearly 32% had trace and counts in urine analysis. Graph 4 describes mode of delivery 63% had normal delivery 36% had LSCS and others were had vacuum and forceps delivery.

Table 3: Comparison of Parity with Outcome of labour

outcome	Primi	Multigravida	Total	Chi sq	p
Term	113	121	234	6.03	0.06
Preterm	40	22	62		
SB/IUD	3	1	4		
Total	156	144	300		

Table 3 shows that majority that is 234 had term delivery, 62 had pre term and 4 still births which was not statistically significant (p = 0.06) between parity and mode of delivery

Table 4: Comparison of Parity with coagulopathy and PPH among study participants

complications	Primi	Multigravida	Total	Chi sq	p
Nil	141	124	265	1.8	0.4
PPH	10	11	21		
coagulopathy	5	9	14		
Total	156	144	300		

Table 4 describes that 14 females had coagulopathy and 21 had PPH which was not signifcantly different due to parity (p=0.4)

Table 5: Distribution of platelet counts in study participants

Platelets	Frequency	Percentage
<1 lk	15	5
1-1.5lk	27	9
1.5-2lk	62	20.7
>2lk	196	65.3
Total	300	100

Table 5 explains that 65.3% had more than two lakh platelet counts, 20.7% had between 1.5 to 2 lakhs and nearly 14% had lesser counts showed in graph 5.

Graph 5: Distribution of Platelet counts of female participants

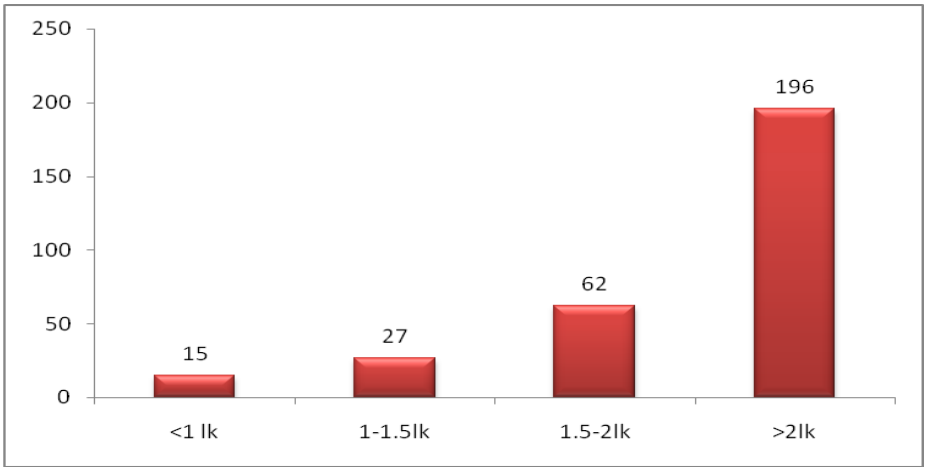


Table 6: Distribution of stages of Eclampsia among mothers

Eclampsia	Frequency	Percent
Nil	150	50
GHT	60	20
Mild Pre	46	15.3
Eclampsia	10	3.3
Severe	34	11.3
Total	300	100

Eclampsia	Platelet counts Mean ± SD
Nil	247000 ± 21417.86
GHT	158000 ± 23301.03
Mild Pre	188000 ± 39099.16
Eclampsia	95700 ± 19505.27
Severe	130000 ± 51019.03
Total	222000 ± 59867.95

Table 6 explains that 11.3% had severe eclampsia 3.3% had Eclampsia and 15.3% had pre eclampsia and 20% had Gestational hypertension; depict in graph 6.

Graph 6: Distribution of stages of Eclampsia among mothers

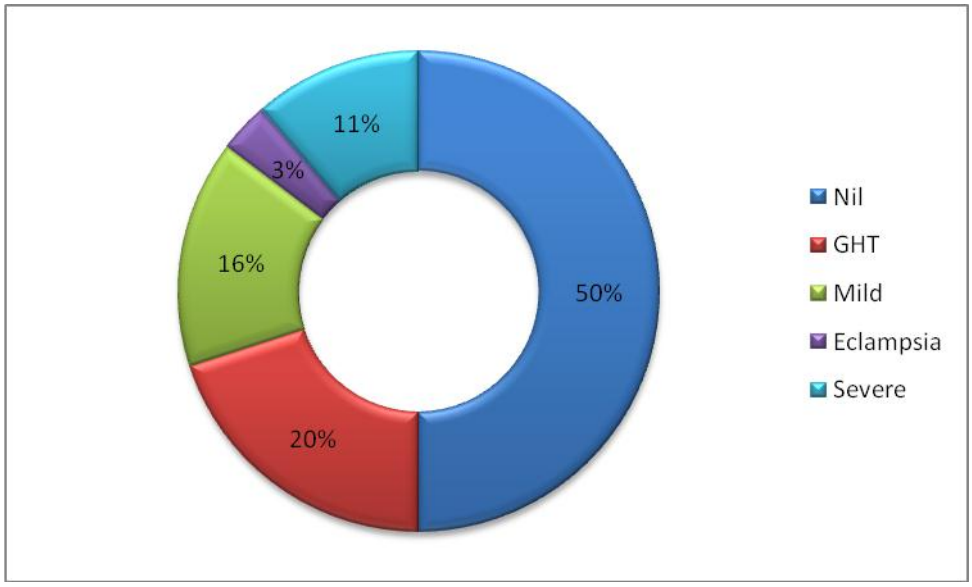


Table 7: Distribution of fetal outcomes

FETALOUTCOME	Frequency	Percent
IUD	2	0.7
Preterm	62	20
Stillbirth	2	0.7
Term	234	78
Total	300	100

Table 7 explains that 78% had term delivery 20 % had pre term delivery and remaing 2% were still born and intrauterine deaths which showed in graph 7.

Graph7: Distribution of fetal outcomes

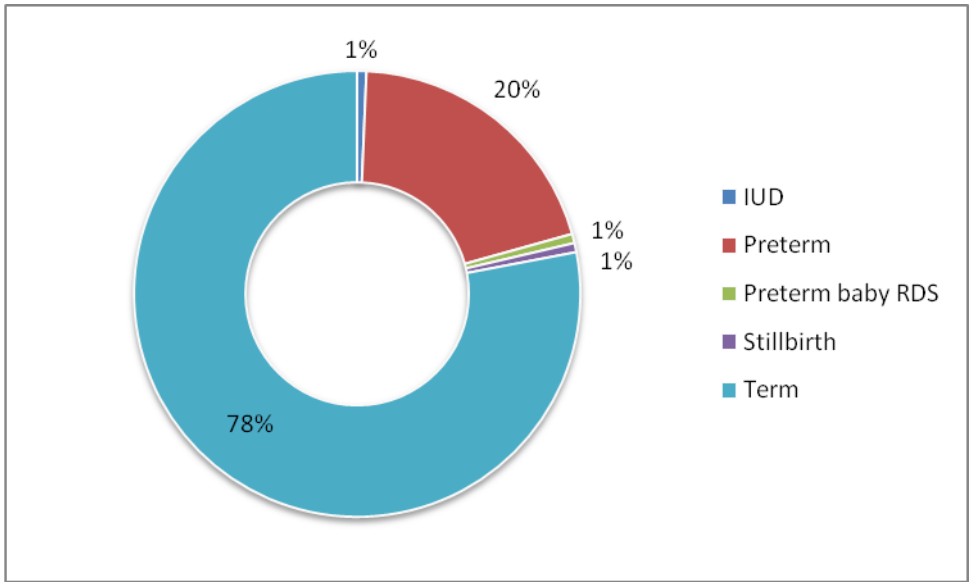


Table 8: Distribution of complications

	Frequency	Percent
HELLP SYNDROME	14	4.7
NO	265	88.3
PPH	21	7
Total	300	100

Table 8 explains that distribution of complications among study mothers among them 7% had PPH and 4.7% had Hellp syndrome which pictorially represented in graph 8.

Graph8: Distribution of complications

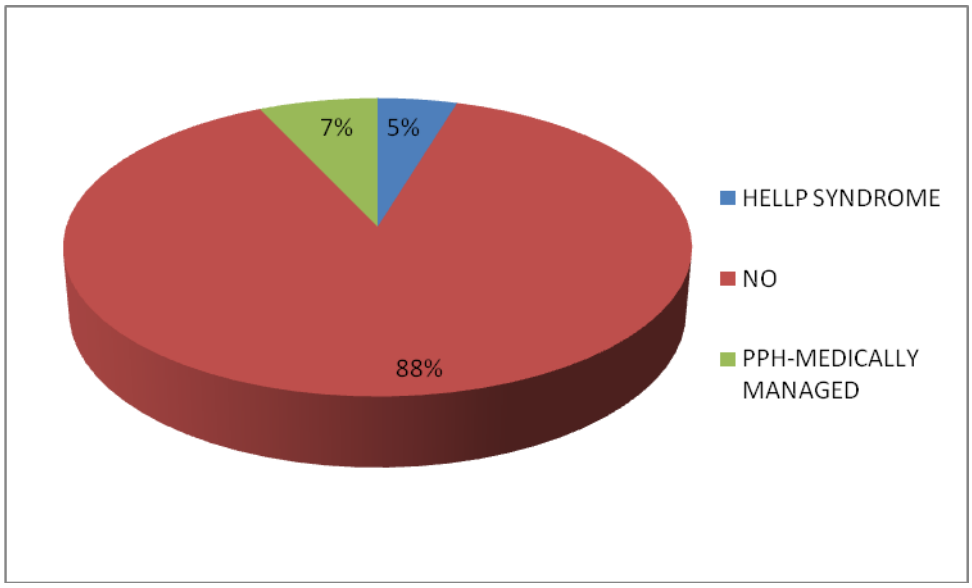


Table 9: Comparison of Parity with platelet counts of study participants

Platelets	Primi	Multigravida	Total	Chi sq	P
<1 lk	10	5	15		
1-1.5lk	19	8	27		
1.5-2lk	16	46	62	23.67	**0.001**
>2lk	111	85	196		
Total	156	144	300		

Table 9 describes majority of the mothers had more than two lakh platelet count and they were in primi in other hand in multigravida mothers had platelet counts between 1.5 to 2 lakhs which showed statistically significant ($p = 0.001$) (Graph 9)

Graph 9: Comparison of Parity with platelet counts of study participants

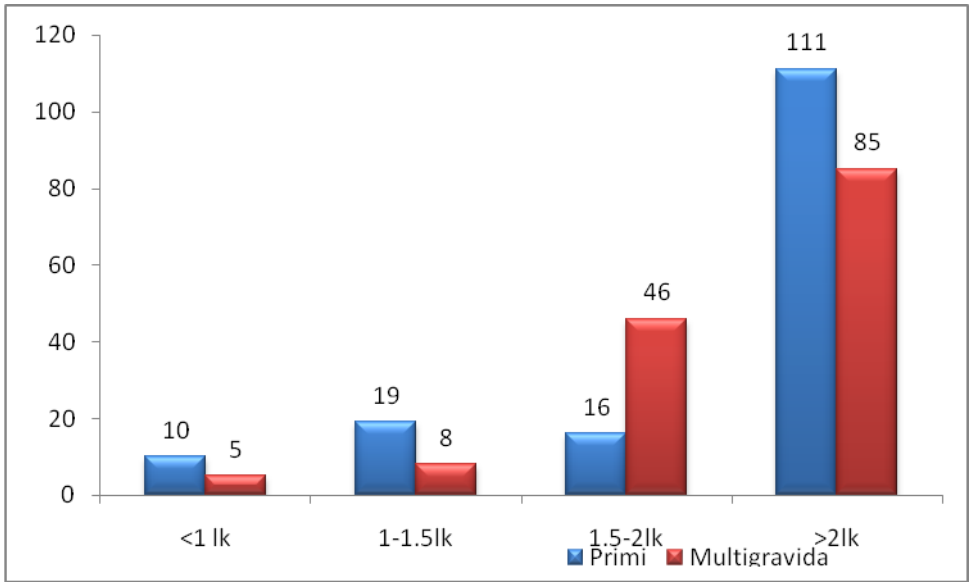


Table 10: Comparison of Parity with Stages of Eclampsia

preeclampsia	Primi	Multigravida	Total	Chi sq	p
Nil	89	61	150	14.81	**0.005**
GHT	19	41	60		
Mild	22	24	46		
Eclampsia	5	5	10		
Severe	21	13	34		
Total	156	144	300		

