"ASSOCIATION OF SERUM LACTATE CONCENTRATION WITH SEVERITY OF PRE-ECLAMPSIA AND MATERNAL COMPLICATIONS: AN OBSERVATIONAL STUDY"

By:

REGISTRATION NO. BJ0119004

Dissertation

Submitted to the
KLE Academy of Higher Education & Research
Belagavi, Karnataka
In partial fulfillment
of the requirements for the degree of

MASTER OF SURGERY (M.S.)
in
OBSTETRICS AND GYNAECOLOGY

J. N. MEDICAL COLLEGE, NEHRU NAGAR
BELAGAVI-590010

APRIL 2022

KLE ACADEMY OF HIGHER EDUCATION AND RESEARCH, BELAGAVI, KARNATAKA

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LIST OF ABBREVIATIONS

PE	:	Pre-eclampsia
PIH	:	Pregnancy induced hypertension
HELLP	:	Hemolysis-Elevated liver enzymes-Low Platelet
DIC	:	Disseminated Coagulopathy
PRES	:	Posterior reversible encephalopathy syndrome
PAPP-A	:	Pregnancy-associated plasma protein A
PlGF	:	Placental growth factor
sFlt-1	:	Soluble Fms-like tyrosine kinase 1
LDH	:	Lactate dehydrogenase
VEGF	:	Vascular endothelial growth factor
sENG	:	Soluble Endoglin
HIF	:	Hypoxia-inducible transcription factors
ROS	:	Reactive oxygen species
AKI	:	Acute kidney injury
CKD	:	Chronic kidney disease
TNF-α	:	Tumor necrosis factor
TLR-4	:	Toll like receptor-4
COT	:	Clot observation test

ABSTRACT

Introduction

Despite development of various prediction models, pre-eclampsia continues to be a major contributor to maternal mortality and morbidity. Assuming that raised serum lactate is a marker of tissue hypoperfusion and hypoxia as associated with preeclampsia, it can be used as a successful biomarker in prediction of maternal complications and aid in successful management of preeclampsia and thus improve maternal and perinatal outcome.

Aims & Objectives

- To evaluate the association of serum lactate concentration with severity of pre-eclampsia and maternal complications.

Materials & Methods

This study was conducted in KLE's Dr Prabhakar Kore Hospital, Belagavi.

Study design – Observational study

Study period – January 2020 to September 2021

Sample size – 170

- All patients fulfilling the criteria for pre-eclampsia and eclampsia were included in the study. Baseline characteristics were noted and serum lactate was collected at the time of admission. Serum lactate >27 mg/dl was considered high. Participants were evaluated for development of complications – Stroke, PRES, HELLP, DIC, Abruption, Pulmonary edema and Acute kidney injury.

Results

170 participants were analyzed in the study. The mean lactate level observed was 40.5 ± 24.8 mg/dl. The serum lactate level was significantly higher in eclampsia than preeclampsia (p=0.03). Maternal complications were noted in 50% of the study participants with 21% having CNS complications. Of these, 77.7% women had lactate >27 mg/dl, although not found statistically significant. Statistical significance was found only when associated with HELLP with 80% having lactate >27 mg/dl (p=0.03).

Conclusion

Serum lactate is a major predictor of severity of pre-eclampsia. In case of pre-eclamptic patients presenting with maternal complications, serum lactate levels were elevated although no statistical significance could be established.

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1. INTRODUCTION

Hypertensive disorders in pregnancy can be broadly classified into those occurring before 20 weeks of gestation like white coat, masked or chronic hypertension and those occurring after 20 weeks, which includes transient gestational hypertension, gestational hypertension and pre-eclampsia[1]. Pre-eclampsia is a major complication in pregnancy, which is responsible for maternal as well as foetal morbidity and mortality. Pre-eclampsia accounts for 44,000 deaths a year and is solely responsible for 14% of the deaths associated with pregnancy, rendering it the 2nd most common reason behind pregnancy related deaths[2].

It can be defined as a sudden emergence of hypertension, proteinuria and other organ complications after 20 weeks of pregnancy in normotensive women prior[3]. It is characterised by systolic blood pressure of ≥ 140 mmHg and/or diastolic ≥ 90 mmHg, along with proteinuria, which is diagnosed by presence of 300 mg of protein in 24 h urine collection or presence of protein/creatinine ratio ≥ 0.3 [4].

Eclampsia is characterized as pre-eclampsia with the onset of seizures or coma during pregnancy or after delivery that is not caused by other neurologic disorders[5]. Eclampsia has a recurrence rate of around 10% if it is not treated[6,7].

The pathology of this disease comprises of both maternal and placental dysfunction. This disease encompasses two stages, the first one characterised by abnormal placentation in early pregnancy and the 2nd stage which develops in 2nd or 3rd trimester and involves release of angiogenic factors. Preeclampsia is characterized by failure of cytotrophoblasts to transform thereby affecting the development of spiral arteries between 8 and 16 weeks of pregnancy, leading to vasoconstriction and placental ischemia, resulting in angiogenic imbalance and impaired vasodilation[8-11].