Table 10 explains that multi gravida mothers had more chance of having gestational hypertension and mild preeclampsia on the other hand primi mothers had more prevelance of having severe eclampsia which proven statistical significance. (p =0.005) (Graph 10)

Graph 10: Comparison of Parity with Stages of Eclampsia

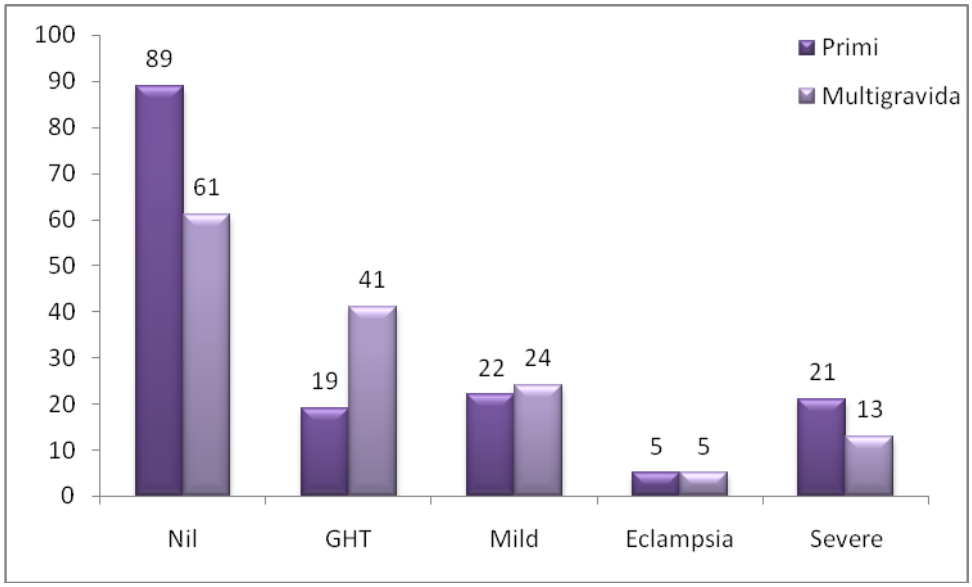


Table 11: Comparison of complications with Thrombocytopenea

Tccodes	<1 lk	1-1.5lk	1.5-2lk	>2lk	Total	Chi sq	P
Nil	15	19	58	173	265	13.13	**0.04**
PPH	0	4	3	14	21		
coagulopathy	0	4	1	9	14		
Total	15	27	62	196	300		

Table 11 explains that there was significant association between complications with platelet counts of mothers which statistically significant with p = 0.04 and it showed in graph 11.

Graph 11: Comparison of complications with Thrombocytopenia

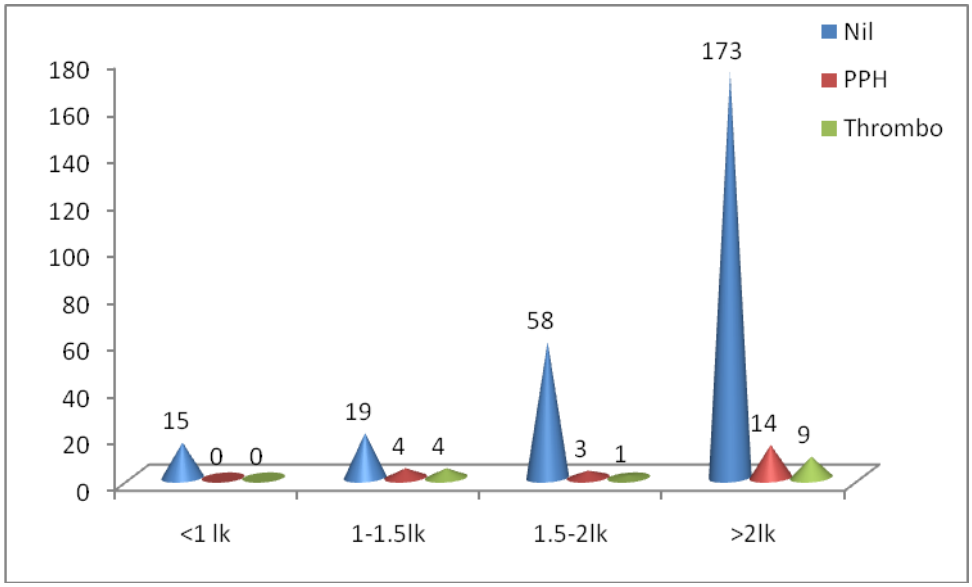


Table 12: Comparison of Platelet counts with outcome of delivery

outcome	<1 lk	1-1.5lk	1.5-2lk	>2lk	Total	Chi sq	P
Term	8	19	48	159	234	6.32	**0.01**
Preterm	6	8	14	34	62		
SB/IUD	1	0	0	3	4		
Total	15	27	62	196	300		

Table 12 describes that there was significant association between outcome of delivery with platelet counts of mothers with p = 0.01. it explains that if mother had lesser platelet counts had more chance of having pre term delivery. (Graph 12)

Graph 12: Comparison of Platelet counts with outcome of delivery

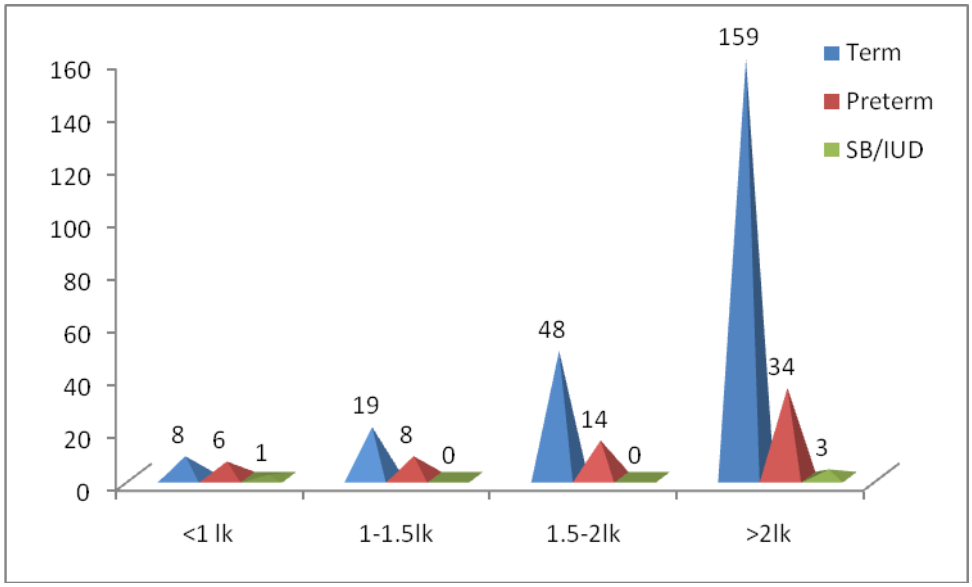


Table 13: Comparison of Platelet counts with stages of Eclampsia

Platelets	Nil	GHT	Mild preeclampsia	Eclampsia	Severe Eclampsia	Chi sq	P
<1 lk	0	0	0	0	15	10.5	**0.001**
1-1.5lk	0	11	0	1	12		
1.5-2lk	0	34	26	3	2		
>2lk	150	15	20	6	5		
Total	150	60	46	10	34		

Table 13 describes that there were significant differnce in stages of eclampsia due to the platelet counts with significant value p = 0.001. patient with lesser than 1.5 lakhs of counts having more chance of being severe eclampsia than others. Similarly patients with 1.5 to 2 lakhs of platelet counts having chance to develop mild pre eclampsia or gestational hypertension. These were represented in graph 13.

Graph 13: Comparison of Platelet counts with stages of Eclampsia

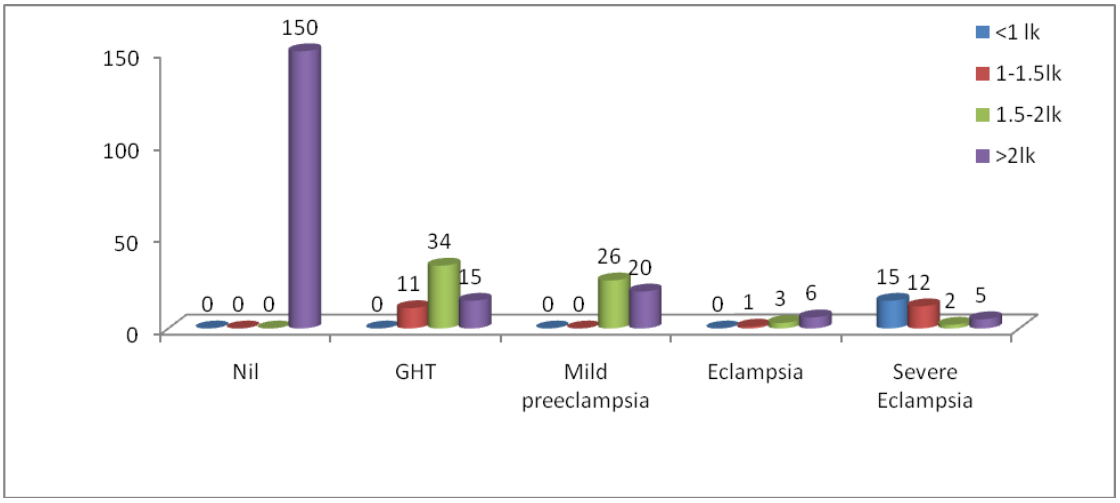


Table 14: Comparison of Complications with stages of Eclampsia

Tccodes	Nil	GHT	Mild preeclampsia	Eclampsia	Severe Eclampsia	Chi sq	p
Nil	138	50	38	2	20	4.46	**0.03**
PPH	12	10	8	0	8		
HELLP	0	0	0	8	6		
Total	150	60	46	10	34		

Table 14 describes that mothers with eclampsia or severe preclampsia develop coagulopathy. Similarly patients with PPH having more chance of being GHT and mild pre eclampsia patients which were statistically significant with p = 0.03. (Graph 14)

Graph 14: Comparison of Coagulopathy with stages of Eclampsia

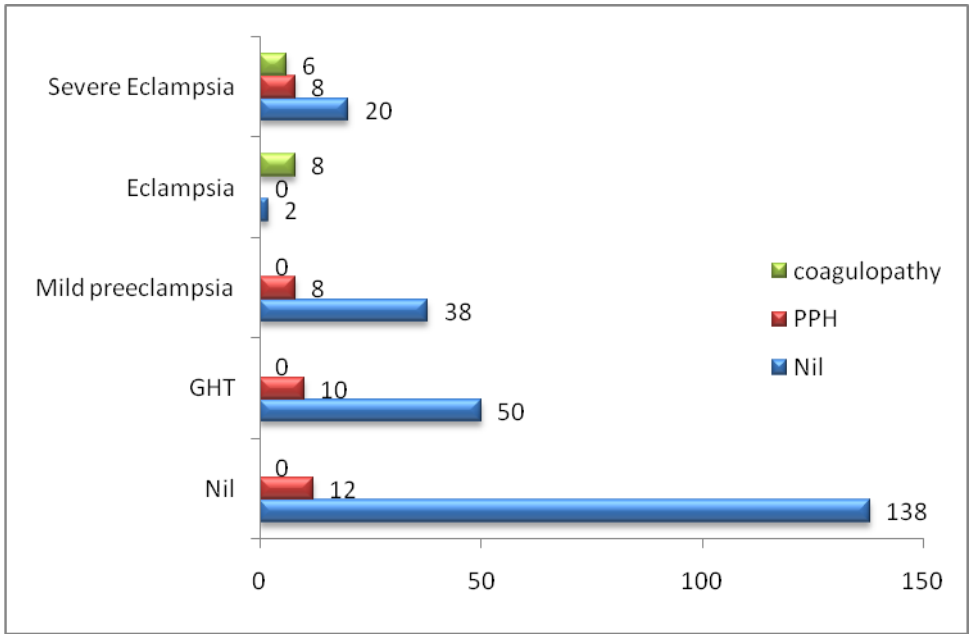


Table 15: Comparison of outcome of delivery with stages of Eclampsia

outcome	Nil	GHT	Mild preeclampsia	Eclampsia	Severe Eclampsia	Total	Chi sq	P
Term	138	54	33	1	8	234	96.14	**0.0001**
Preterm	9	6	13	8	26	62		
SB/IUD	2	0	1	1	0	4		
Total	150	60	46	10	34	300		

Table 15 describes that there was significant assocation between outcome of delivery and stages of eclampsia with p = 0.0001. it explains that pre term delivery having significant association with severe eclampsia and eclampsia where in GHT and Mild pre eclampsia patients went in to term delivery. (Graph 15)

Graph 15: Comparison of outcome of delivery with stages of Eclampsia

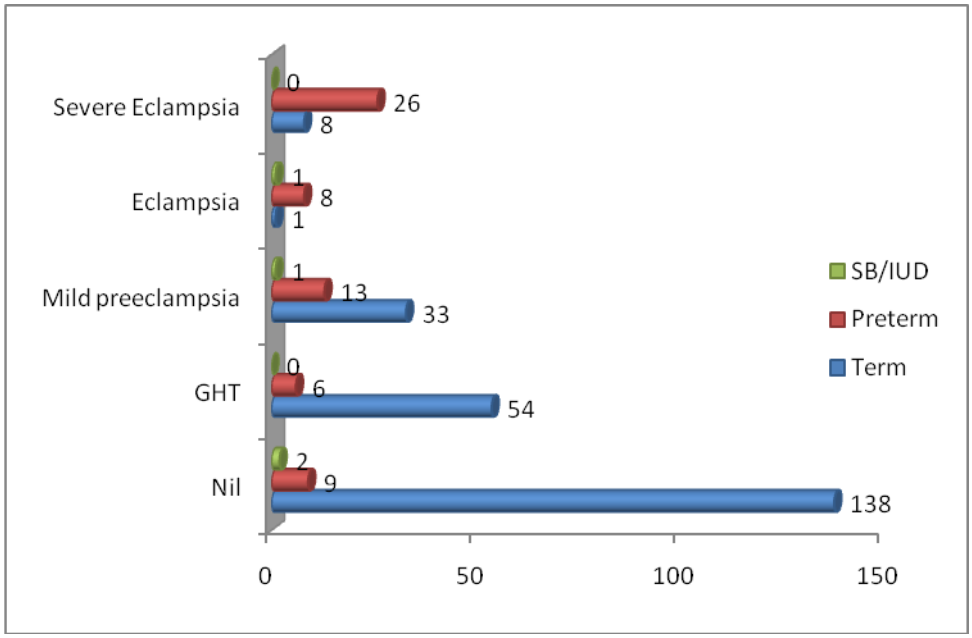


Table 16: Comparison of Mode of delivery with stages of Eclampsia

Eclampsia	Vaginal	Forceps/vacuum	LSCS	Total	Chi sq	p
Nil	115	0	35	150	37.51	**0.0001**
GHT	40	1	19	60		
Mild	21	1	24	46		
Eclampsia	3	1	6	10		
Severe	10	2	22	34		
Total	189	5	106	300		

Table 16 explains that there was significant assocation between mode of delivery and stages of eclampsia with p = 0.0001. it explains that LSCS having significant association with severe eclampsia and eclampsia. (Graph 16)

Graph 16: Comparison of Mode of delivery with stages of Eclampsia

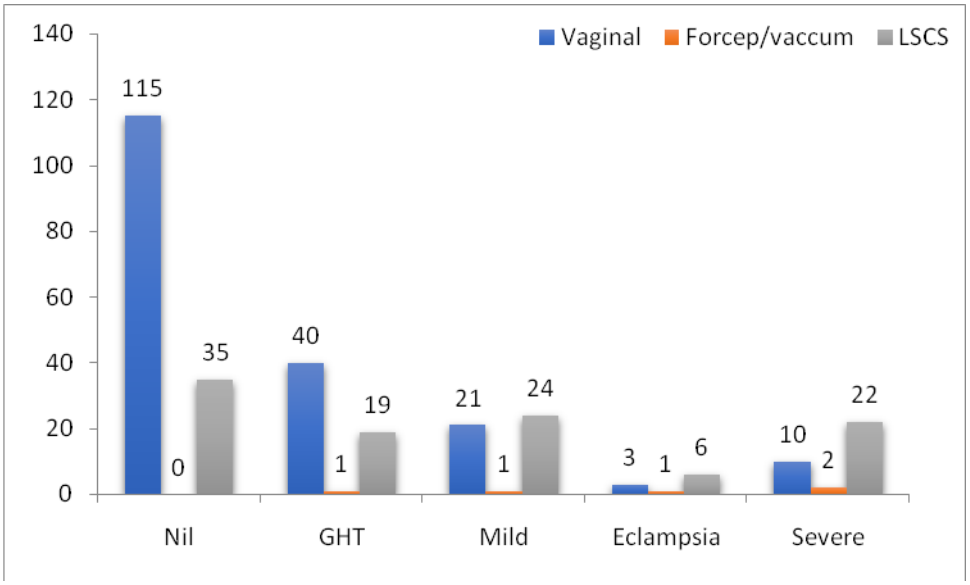


Table 17: Comparison of Mode of delivery with Platelet counts

Platelets	Vaginal	Forceps/vacuum	LSCS	Total	Chi sq	p
<1 lk	6	0	9	15		
1-1.5lk	6	5	16	27		
1.5-2lk	28	0	34	62	31.28	**0.0001**
>2lk	149	0	47	196		
Total	189	5	106	300		

Table 17 describes that there was significant assocation between mode of delivery and platelet counts with p = 0.0001. (Graph 17)

Graph 17: Comparison of Mode of delivery with Platelet counts

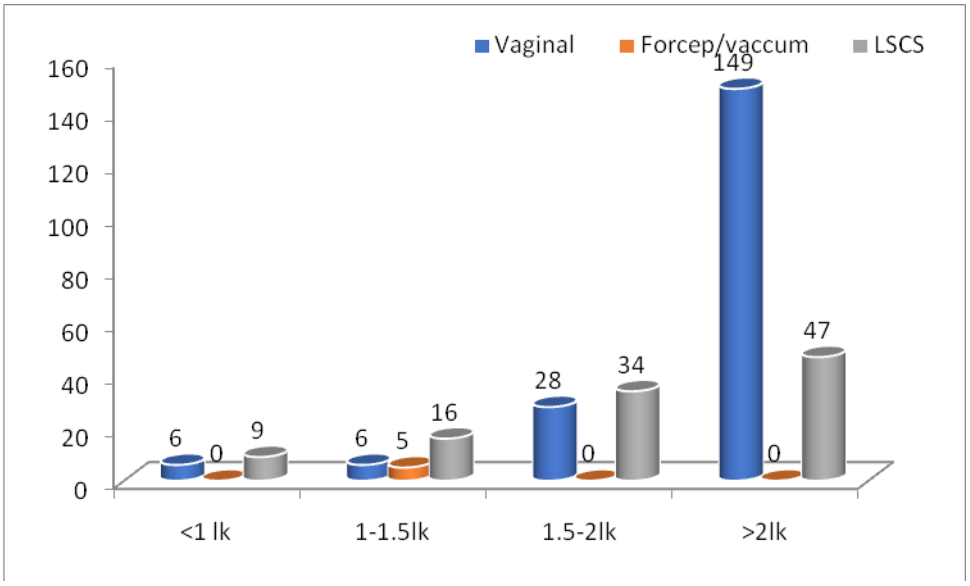
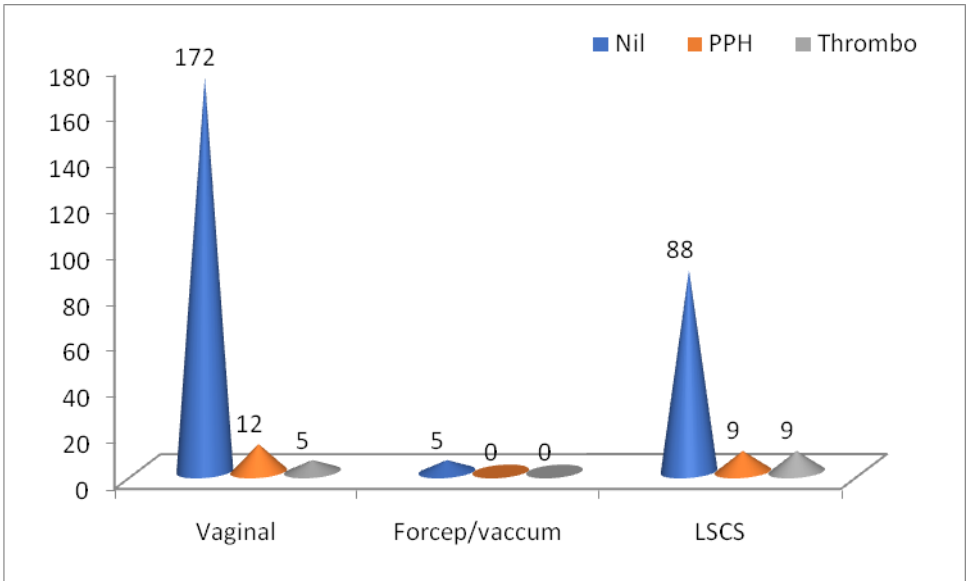


Table 18: Comparison of Mode of delivery with Thrombocytopenia

complications	Vaginal	Forceps/vacuum	LSCS	Total	Chi sq	p
nil	172	5	88	265	5.55	**0.02**
PPH	12	0	9	21		
coagulopathy	5	0	9	14		
total	189	5	106	300		

Table 18 shows that there was significant assocation between mode of delivery and complications with p = 0.0001. (Graph 18)

Graph 18: Comparison of Mode of delivery with complications



DISCUSSION

The current study was conducted in 300 female study participants among them majority were in the age group of 21 years to 30 years (70%). Their mean age was 24.96 $\pm$ 4.59 years. Sameer et al study had similar mean age 24.41 years and Purnima et al and Guptha et al had mean age 26.26 + 4.5 years. In this study the association of age of the participants with stages of was found statistically not significant.

The prevalance of gestational hypertension is 60(40 %) .mild preclampsia is 46.(30%)The prevalance of eclampsia and severe eclampsia were 10 (3%) and 34 (11%) respectively. The prevalance of HELLP was 14 (4.5%) and PPH was 21 (7%).

The prevelance of platelet count lesser than 1.5 lakh were 42 (14%) and 1.5 – 2lakhs were 62 (21%)

Majority of the study participants 234 (78%) had term delivery, 62 (21%) had pre term and 4 (1%) still births which was not statistically significant which was coincided with study by sameer etal where they found

78% had term delivery 20 % had pre term delivery and remaing 2% were still born and intrauterine deaths.

This study showed that pre term delivery having significant association with Primi whereas in multigravida associated more with term delivery.