Determination of a biomarker that can predict development of maternal complications and progression, could aid in successful management. The most effective include the ones associated with placental function. These include pregnancy-associated plasma protein A (PAPP-A), placental growth factor (PlGF), and sFMS-like tyrosine kinase 1 (sFlt-1)[12–14] . High levels of soluble FMS-like tyrosine kinase-1 in the blood, as well as low levels of free placental growth factor and free vascular endothelial growth factor are present in abnormal concentrations not only during clinical preeclampsia, but also several weeks before clinical symptoms appear. Hence these angiogenic proteins can be used for prediction of pre-eclampsia associated complications[15].

Exposure to maternal hypoxia early in pregnancy significantly affects placentation by altering trophoblast proliferation. Hypoxia has been proved to play an important role in the early stage of preeclampsia, supported by the fact that, hypoxia-inducible transcription factors (TFs) are upregulated, and hypoxia-related gene signatures are present[16]. Hypoxia in normal tissues is accompanied by an increase in glycolysis, leading to an increase in lactate formation. Lactate is a product of pyruvate by the action of lactate dehydrogenase enzyme (LDH) during glycolysis. Hypoxia in pre-eclampsia is associated with high activity and gene expression of lactate dehydrogenase in placentas. Since elevated levels of LDH indicate cellular damage and dysregulation and are also shown to increase in HELLP syndrome, it can be used as a marker to indicate the seriousness of the disease, associated complications, and foetal outcome[17]. As the end product of LDH activity is lactate, measurement of lactate concentration could also prove beneficial in predicting complications associated with pre-eclampsia.

As placenta is a highly vascular organ, increased glycolysis resulting in high glucose consumption and production of lactate is observed normally. Under aerobic conditions the rate of production and consumption of lactate is constant. 70% of lactate is metabolized by the liver and the rest by the kidneys. However, in pre-eclampsia, hypoxia increases the rate of glycolysis resulting in an increased concentration of lactate for ATP production. Moreover, it has been reported that the activity and gene expression of LDH enzyme in placenta increases in pre-eclampsia, which leads to higher concentration of lactate[18]. Lactate levels are differentiated into low (0-2mmol/L), intermediate (2.1-3.9mmol/L) and high (0.4mmol/L)[19].

With the supposition that lactate levels indicate tissue hypoxia and hypoperfusion in pre-eclampsia, it can be used as a reliable indicator for predicting the severity or pre-eclampsia. The current study aims at evaluating association between serum lactate concentration and severity of pre-eclampsia as well as risk of developing complications.

2. OBJECTIVE

Researchers have established an increase in lactate levels in early stages of pre-eclampsia, measurement of lactate levels can act as a predictor of maternal complications. With the supposition that lactate levels indicate tissue hypoxia and hypoperfusion in preeclampsia, it can be used as a reliable indicator for predicting the severity of this condition.

OBJECTIVE OF THE STUDY

Primary objective - To evaluate the association of serum lactate concentration with severity of pre-eclampsia and maternal complications.

Secondary objective - To assess the maternal and perinatal outcomes associated with re-eclampsia and eclampsia.

3. REVIEW OF LITERATURE

Hypertensive disorders are a common complication associated with pregnancy which predisposes women and fetus to a high risk of subsequent complications and long-term sequelae. These include chronic hypertension, gestational hypertension, pre-eclampsia-eclampsia and chronic hypertension with superimposed pre-eclampsia[8].

Hypertension manifesting before 20 weeks comprises of:

- essential chronic or secondary arterial hypertension
- white coat hypertension
- masked hypertension

Hypertension manifesting beyond 20 weeks includes:

- transient gestational hypertension
- gestational hypertension
- pre-eclampsia

Chronic hypertension can be defined as high blood pressure before pregnancy, within first 20 weeks of pregnancy, or which is present even during 12-weeks postpartum. Chronic hypertension results in complications in around 5% of all pregnancies[20].

Gestational hypertension develops after 20 weeks of pregnancy, in the absence of proteinuria or biochemical or haematological abnormalities. It usually has a favourable outcome with a quarter progressing to pre-eclampsia[21].

Preeclampsia is characterised by the presence of hypertension after 20 weeks of gestation, complicated by proteinuria, acute kidney injury (AKI), liver dysfunction, neuro-dysfunction, hemolysis or thrombocytopenia, or fetal growth restriction[21].

3.1 DEFINITION OF PREECLAMPSIA AND ECLAMPSIA

Recent definitions by International Society for the Study of Hypertension in Pregnancy(ISSHP) and American College of Obstetricians and Gynecologists (ACOG) state that preeclampsia is a syndrome characterised by presence of de novo hypertension and the coexistence of one or more of : proteinuria, renal insufficiency characterised by creatinine more than 90 mmol/L, hepatic involvement denoted by presence of elevated transaminases and neurological complications which include eclampsia, altered mental state, blindness, stroke, hyperreflexia with clonus, severe headaches or persistent visual scotomata, hhematological complications like thrombocytopenia which is characterised by platelet count <150 000/μL, DIC, utero-placental abnormalities like intra-uterine growth restriction[22-23].