The study proved that pre term delivery having significant association with severe eclampsia and eclampsia whereas in GHT and Mild pre eclampsia patients undergone term delivery.

In our study the distribution of parity 52% were in Primi where 48% had atleast a child which was almost similar to sameer et al 66.5% primi and 33.5% were multigravida.

This study showed multi gravida mothers had gestational hypertension and mild preeclampsia on the other hand primi mothers more prone for severe eclampsia which was almost similar with study done by purnima etal and sameer et al

In present study 15.3% mild preeclampsia, 3.3 % Eclampsia 11.3% had severe Eclampsia which was almost similar to sameer etal. Majority of patients 79% with lesser than 1 to 1.5 lakh platelet counts were had severe eclampsia. 56% of mild preeclampsia patients have platelet count 1.5 – 2 lakhs which were correlating with study done by kumar et al.

In study done by feroza et al shameem et al cases belonged to the mild preeclampsia (56%) group followed by cases with severe preeclampsia (30%).

In this study the mean platelet counts were 158000 ± 23301.03 in gestational hypertension, 188000 ± 39099.16 in mild preeclampsia, 95700 ± 19505.27 in eclampsia.

The study done by sameer et al. Platelet count in mild preeclampsia was 2.39± 0.61 and in severe preeclampsia and eclampsia was 1.60± 0.51 lakhs/cumm.

Jaremo P. *et al*., 2000 mentioned in their study [10]. Srivastava. *et al*., (1995) reported mean platelet count of 1.94 lakh/cumm in normal pregnant control, 1.79 lakh/cumm in mild preeclampsia, & significantly low platelet count in severe preeclampsia i.e. 1.64 lakh/cumm and in eclampsia i.e. 1.52 lakh/cumm [11].

Kulkarni and Sutaria, *et al*., in their study observed platelet count as follows, in mild Preeclampsia 1.84lacs/cumm, in severe preeclampsia 1.94lacs/cumm, in eclampsia 1.18 lacs/cumm and in control 2.5lacs/cu mm respectively with these results, mentioned platelet count reduces as the severity of disease increases with significant difference between each group [8].

Guptha et al found that 72.4% of the patients in the mild preeclampsia group had the platelet count more than 2 lakh/mm3. 41% patients with severe preeclampsia had the platelet count in the range of 1-1.5 lakh/mm3 and 64.7% eclampsia cases had platelet count between 1-1.5 lakh/mm3.

Ostin et al had 259.1± 73.9 in mild preeclampsia, 262.6± 86.5 in moderate and 159.6 ± 90.8 in severe eclampsia which was statistically significant.

Joshikale vrunda saple shaila et al found that 21.8% of severe preeclampsia and 39.3% of eclampsia patients with platelet count less than l lakh Sibai et al found 30% eclamptic patients have platelet count less than 1 lakh

In our study 50% of severe preclampsia patients have platelet count less than 1 lakh.

Patients with platelet count 1.5 and lesser had undergone caesarean section than other type of delivery which was statistically significant. Similarly the mothers who had eclampsia and severe preeclampsia had more caesarean section delivery which also significant.

kumar et al found 41% had PPH and HELLP syndrome in severe eclampsia which explains that derangement in coagulation profile would have significantly associated with fetal morbidity.

The present study explained that the patients who had platelet count lesser than 1.5 lakhs associated with risk of having pre term delivery and caesarean section would be mode of delivery. They had highly correlation between PPH and thrombocytopenia

3 Our finding of a trend of lowering of platelet count with increasing severity of pregnancy induced hypertension is consistent with Srivastava , Jambhulkar (2001)[], Joshi et al (2004) J.Davies et al (2007)[15] and Ellora Devi et al (2012)

Giles C and Inglis TC., also observed significant difference in between each group, platelet count reduces with severity of disease .Agarwal and baradkar., and Dube *et al*., in their studies mentioned platelet count is reduced significantly and it is correlated with severity of disease . In the study of Vrunda *et al*., 2004 mentioned severity of disease and thrombocytopenia closely correlated, which indicates that thrombocytopenia is directly proportional to the severity of toxemia of pregnancy.

Leduc et al reported significant association between thrombocytopenia and maternal complications and reported that platelet nadir is the best predictor of maternal outcome. Savita et al (2009)[19] reported higher incidence of neonatal complications in patients with preeclampsia and thrombocytopenia. Our findings regarding the relation of the deranged coagulation profile and maternal and fetal outcome are consistent with all the studies mentioned above.

CONCLUSION

The hypertensive diseases complicating pregnancy still remains the major problem in developing countries. The fact that pregnancy induced hypertension is largely a preventable condition is established by observing the negligible incidence of pre-eclampsia and eclampsia with the institution of early management.

In present study we observed a specific pattern of disease and its related variation in coagulation status. Simple and routine tests like CBC and platelet count are highly helpful in suspecting a derangement in the coagulation status early in the course of the disease and plan preemptive management strategies.

Finally, with present study results and interpretation with previous worker's studies, came to a conclusion that estimation of platelet count may be considered as an early, economical and rapid method of assessment of severity of hypertensive disorders of pregnancy cases. It can also be a useful screening test to assess the prognosis of the disease and outcome of pregnancy in pregnant women.

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PROFORMA

Name : Obstetric score:

Age : LMP :

Address : EDD :

Ip. No : Blood group:

Occupation : Ht : Wt:

Booked/ not : HIVstatus:

Referred/not :

Complaints

Present h/o :

period of amenorrhea pain abdomen

edema feet vomiting

headache oliguria

bluring of vision palpitation

Present Pregnancy

I trimester

II trimester

III trimester

Past obstetric history

Medical history

Family history

General Examination :

Temperature : CVS :

Pulse rate : RS :

Blood pressure : CNS :

Respiratory rate :

BMI :

Edema :

Anaemia :

Per abdomen :

Pervaginal :

Investigation

Urine - albumin: : Hb :

sugar : Pcv :

Platelet :

Blood - sugar : LFT :

urea :

creatinine :

uricacid :

electrolyte :

Ultra sound :

Anti-hypertensive : Drug : dose :

Gravidogram

Date U/A Wt Sfh AG BP Immiment symptom

$Mgso_4$:

Time Dose Temp RR I/O DTR PERL U/A

Maternal complications :

Mode of induction :

Indication for termination : Maternal / fetal

Vaginal delivery / LSCS

Latency interval:

Intra/ post partum complications

Baby : alive / still birth

gestational age

birth weight

apgar score

admitted / not

Follow up : NICU stay

Neonatal complications

CONSENT FORM

PATIENT NAME:

IP/OP NO.

STUDY TITLE :

“LOWERING PLATELET COUNT ,A PROGNOSTIC INDEX IN PREECLAMPSIA”

1.I have been explained and have understood the procedures involved in the study

2. I confirm that I have read and understand the information sheet for the above study.

3. I have had the opportunity to consider the information, ask questions and have had these answered satisfactorily.

4. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected.

5. I understand that relevant sections of any of my medical notes and data collected during the study may be looked at by responsible individuals from [Madras Medical College], where it is relevant to my taking part in this study. I give permission for these individuals to have access to my records.

6. I agree to take part in the above study.

Name and signature of interviewer Signature of Participant

Information Sheet

We are conducting a study on **““LOWERING PLATELET COUNT ,A PROGNOSTIC INDEX IN PREECLAMPSIA”**” among patients attending Institute of obstetrics and gynaecology, Chennai and for that your clinical details may be valuable to us.

- We are selecting certain patients and if you are found eligible, we may be using your clinical details in such a way so as to not affect your final report or management in any of the two groups assigned in my study.

- The privacy of the patients 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 this 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.

Signature of investigator Signature of participant

Date: .

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI 600 003

EC Reg.No.ECR/270/Inst./TN/2013
Telephone No.044 25305301
Fax: 011 25363970

CERTIFICATE OF APPROVAL

To
Dr. **C. SHAMRATH BANU**
I Yr. PG in MS (OG)
ISO/KGH/MMC
CHENNAI

Dear Dr. C. SHAMRATH BANU,

The Institutional Ethics Committee has considered your request and approved your study titled **"LOWERING PLATELET COUNT A PROGNOSTIC INDEX IN PREECLAMPSIA" - NO.28042018**

The following members of Ethics Committee were present in the meeting held on **03.04.2018** conducted at Madras Medical College, Chennai 3

1. Prof.P.V.Jayashankar :Chairperson
2. Prof.R.Jayanthi,MD.,FRCP(Glasg) Dean,MMC,Ch-3 : Deputy Chairperson
3. Prof.Sudha Seshayyan,MD., Vice Principal,MMC,Ch-3 : Member Secretary
4. Prof.N.Gopalakrishnan,MD,Director,Inst.of Nephrology,MMC,Ch : Member
5. Prof.S.Mayilvahanan,MD,Director,Inst. of Int.Med,MMC, Ch-3 : Member
6. Prof.A.Pandiya Raj,Director, Inst. of Gen.Surgery,MMC : Member
7. Prof.Shanthy Gunasingh, Director, Inst.of Social Obstetrics,KGH : Member
8. Prof.Rema Chandramohan,Prof.of Paediatrics,ICH,Chennai : Member
9. Prof. S. Purushothaman, Associate Professor of Pharmacology, MMC,Ch-3 : Member
10. Prof.K.Ramadevi,MD., Director, Inst. of Bio-Chemistry,MMC,Ch-3 : Member
11. Prof.Bharathi Vidya Jayanthi,Director, Inst. of Pathology,MMC,Ch-3: Member
12. Thiru S.Govindasamy, BA.,BL,High Court,Chennai : Lawyer
13. Tmt.Arnold Saulina, MA.,MSW., :Social Scientist
14. Thiru K.Ranjith, Ch- 91 : Lay Person

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

18/4/18

Member Secretary - Ethics Committee

ஆராய்ச்சி ஒப்புதல் படிவம்

ஆராய்ச்சி தலைப்பு: கர்ப்பகாலத்தில் இரத்த அழுத்தத்தில் தட்டணுக்கள் குறைபாடு.

எண். தேதி:

இந்த ஆராய்ச்சியின் விவரங்களும், அதன் நோக்கங்களும் முழுமையாக எனக்கு தெளிவாக விளக்கப்பட்டது. எனக்கு விளக்கப்பட்ட விஷயங்களை நான் புரிந்து கொண்டு, நான் எனது சம்மதத்தை தெரிவிக்கிறேன். இந்த ஆராய்ச்சியில் பிறரின் நிர்ப்பந்தமின்றி என் சொந்த விருப்பத்தின்பேரில்தான் பங்கு பெறுகிறேன் மற்றும் நான் இந்த ஆராய்ச்சியிலிரந்து எந்நேரமும் பின்வாங்கலாம் என்பதையும் அதனால் எந்த பாதிப்பும் ஏற்படாது. நான் என்னுடைய சுயநினைவுடன்மற்றும் முழு சுதந்திரத்துடன் இந்த மருத்துவ ஆராய்ச்சியில் என்னை சேர்த்துக் கொள்ள சம்மதிக்கிறேன்.

பங்கேற்பாளர் கையொப்பம்.

ஆராய்ச்சி தலைப்பு: கர்ப்பகாலத்தில் இரத்த அழுத்தத்தில் இரக்கத்தில் தட்டணுக்கள் குறைபாடு அரசு மருத்துவமனையில் ஆராய்ச்சி.

- நீங்கள் இந்த ஆராய்ச்சியில் பங்கேற்க நாங்கள் விரும்புகிறோம். இந்த ஆராய்ச்சியில் உங்களுடைய உடலிக்க குறிகளை பரிசோதனை செய்து அதன் தகவல்கள் ஆராய்வோம். அதனால் தங்களது நோயின் ஆய்வறிக்கையோ அல்லது சிகிச்சையோ பாதிப்பு ஏற்படாது என்று தெரிவித்துக் கொள்கிறோம்.
- முடிவுகளை அல்லது கருத்துக்களை வெளியிடும் போதோ அல்லது ஆராய்ச்சியின்போதோ தங்களது பெயரோ அல்லது அடையாளங்களையோ வெளியிடமாட்டோம்.
- இந்த ஆராய்ச்சியில் பங்கேற்பது தங்களுடைய விருப்பத்தின்பேரில்தான் இருக்கிறது. மேலும் நீங்கள் எந்நேரமும் இந்த ஆராய்ச்சியிலிருந்து பின்வாங்கலாம் என்பதை தெரிவித்துக் கொள்கிறோம்.
- இந்த சிறப்பு பரிசோதனைகளின் முடிவுகளை ஆராய்ச்சியின் முடிவின்போது தங்களுக்கு அறிவிப்போம் என்பதை தெரிவித்துக் கொள்கிறோம்.

ஆராய்ச்சியாளர் கையொப்பம்

பங்கேற்பாளர் கையொப்பம்

URKUND

Urkund Analysis Result

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PREDICTION OF PREECLAMPSIA BASED ON THE LATERAL LOCATION OF PLACENTA ON ULTRASONOGRA1.docx (D42395896)
full.docx (D58288569)
Thesis.docx (D31131174)
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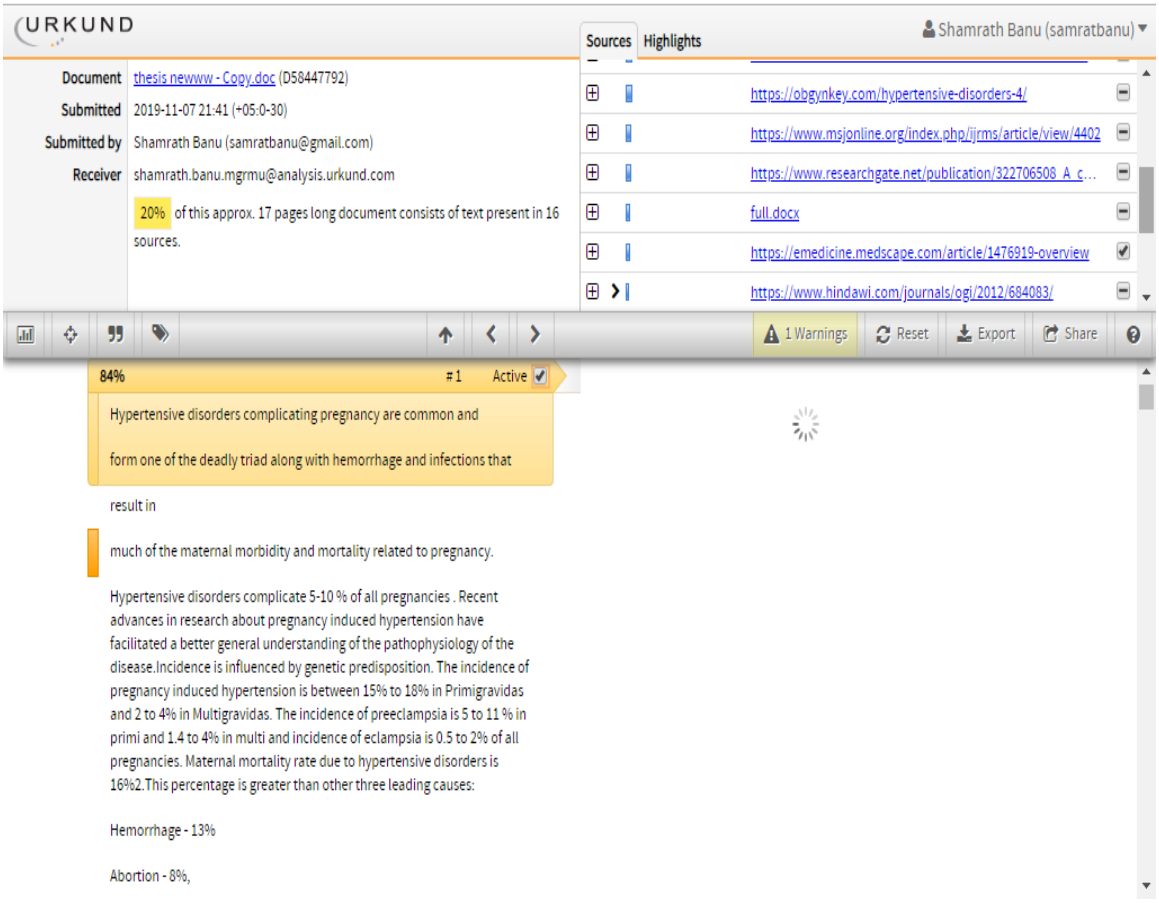
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Sources
Highlights
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https://obgynkey.com/hypertensive-disorders-4/
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1 Warnings
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Active
Hypertensive disorders complicating pregnancy are common and
form one of the deadly triad along with hemorrhage and infections that
result in
much of the maternal morbidity and mortality related to pregnancy.
Hypertensive disorders complicate 5-10 % of all pregnancies . Recent advances in research about pregnancy induced hypertension have facilitated a better general understanding of the pathophysiology of the disease.Incidence is influenced by genetic predisposition. The incidence of pregnancy induced hypertension is between 15% to 18% in Primigravidas and 2 to 4% in Multigravidas. The incidence of preeclampsia is 5 to 11 % in primi and 1.4 to 4% in multi and incidence of eclampsia is 0.5 to 2% of all pregnancies. Maternal mortality rate due to hypertensive disorders is 16%2.This percentage is greater than other three leading causes:
Hemorrhage - 13%
Abortion - 8%,

PLAGIARSM CERTIFICATE

This is to certify that this dissertation work titled **"LOWERING PLATLET COUNT, A PROGNOSTIC INDEX IN PREECLAMPSIA"** of the candidate **Dr.SHAMRATH BANU. C.** with **Reg. No.221716016** for the award of M.S in the branch of **OBSTETRICS AND GYNAECOLOGY**. I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion pages and the result shows twenty percentage of plagiarism in the dissertation (D58447792)

Signature and Seal of the Guide

Prof. KANMANI, M.D., O. G.,
Professor
Kasturba Gandhi Hospital
Triplicane,
Chennai

LIST OF ABBREVIATIONS

PIH	-	Pregnancy Induced Hypertension
GHT	-	Gestational HyperTension
Mild PE	-	Mild Pre Eclampsia
Severe PE	-	Severe Pre Eclampsia
AP Eclampsia	-	AntePartum Eclampsia
SES	-	Socio Economic Class
HELLP	-	Hemolysis, Elevated Liver Enzymes, Low Platelet count
U/A	-	Urine Albumin
MMR	-	Maternal Mortality Rate
DIVC	-	Disseminated Intra Vascular
IUGR	-	Intra Uterine Growth Retardation
ACOG	-	American College Obstetrics and Gynecology
NHBPEP	-	National High Blood Pressure Education Program
BP	-	Blood Pressure
SBP	-	Systolic Blood Pressure

S.NO	NAME	AGE	PARITY	BP	U/A	PRE-ECLAMPSIA	PERIOD OF	PLATELET	FETAL	MODE OF	THROMBOCYTOPENIA
				mm Hg		MILD/SEVERE/ ECLAMPSIA	GA AT	COUNT	OUTCOME	DELIVERY	WITH COAGULOPATHY
							WHICH				AND OTHER
							TERMINATED				MATERNAL
											COMPLICATIONS
1	MONISHA	25	PRIMI	100/60	NIL	NORMAL	40wks+3d	3.2L	Alive term girl baby	Vaginal	NO
2	SOUNDARYA	32	G2P1L1	120/70	NIL	NORMAL	37wks	1L	Late preterm boy	LSCS	NO
3	GANGA	21	G2A1L0	120/80	NIL	NORMAL	38wks	2.5L	Term boy	Vaginal	NO
4	ILAKYA	20	PRIMI	110/80	NIL	NORMAL	32wks	1.2L	Preterm baby RDS	Vaginal	PPH-MEDICALLY MANAG
5	SAMUNDEESHWARI	18	PRIMI	120/70	NIL	NORMAL	40wks+3d	1.8L	Term boy	Vaginal	NO
6	KAVITHA	23	G3P2L1	110/60	NIL	NORMAL	37wks+4d	2.5L	Term boy	Vaginal	NO
7	PRABHA	35	PRIMI	100/70	NIL	NORMAL	38wks+5d	3.2L	Term girl	LSCS	NO
8	KAUSALYA	38	G3P2L2	120/20	NIL	NORMAL	35wks+5d	2.8L	Preterm baby RDS	LSCS	NO
9	FARHANA	18	PRIMI	110/80	NIL	NORMAL	38wks	3.2L	Term	Vaginal	NO
10	KRIBAVATHY	21	PRIMI	110/70	NIL	NORMAL	40wks	2.7L	Term	Vaginal	NO
11	Gayathri	32	PRIMI	122/80	NIL	NORMAL	40wks	2.8L	Term	Vaginal	NO
12	Nayagi	30	PRIMI	110/80	NIL	NORMAL	40wks	2.8L	Term	Vaginal	NO
13	Abirami	21	G2P1L1	124/70	NIL	NORMAL	35wks+5d	3.2L	Preterm	Vaginal	NO
14	Subhana	28	PRIMI	120/80	NIL	NORMAL	40w+2d	2.5L	Term	LSCS	PPH-MEDICALLY MANAG
15	Charmila	26	G2P1L1	100/70	NIL	NORMAL	38wks	2.6L	Term	Vaginal	NO
16	Gowri	18	G2P1L1	110/70	NIL	NORMAL	37w+4d	2.8L	Term	LSCS	NO

17	Dnapriya	22	PRIMI	120/80	NIL	NORMAL	38wks	3.1L	Term	LSCS	NO
18	Pratiksha	38	PRIMI	120/80	NIL	NORMAL	40w+3d	2.5L	Term	Vaginal	NO
19	Sarah	40	PRIMI	120/70	NIL	NORMAL	40wks	1.5L	Term	Vaginal	NO
20	Jenifer	20	G3P2L2	110/60	NIL	NORMAL	36wks	1.8L	Term	Vaginal	NO
21	Joharah	25	PRIMI	120/70	NIL	NORMAL	40w+2d	2.5L	Term	Vaginal	NO
22	Ramya	33	PRIMI	110/80	NIL	NORMAL	35wks+5d	2.4L	Preterm	Vaginal	NO
23	Subathra	40	PRIMI	120/80	NIL	NORMAL	38w+2d	2.1L2.5	Term	LSCS	NO
24	Kavitha	32	G2A1	100/80	NIL	NORMAL	37wks	3.0L	Term	LSCS	NO
25	Sundaravalli	21	G2P1L1	120/80	NIL	NORMAL	38w+5d	2.3L	Term	LSCS	NO
26	Manomani	28	PRIMI	110/70	NIL	NORMAL	38wks	3.8L	Term	Vaginal	NO
27	Savitha	25	PRIMI	100/60	NIL	NORMAL	35wks+5d	2.8L	Preterm	LSCS	NO
28	Karuna	23	G2P1L1	100/80	NIL	NORMAL	37wks	3.5L	Term	LSCS	NO
29	Malliga	22	G3P2L1	120/70	NIL	NORMAL	40w+3d	1.9L	Term	Vaginal	NO
30	Tamilselvi	18	PRIMI	120/80	NIL	NORMAL	40wks	2.5L	Term	LSCS	NO
31	Markalai	30	PRIMI	120/70	NIL	NORMAL	38wks	2.5L	Term	Vaginal	NO
32	Sulekha	18	PRIMI	110/80	NIL	NORMAL	38w+5d	2.8L	Term	Vaginal	NO
33	Sarala	21	PRIMI	120/70	NIL	NORMAL	32wks	2.8L	Preterm	Vaginal	NO
34	Geethadevi	19	PRIMI	120/80	NIL	NORMAL	38wks	3.2L	Term	Vaginal	NO
35	Vaishnavi	30	PRIMI	110/80	NIL	NORMAL	32wks	1.8L	Preterm	Vaginal	NO
36	Shanmugapriya	25	PRIMI	112/80	NIL	NORMAL	38w+5d	1.5L	Term	Vaginal	PPH-MEDICALLY MANAG
37	Vasanthi	28	G2P1L1	116/72	NIL	NORMAL	40wks	#####	Term	Vaginal	NO
38	Anandhi	22	G2P1L1	120/70	NIL	NORMAL	40w+4d	2.2L	Stillbirth	LSCS	NO
39	Sundaradevi	28	PRIMI	100/80	NIL	NORMAL	39wks	3.6L	Term	LSCS	NO
40	Parvathi	27	G2A1	120/70	NIL	NORMAL	38wks	2.0L	Term	LSCS	NO
41	Madhavi	26	PRIMI	120/80	NIL	NORMAL	40w+2d	3.2L	Term	Vaginal	NO
42	Shanthi	22	G2P1L1	110/70	NIL	NORMAL	40w+3d	3.0L	Term	LSCS	NO
43	Yashodha	28	G2P1L0	100/80	NIL	NORMAL	38wks	2.8L	Term	LSCS	NO
44	Vanitha	35	PRIMI	120/70	NIL	NORMAL	37wks	2.5L	Term	Vaginal	NO
45	Ragavi	24	PRIMI	120/70	NIL	NORMAL	37w+5d	2.8L	IUD	Vaginal	NO
46	Ananya	22	PRIMI	110/80	NIL	NORMAL	35wks+5d	3.2L	Term	Vaginal	NO
47	Rajeshwari	21	G3P2L1	100/80	NIL	NORMAL	38wks	1.8L	Term	LSCS	NO
48	Vijaya	20	PRIMI	120/70	NIL	NORMAL	40wks	1.9L	Term	LSCS	NO
49	Saranya	23	G2P1L1	120/80	NIL	NORMAL	40w+2d	2.2L	Term	Vaginal	NO

50	Vanishree	28	G2P1L1	110/70	NIL	NORMAL	40w+3d	2.5L	Term	LSCS	NO
51	Leelavathy	22	PRIMI	100/60	NIL	NORMAL	40w+3d	1.5L	Term	Vaginal	NO
52	Sandhiya	28	PRIMI	120/80	NIL	NORMAL	40w+3d	2.8L	Term	Vaginal	NO
53	Fathima	31	G2P1L1	124/70	NIL	NORMAL	38wks	3.2L	Term	Vaginal	PPH-MEDICALLY MANAG
54	Julie	27	PRIMI	110/60	NIL	NORMAL	38w+5d	1,5L	Term	Vaginal	NO
55	Arokya	25	PRIMI	100/80	NIL	NORMAL	37wks	2.6L	Term	Vaginal	NO
56	Tamilarasi	30	G3A2	110/70	NIL	NORMAL	38w+2d	2.8L	Term	Vaginal	NO
57	Buvana	18	G2P1L1	120/70	NIL	NORMAL	39wks	1.6L	Term	LSCS	NO
58	Beluah	23	PRIMI	120/70	NIL	NORMAL	39w+5d	2.3L	Term	LSCS	NO
59	lLalitha	24	PRIMI	100/80	NIL	NORMAL	38wks	2.5L	Term	LSCS	NO
60	Elavarasi	26	PRIMI	124/70	NIL	NORMAL	37wks	2.7L	Term	Vaginal	NO
61	Brgavi	26	PRIMI	110/60	NIL	NORMAL	38w+5d	2.8L	Term	Vaginal	NO
62	Bavana	27	G2A1	116/80	NIL	NORMAL	39w+4d	2,6L	Term	LSCS	NO
63	Aneesha	32	PRIMI	110/74	NIL	NORMAL	39wks	2.8L	Term	Vaginal	PPH-MEDICALLY MANAG
64	Maheshwari	18	PRIMI	114/74	NIL	NORMAL	40wks	1.4L	Term	Vaginal	NO
65	Mary Joseph	36	G2P1L1	120/84	NIL	NORMAL	40w+2d	2.5L	Term	Vaginal	NO
66	Eswari	16	PRIMI	120/76	NIL	NORMAL	40w+1d	3.2L	Term	Vaginal	NO
67	Buvanehwari	22	PRIMI	112/68	NIL	NORMAL	39w+5d	3.4L	Term	Vaginal	NO
68	Andaleshwari	25	G2P1L1	122/78	NIL	NORMAL	40wks	3.8L	Term	LSCS	NO
69	Panula	28	PRIMI	120/80	NIL	NORMAL	39wks	3.2L	Term	LSCS	NO
70	Elakya	30	G3P2L2	126/64	NIL	NORMAL	40w+4d	1.8L	Term	Vaginal	NO
71	Alamelu	22	PRIMI	110/80	NIL	NORMAL	40w+3d	3.5L	Term	Vaginal	NO
72	Pradeepa	24	G2P1L1	110/70	NIL	NORMAL	40w+2d	2.5L	Term	Vaginal	PPH-MEDICALLY MANAG
73	Rosy	25	G4A3	112/64	NIL	NORMAL	38wks	2.1L	Term	Vaginal	NO
74	Archana	24	PRIMI	110/80	NIL	NORMAL	38wks	2.6L	Term	Vaginal	NO
75	Malarvizhi	23	PRIMI	120/60	NIL	NORMAL	38w+5d	2.7L	Term	Vaginal	NO
76	Gayathri	22	PRIMI	126/86	NIL	NORMAL	32wks	3.1L	Preterm	Vaginal	NO
77	Arpita	18	G2P1L1	124/84	NIL	NORMAL	39w+4d	2.5L	Term	Forceps	NO
78	Sruthi	20	G2P1L1	120/84	NIL	NORMAL	36wks	2.4L	Term	LSCS	NO
79	Deepna	21	G3P1L1	122/88	NIL	NORMAL	38wks	1.4L	Term	LSCS	PPH-MEDICALLY MANAG
80	Krishnaveni	32	PRIMI	100/80	NIL	NORMAL	38wks	2.5L	Term	LSCS	NO
81	Mary Joseph	22	G2P1L1	120/84	NIL	NORMAL	40wks	3.2L	Term	LSCS	NO
82	Gajalakshmi	18	PRIMI	114/86	NIL	NORMAL	40wks	3.1L	Term	LSCS	NO

83	Sowmiya	26	G3P2L2	126/80	NIL	NORMAL	38wks	2.8L	Term	LSCS	NO
84	Arpita	28	PRIMI	120/70	NIL	NORMAL	38w+5d	2.8L	Term	LSCS	NO
85	Vasuki	26	PRIMI	110/70	NIL	NORMAL	37w+2d	1.5L	Term	LSCS	NO
86	Elakya	32	G2A1	100/60	NIL	NORMAL	38wks	#####	Term	Vaginal	NO
87	Gajapriya	30	PRIMI	112/80	NIL	NORMAL	37wks	2.5L	Term	Vaginal	PPH-MEDICALLY MANAG
88	Brindha	25	PRIMI	128/78	NIL	NORMAL	38w+2d	2.6L	Term	Vaginal	NO
89	Balapriya	28	PRIMI	110/70	NIL	NORMAL	38wks	2.8L	Term	Vaginal	PPH-MEDICALLY MANAG
90	Visalatchi	22	G2P1L1	100/76	NIL	NORMAL	40w+3d	1.4L	Term	Forceps	NO
91	Ganga	24	PRIMI	124/68	NIL	NORMAL	40wks	2.3L	Term	Vaginal	NO
92	Gandhimathi	28	G2P1L1	120/70	NIL	NORMAL	40wks	2.8L	Term	Vaginal	NO
93	Margali	25	PRIMI	110/80	NIL	NORMAL	38wks	3.2L	Term	Vaginal	NO
94	Vidhya	32	G2A1	106/84	NIL	NORMAL	37wks	1.8L	Term	Vaginal	NO
95	Vijayalakshmi	31	G2A1	112/86	NIL	NORMAL	38k	2.5L	Term	LSCS	NO
96	Divya	18	PRIMI	120/60	NIL	NORMAL	40w+3d	2.8L	Term	LSCS	NO
97	Muthulakshmi	20	PRIMI	110/80	NIL	NORMAL	40w+3d	1.7L	Term	Vaginal	NO
98	Yamuna	22	G2P1L1	100/70	NIL	NORMAL	38wks	2.8L	Term	Vaginal	NO
99	Supriya	21	G2P1L1	124/74	NIL	NORMAL	38w+2d	2.4L	Term	Vaginal	NO
100	Geethanjali	30	G3P2L2	100/84	NIL	NORMAL	38w+6d	2.1L	Term	Vaginal	PPH-MEDICALLY MANAG
101	Vinodha	30	PRIMI	120/68	NIL	NORMAL	40wks	1.4L	Term	Vaginal	NO
102	Mahalakshmi	32	G3A2	120/84	NIL	NORMAL	4ow+2d	2.5L	Term	Vaginal	NO
103	Bavya	21	PRIMI	100/80	NIL	NORMAL	40w+3d	2.8L	Term	Vaginal	NO
104	Deepa	20	PRIMI	126/88	NIL	NORMAL	38wks	2.1L	Term	Vaginal	NO
105	Kanagavalli	21	PRIMI	100/70	NIL	NORMAL	38wks	3.8L	Term	Vaginal	NO
106	Anjali	18	PRIMI	110/84	NIL	NORMAL	33wks	3.4L	Preterm	LSCS	PPH-MEDICALLY MANAG
107	Pushpa	16	PRIMI	100/88	NIL	NORMAL	38w+4d	2.5L	Term	LSCS	NO
108	Reshma	18	PRIMI	120/80	NIL	NORMAL	38w+5d	2.8L	Term	LSCS	NO
109	Maya	22	PRIMI	110/84	NIL	NORMAL	36wks	1.2L	Term	Vaginal	NO
110	Saraswathi	25	PRIMI	100/78	NIL	NORMAL	38w+2d	1.8L	Term	Vaginal	NO
111	Divyadarshini	25	G2P1L1	120/84	NIL	NORMAL	40wks	1.8L	Term	Vaginal	NO
112	Janani	22	G2P1L1	110/80	NIL	NORMAL	38wks	2.5L	Term	LSCS	NO
113	Darshana	25	PRIMI	110/86	NIL	NORMAL	38w+2d	2.4L	Term	LSCS	NO
114	Deepika	26	PRIMI	120/76	NIL	NORMAL	38w+3d	2.8L	Term	LSCS	NO
115	Nivetha	21	PRIMI	100/86	NIL	NORMAL	34wks	3.2L	Preterm	Vaginal	NO

116	Selvi	18	PRIMI	120/74	NIL	NORMAL	38wks	1.7L	Term	LSCS	NO
117	Brinda	30	PRIMI	100/88	NIL	NORMAL	39w+4d	1.8L	Term	LSCS	NO
118	Saraswathi	25	G2A1	110/86	NIL	NORMAL	39w+3d	2.7L	Term	Vaginal	PPH-MEDICALLY MANAG
119	Priyanka	26	PRIMI	120/76	NIL	NORMAL	40w+3d	2.5L	Term	Vacuum	NO
120	Anjana	25	G2P1L1	110/70	NIL	NORMAL	40+2d	2.4L	Term	Vaginal	NO
121	Deepalakshmi	28	PRIMI	120/86	NIL	NORMAL	38wks	2.4L	Term	Vaginal	NO
122	Danshiks	18	PRIMI	120/80	NIL	NORMAL	38w+2d	1.8L	Term	Vaginal	NO
123	Priyanka	21	PRIMI	110/76	NIL	NORMAL	40w+2d	2.4L	Term	Vaginal	NO
124	Senthamarai	27	G2P1L1	100/88	NIL	NORMAL	38wks	2.5L	Term	Vaginal	NO
125	Krithi	24	PRIMI	120/86	NIL	NORMAL	39wks	2.8L	Term	Vaginal	NO
126	Priyadarshini	26	G2P1L1	110/84	NIL	NORMAL	40w+1d	3.1L	Term	Vaginal	NO
127	Jameela	28	PRIMI	100/74	NIL	NORMAL	40w+3d	2,5L	Term	Vaginal	NO
128	Amala	30	G2P1L1	110/74	NIL	NORMAL	38w+2d	3,5L	Term	Vaginal	NO
129	Zubaida	18	G2A1	120/88	NIL	NORMAL	35eks	1.2L	Term	Vaginal	NO
130	Priya	21	PRIMI	124/80	NIL	NORMAL	38wks	2.2L	Term	LSCS	NO
131	Danalakshmi	25	PRIMI	100/78	NIL	NORMAL	38wks	2.1L	Term	Vaginal	NO
132	Ankita	26	PRIMI	100/68	NIL	NORMAL	38w+4d	2.2L	Term	Vaginal	NO
133	Arifa	24	PRIMI	120/84	NIL	NORMAL	38w+5d	2.8L	Term	Vaginal	NO
134	Keerthika	27	G2P1L1	120/88	NIL	NORMAL	40w+3d	1.8L	Term	Vaginal	NO
135	Priya	30	G2P1L1	120/64	NIL	NORMAL	40w+2d	3.2L	Term	Vaginal	NO
136	Vanathi	18	PRIMI	120/88	NIL	NORMAL	35wks+5d	2.2L	Preterm	Vaginal	NO
137	Kavya	28	G2P1L1	126/78	NIL	NORMAL	38w+3d	2.4L	Term	Vaginal	NO
138	Garudalakshmi	23	G2A1	110/80	NIL	NORMAL	38w+4d	2.5L	Term	LSCS	NO
139	Saleema	27	PRIMI	100/66	NIL	NORMAL	40w+3d	2.5L	Term	LSCS	NO
140	Thilaga	28	G2P1L1	120/84	NIL	NORMAL	40w+3d	2.4L	Term	Vaginal	NO
141	Gunavathy	30	PRIMI	110/88	NIL	NORMAL	40w+2d	2.8L	Term	Vaginal	NO
142	Thilsath	28	PRIMI	120/68	NIL	NORMAL	40w+3d	2.1L	Term	LSCS	NO
143	Sameena	25	G2A1	126/88	NIL	NORMAL	38wks	2.5L	Term	LSCS	NO
144	Subhulakshmi	31	PRIMI	120/84	NIL	NORMAL	38wks	2.6L	Term	LSCS	NO
145	Karthiga	18	G2P1L1	110/60	NIL	NORMAL	38w+2d	1.9L	Term	Vaginal	NO
146	Gayathri	26	G3P2L2	100/86	NIL	NORMAL	37w+4d	1.8L	Term	Vaginal	NO
147	Keerthika	22	G3P2L2	120/68	NIL	NORMAL	34wks	2.6L	Preterm	Vaginal	NO
148	Thilothama	28	PRIMI	114/78	NIL	NORMAL	36wks	1.5L	Term	Vaginal	NO

149	Ishwarya	22	PRIMI	124/80	NIL	NORMAL	38wks	2.8L	Term	Vaginal	NO
150	Aysha	30	G2P1L1	118/74	NIL	NORMAL	40w+2d	2.8L	Term	Vaginal	NO
151	Malathy	22	PRIMI	140/90	NIL	GHT	38wks	2.5L	Term	Vaginal	NO
152	Vijaya	24	PRIMI	142/96	NIL	GHT	38wks	2.5L	Term	Vaginal	NO
153	Nandhini	20	PRIMI	140/94	NIL	GHT	38wks	2.5L	Term	Vaginal	NO
154	Delphin	18	PRIMI	1444/94	NIL	GHT	38wks	2.5L	Term	Vaginal	NO
155	Farhana	32	PRIMI	140/90	TRAC	GHT	38wks	2.5L	Term	Vaginal	NO
156	Yogalakshmi	36	PRIMI	142/98	NIL	GHT	38wks	2.4L	Term	Vaginal	NO
157	Monisha	31	PRIMI	140/90	NIL	GHT	37wks	2,4L	Term	LSCS	NO
158	Sindhiya	25	PRIMI	142/94	NIL	GHT	36wks	2.4L	Preterm	Vaginal	NO
159	Valarmathy	21	PRIMI	140/9 6	NIL	GHT	36wks	2.4L	Preterm	Vaginal	NO
160	Ganga	19	PRIMI	140/90	NIL	GHT	36wks	1.9L	Preterm	Vaginal	NO
161	Abirami	24	G2P1L1	148/96	NIL	GHT	38wks	2.2L	Term	Vaginal	PPH-MEDICALLY MANAG
162	Seetha	26	PRIMI	140/90	NIL	GHT	38wks	2.1L	Term	Vaginal	NO
163	Saranya	25	G2P1L1	140/90	NIL	GHT	38wks	2.3L	Term	Vaginal	NO
164	Vinodhini	22	PRIMI	148/96	NIL	GHT	38w+2d	2.5L	Term	Vaginal	NO
165	Anbarai	21	PRIMI	140/94	NIL	GHT	35wks+5d	2.4L	Preterm	Vaginal	NO
166	Meera	20	PRIMI	140/90	NIL	GHT	36wks	2.6L	Preterm	Vaginal	NO
167	Matilda	26	PRIMI	148/96	NIL	GHT	37w+2d	1.9L	Term	Vaginal	NO
168	Deepthi	28	G2P1L1	142/90	NIL	GHT	37w+2d	1.8L	Term	LSCS	NO
169	Selvi	30	G2P1L1	148/94	NIL	GHT	38wks	2.5L	Term	LSCS	NO
170	Mookamal	32	G2P1L1	146/90	TRAC	GHT	38wks	2.1L	Term	LSCS	NO
171	Kavya	30	G2A1	146/98	NIL	GHT	38wks	2.5L	Term	Vaginal	NO
172	Nivetha	31	G2A1	144/90	NIL	GHT	37wks	2.4L	Term	Vaginal	NO
173	Sowmya	21	PRIMI	142/98	NIL	GHT	37w+3d	1.9L	Term	Vaginal	NO
174	Ponmani	22	PRIMI	140/96	TRAC	GHT	38wks	2.3L	Term	Vaginal	NO
175	Jenny	25	PRIMI	142/90	TRAC	GHT	36wks	2.4L	Preterm	Vaginal	NO
176	Subitha	26	G2P1L1	144/98	NIL	GHT	37w+3d	1.8L	Term	Vaginal	NO
177	Thasleema	26	G2A1	140/90	NIL	GHT	38w+2d	1.7L	Term	Vaginal	NO
178	Tamilarasi	28	G2A1	146/98	NIL	GHT	38wks	2.2L	Term	LSCS	NO
179	Kumaravati	19	G2A1	140/96	NIL	GHT	38wks	2.4L	Term	LSCS	NO
180	Rajameena	20	PRIMI	140/90	NIL	GHT	35wks	2.6L	Preterm	Vaginal	NO
181	Lakshmi	22	G2P1L1	142/94	NIL	GHT	38wks	2.2L	Term	Vaginal	NO

182	Narmadha	28	G3P2L2	140/92	NIL	GHT	37wks	2.4L	Term	Vaginal	NO
183	Kalaiarasi	25	G23P2L2	140/92	NIL	GHT	38wks	2.8L	Term	Vaginal	NO
184	Bakyalakshmi	23	G3P2L2	142/96	NIL	GHT	37w+4d	1.8L	Term	Vaginal	NO
185	Ragini	23	G3P2L2	140/90	NIL	GHT	38wks	1.7L	Term	Vaginal	NO
186	Shobana	26	G3P2L2	142/98	NIL	GHT	37w+6d	2.2L	Term	Vaginal	NO
187	Anjali	21	G3P2L2	144/92	TRAC	GHT	38wks	2,5L	Term	Vaginal	NO
188	Susheela	19	G3P2L2	144/96	NIL	GHT	38wks	2.4L	Term	Vaginal	NO
189	Jamine	31	G3P2L2	146/98	NIL	GHT	38wks	2.6L	Term	LSCS	NO
190	Lalitha	36	G3P2L2	140/90	NIL	GHT	38w+2d	1.8L	Term	LSCS	NO
191	Karpagam	19	G3P2L1	140/100	NIL	GHT	38wks	#####	Term	Vaginal	NO
192	Jabavalli	21	G3P2L1	142/98	NIL	GHT	37w+2d	1.5L	Term	LSCS	NO
193	Swathika	26	G3P2L1	144/90	NIL	GHT	38wks	2.1L	Term	LSCS	NO
194	Gousalya	25	G3PL1	140/96	NIL	GHT	38wks	2.5L	Term	Vaginal	NO
195	Alangu	25	G3P2L1	142/98	NIL	GHT	37w+2d	2.7L	Term	Vaginal	NO
196	Suganthi	28	G3P2L1	140/94	NIL	GHT	38wks	2.2L	Term	Vaginal	NO
197	Divya	31	G3P2L1	140/98	NIL	GHT	36wks	2.1L	Preterm	Vaginal	NO
198	Sudha	19	G3P2L1	146/98	NIL	GHT	37w+2d	1.9L	Term	Vaginal	NO
199	Arundhthi	22	G3P2L1	142/96	NIL	GHT	38wks	2.3L	Term	Vaginal	NO
200	Kalaivani	23	G3P2L1	146/94	NIL	GHT	38wks	2.3L	Term	Vaginal	NO
201	Kannagi	23	G2P1L0	142/98	NIL	GHT	38wks	2.5L	Term	Vaginal	NO
202	Ragusudha	26	G2P1L0;G	144/96	NIL	GHT	38wks	2.4L	Term	Vaginal	NO
203	Valli	25	G2P1L0	142/96	NIL	GHT	38w+2d	2.1L	Term	Vaginal	NO
204	Arokyamary	25	G2P1L0	142/90	NIL	GHT	37w+4d	2.1L	Term	Vaginal	NO
205	Regina	28	G2P1L0	142/96	NIL	GHT	38wks	2.1L	Term	Vaginal	NO
206	Kala	31	G2P1L0	134/96	NIL	GHT	37wks	2.1L	Term	Vaginal	NO
207	Alagu	19	G2P1L0	142/98	NIL	GHT	38wks	2.1L	Term	LSCS	NO
208	Krishnapriya	21	G2P1L0	138/90	NIL	GHT	37w+3d	1.5L	Term	LSCS	NO
209	Pratiba	23	G2P1L0	140/94	NIL	GHT	36wks	1.5L	Preterm	Vaginal	NO
210	Ramani	28	G2P1L0	148/96	NIL	GHT	35wks	2.1L	Preterm	LSCS	NO
211	Rubitha	28	PRIMI	146/90	1+	Mild pre-eclamps	38wks	2.2L	Term	Vaginal	NO
212	Sridevi	22	PRIMI	144/90	1+	Mild pre-eclamps	36wks	2.5L	Preterm	Vaginal	NO
213	Linu	28	PRIMI	142/96	1+	Mild pre-eclamps	38wks	2.4L	Term	Vaginal	NO
214	Roja	26	G2A1	144/96	1+	Mild pre-eclamps	38wks	1.9L	Term	Vaginal	NO

215	Kalyani	21	G2P1L1	148/94	1+	Mild pre-eclamps	37wks	2.2L	Term	Vaginal	PPH-MEDICALLY MANAG
216	Rukmani	31	PRIMI	140/90	1+	Mild pre-eclamps	35wks	2.3L	Preterm	Vaginal	NO
217	RushyaBegum	19	PRIMI	142/90	1+	Mild pre-eclamps	32wks	2.5L	Preterm	LSCS	HELLP SYNDROME
218	Nagarani	21	G2P1L0	142/96	1+	Mild pre-eclamps	38wks	#####	Term	LSCS	NO
219	Annakodi	23	G3A2	142/94	1+	Mild pre-eclamps	38wks	1.9L	Term	Vaginal	NO
220	Aparna	25	G2P1L1	142/96	1+	Mild pre-eclamps	38wks	1.8L	Term	LSCS	NO
221	Arthi	28	PRIMI	138/96	1+	Mild pre-eclamps	38wks	2.5L	Term	Vaginal	HELLP SYNDROME
222	Soundarya	28	G2A1	142/96	1+	Mild pre-eclamps	37wks	#####	Term	Vaginal	NO
223	Monika	25	G2P1L1	140/90	1+	Mild pre-eclamps	38wks	1.9L	Term	Vaginal	NO
224	Aruna	24	PRIMI	148/94	1+	Mild pre-eclamps	38wks	2.1L	Term	Vaginal	NO
225	Preethi	23	G2P1L1	140/94	1+	Mild pre-eclamps	35wks	2.1L	Preterm	Vaginal	NO
226	Srividhya	28	G3P2L2	140/94	1+	Mild pre-eclamps	32wks	2.2L	Preterm	LSCS	NO
227	Nirosha	30	G2P1L0	140/96	1+	Mild pre-eclamps	37wks	1.9L	Term	LSCS	NO
228	Nirmala	31	PRIMI	142/96	1+	Mild pre-eclamps	37wks	1.8L	Term	Vaginal	NO
229	Bavani	23	PRIMI	142/98	1+	Mild pre-eclamps	38wks	1.9L	Term	Vaginal	NO
230	Jayaseela	18	PRIMI	140/90	1+	Mild pre-eclamps	37wks	2.5L	Term	Forceps	NO
231	Princy	19	PRIMI	140/104	1+	Mild pre-eclamps	35wks	2.5L	Preterm	Vaginal	NO
232	Sharadha	22	PRIMI	140/100	1+	Mild pre-eclamps	38wks	2.5L	Term	Vaginal	NO
233	Deepapriya	26	PRIMI	142/106	1+	Mild pre-eclamps	38wks	2.5L	Term	Vaginal	NO
234	Vandana	28	PRIMI	142/108	1+	Mild pre-eclamps	37wks	2.4L	Term	Vaginal	NO
235	Chandra	31	PRIMI	146/100	1+	Mild pre-eclamps	36wks	2.4L	Preterm	Vaginal	NO
236	Swathika	20	PRIMI	140/100	1+	Mild pre-eclamps	36wks	1.9L	Preterm	Vaginal	NO
237	Nila	21	PRIMI	142/104	1+	Mild pre-eclamps	37wks	1.9L	preterm	LSCS	NO
238	Janani	25	PRIMI	148/108	1+	Mild pre-eclamps	36wks	2.2L	preterm	LSCS	NO
239	Durga	32	PRIMI	146/102	1+	Mild pre-eclamps	38wks	1.8L	Term	LSCS	NO
240	Charu	21	PRIMI	142/110	1+	Mild pre-eclamps	38wks	1.8L	Term	LSCS	NO
241	Shageerabanu	25	GWP1L1	144/106	1+	Mild pre-eclamps	38wks	2.4L	Term	LSCS	NO
242	Kujil	28	G2P1L1	142/100	1+	Mild pre-eclamps	37wks	2.5L	Term	LSCS	NO
243	Yazhini	31	G2P1L1	140/108	1+	Mild pre-eclamps	38wks	2.4L	Term	LSCS	NO
244	Usha	19	G2P1L1	148/104	1+	Mild pre-eclamps	36wks	2.2L	Preterm	LSCS	NO
245	Sridivya	22	G2P1L1	140/100	1+	Mild pre-eclamps	37wks	2.2L	Term	LSCS	NO
246	Banu	26	G2P1L1	142/108	1+	Mild pre-eclamps	38wks	1.9L	Term	LSCS	HELLP SYNDROME
247	Vanitha	30	G2A1	140/100	1+	Mild pre-eclamps	37wks	1.5L	Term	Vaginal	NO

248	Reeta	19	G2A1	144/108	1+	Mild pre-eclamps	36wks	2.1L	Preterm	Vaginal	NO
249	Jenitha	22	G2A1	148/100	1+	Mild pre-eclamps	35wks	2.1L	Preterm	Vaginal	NO
250	Arul	25	G2A1	142/100	1+	Mild pre-eclamps	35wks	2.2L	preterm	Vaginal	NO
251	Malavika	22	PRIMI	160/120	2+	Severe pre-eclam	38wks	1.8L	Term	Vaginal	NO
252	Malini	19	G2P1L1	170/112	2+	Severe pre-eclam	37wks	1.8L	Term	Vaginal	NO
253	Veera	25	PRIMI	180/110	2+	Severe pre-eclam	35wks	1.9L	Preterm	Vaginal	NO
254	Ruku	28	PRIMI	168/112	2+	Severe pre-eclam	32wks	2.2L	Preterm	Vaginal	NO
255	Lakshmi	32	G2A1	160/112	2+	Severe pre-eclam	36wks	1.2L	Preterm	LSCS	NO
256	Aparna	28	PRIMI	160/110	2+	Severe pre-eclam	38wks	1.6L	Term	LSCS	NO
257	Nagamani	28	G2P1L1	170/120	2+	Severe pre-eclam	33wks	#####	Preterm	LSCS	HELLP SYNDROME
258	Vinitha	19	G2A1	160/110	2+	Severe pre-eclam	36wks	1.8L	Preterm	LSCS	NO
259	Punitha	24	G2P1L1	162/72	2+	Severe pre-eclam	37wks	1L	Term	LSCS	PPH-MEDICALLY MANAG
260	Gayathri	24	PRIMI	160/110	2+	Severe pre-eclam	37wks	1L	Term	LSCS	NO
261	Rithika	25	PRIMI	162/110	2+	Severe pre-eclam	35wks	2.1L	Preterm	Vaginal	HELLP SYNDROME
262	Sangeetha	24	PRIMI	164/108	2+	Severe pre-eclam	36wks	1.8L	Preterm	Vaginal	NO
263	Valli	26	PRIMI	170/112	1+	Severe pre-eclam	38wks	1.6L	Term	Vaginal	NO
264	Pushpalatha	27	G2P1L1	170/108	1+	Severe pre-eclam	34wks	1.8L	Preterm	Vaginal	NO
265	Prathiba	28	PRIMI	168/108	1+	Severe pre-eclam	32wks	2L	Preterm	LSCS	PPH-MEDICALLY MANAG
266	Vadivu	30	G2P1L1	168/112	2+	Severe pre-eclam	38wks	2.2L	Term	LSCS	NO
267	Sahana	31	G2A1	170/104	2+	Severe pre-eclam	38wks	1.9L	Term	LSCS	NO
268	hanmugapriya	22	G3P2L2	162/112	2+	Severe pre-eclam	37w+2d	1.7L	Term	LSCS	HELLP SYNDROME
269	Ragavi	21	PRIMI	172/110	2+	Severe pre-eclam	36wks	2.2L	Term	LSCS	NO
270	Poonguzhali	19	PRIMI	178/112	2+	Severe pre-eclam	34wks	2L	preterm	Forceps	NO
271	Issakiammal	19	G2A1	164/112	1+	Severe pre-eclam	37wks	1.8L	Term	Vaginal	NO
272	Panchavarnam	20	G2P1L1	166/114	2+	Severe pre-eclam	37w+5d	2.2L	Term	Vaginal	NO
273	Pavithra	24	PRIMI	174/122	2+	Severe pre-eclam	36wks	1.1L	preterm	LSCS	NO
274	Vidhya	25	PRIMI	170/112	2+	Severe pre-eclam	34wks	2L	Preterm	Vaginal	NO
275	Yamuna	28	PRIMI	160/116	1+	Severe pre-eclam	32wks	1.2L	Preterm	LSCS	HELLP SYNDROME
276	Divya	28	G2P1L1	172/120	2+	Severe pre-eclam	37wks	1.3L	Term	LSCS	PPH-MEDICALLY MANAG
277	Ramya	31	PRIMI	168/122	2+	Severe pre-eclam	33wks	#####	preterm	LSCS	NO
278	Darshini	30	G2P1L1	170/116	2+	Severe pre-eclam	38wks	1L	Term	LSCS	PPH-MEDICALLY MANAG
279	Geetha	19	PRIMI	170/110	2+	Severe pre-eclam	37wks	1.8L	Term	Vaginal	NO
280	Sindhu	20	PRIMI	162/112	2+	Severe pre-eclam	37w+4d	1.9L	Term	LSCS	NO

281	Priya	19	PRIMI	146/98	1+	Severe pre-eclam	37w+2d	2L	Term	LSCS	NO
282	Nirmala	22	PRIMI	162/112	2+	Severe pre-eclam	35wks	1.8L	preterm	Vaginal	HELLP SYNDROME
283	Mumtaz	26	PRIMI	160/118	2+	Severe pre-eclam	36wks	1.6L	Preterm	LSCS	HELLP SYNDROME
284	Tamilrani	27	PRIMI	172/120	2+	Severe pre-eclam	37wks	1.8L	Term	LSCS	PPH-MEDICALLY MANAG
285	Vinupriya	28	G2A1	142/100	2+	Mild pre-eclamps	38wks	2.2L	Term	Vaginal	NO
286	Anitha	29	G2P1L1	148/106	1+	Mild pre-eclamps	38wks	1.4L	Term	Vaginal	PPH-MEDICALLY MANAG
287	Uma	20	G2A1	140/100	1+	Mild pre-eclamps	35wks	2.5L	preterm	Vaginal	NO
288	Vanishree	21	PRIMI	140/100	1+	Mild pre-eclamps	38wks	2.2L	Term	Vaginal	NO
289	Nancy	22	PRIMI	146/92	1+	Mild pre-eclamps	35wks	2.1L	Preterm	Vaginal	HELLP SYNDROME
290	Suganya	28	G3P2L2	140/100	1+	Mild pre-eclamps	36wks	#####	Preterm	LSCS	NO
291	Priya	28	PRIMI	158/106	1+	Eclampsia	37w+4d	1.2L	Term	Vaginal	NO
292	Charmila	21	PRIMI	140/120	1+	Eclampsia	36wks	#####	Stillbirth	LSCS	HELLP SYNDROME
293	Saroja	20	G2P1L1	160/128	2+	Eclampsia	35wks	1L	Preterm	LSCS	PPH-MEDICALLY MANAG
294	Shobana	24	PRIMI	150/108	1+	Eclampsia	36wks	2.2L	IUD	Vaginal	NO
295	Vaijayanthi	26	G2A1	140/90	2+	Eclampsia	36wks	1.1L	Preterm	LSCS	HELLP SYNDROME
296	Rani	27	PRIMI	140/110	2+	Eclampsia	37wks	1.2L	Term	LSCS	NO
297	Sandhya	28	G3P2L2	168/112	2+	Eclampsia	34wks	1.5L	Preterm	Vaginal	HELLP SYNDROME
298	Roja	31	G3P2L1	172/120	2+	Eclampsia	36wks	2L	Preterm	LSCS	NO
299	Aparna	19	G3A2	170/120	2+	Eclampsia	36wks	1.2L	Preterm	LSCS	HELLP SYNDROME
300	Bakyam	24	PRIMI	172/118	2+	Eclampsia	34wks	1L	Preterm	LSCS	NO

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