Clinical features of Preeclampsia are presented in Table 2-1[24].

Table 3-1. Clinical Features of Preeclampsia

- Elevated blood pressure (systolic ≥ 160 mm Hg, diastolic ≥ 110 mm Hg)
- Elevated creatinine level (> 1.1 mg per dL or ≥ 2 times increase from baseline)
- Hepatic dysfunction (≥ 2 fold increase in transaminase level) or right upper-quadrant or epigastric pain
- New-onset headache or visual disturbances
- Platelet count < 100×10^9 per L
- Pulmonary edema

Preeclampsia is also responsible for around 12 to 25% of fetal growth restriction and accounts for 15 to 20% of all preterm births which in turn leads to neonatal mortality and long-term morbidity such as stroke, coronary heart disease, and metabolic syndrome in adulthood. Preeclampsia and eclampsia are also linked to one-quarter of stillbirths and neonatal mortality in underdeveloped nations[25-26].

Eclampsia is an obstetric emergency characterized as the onset of convulsions in the antepartum, intrapartum or postpartum period in conjunction with signs and symptoms of pre-eclampsia. In underdeveloped nations a prevalence of 2.3 and 6 percent for every 10,000 births has been reported[7]. Seizures before the 20 weeks of gestation have been reported in cases of prenatal trophoblastic illness[27].

Eclampsia is usually accompanied by central nervous system symptoms such as headache (80%) and visual changes (45%). 15% of women with eclampsia have a diastolic BP less than 90 mm Hg. Eclamptic seizures are 60- to 90-second seizures followed by postictal confusion, agitation, or combativeness. They may also result in hypoxia-related bradycardia in the foetus[24].

3.2. INCIDENCE OF PREECLAMPSIA

Preeclampsia and eclampsia cause over 50,000 maternal deaths worldwide per year. Incidence of preeclampsia varied between 2% and 10% of pregnancies worldwide. According to WHO, preeclampsia is seven times more common in developing countries (2.8 percent of live births) than in developed (0.4 percent)[28].

According to India's third National Family Health Survey (NFHS-3,2005-06), the incidence of preeclampsia and eclampsia in India is supposed to be significantly more (28% and 7.4–11.3% respectively) in comparison to its incidence worldwide. The number of preterm births reported in India is the highest in the world and

preeclampsia (36%), chronic hypertension (5%), eclampsia (4.8%), and gestational hypertension (4.8%) are the most common risk factors for preterm births in India. Hence, India is currently aiming at providing quality antenatal care to pregnant women with a special emphasis on preeclampsia[26,29].

3.3. RISK FACTORS FOR PREECLAMPSIA

Several factors are involved in the development of Preeclampsia during pregnancy which include the following:

1. **History of preeclampsia**: Preeclampsia in the second trimester is associated with a recurrence rate of 25 to 65 percent. The incidence is 5 to 7% in women who did not have history of pre-eclampsia in first pregnancy.
2. **Pre-existing medical conditions**: Pre-gestational diabetes, chronic hypertension account for 5 to 10 percent of preeclampsia cases.
3. **Autoimmune disorders:** Some autoimmune diseases like systemic lupus erythematosus and antiphospholipid syndrome elevate the risk for preeclampsia.
4. **Obesity or higher body mass index**: With each increase in BMI by 5 to 7 kg/m2 before pregnancy, the risk of developing pre-eclampsia doubles. Overweight and obesity account for over 40 percent of preeclampsia worldwide.
5. **Chronic kidney disease**: Incidence is as high as 40 to 60 percent in women with advanced chronic renal disease.
6. **Multifetal pregnancy**: Rates above 20 percent are observed in multiple gestations.
7. **Nulliparity**

8. **Family history:** of hypertension in 1st degree relatives increases the risk.
9. **Prior pregnancy complications especially abnormalities in placentation**: Fetal growth restriction, abruption and stillbirth are outputs of placental insufficiency and pose as risk factors for occurrence of preeclampsia.
10. **Higher maternal age**: Owing to associated obesity, diabetes mellitus, and chronic hypertension with increasing age which in turn influence the development of preeclampsia[30].
11. **Genetic factors:** The protein product of s-FLT1 (FMS-like tyrosine kinase 1) gene is a well-known causative factor in preeclampsia and is associated with an increased incidence in late-onset preeclampsia. It shows effect only on fetal and not maternal—genome[8]. Pre-eclampsia heritability is believed to be around 55%, with maternal and fetal genetic factors (30–35% and 20%, respectively) to risk[3].

The National Institute for Health and Clinical Excellence (NICE) proposed that presence of two moderate or one high risk factor should involve administration of prophylactic measures to pregnant women[1].

The factors termed as high risk include:

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

Factors termed as moderate risk include:

- Primiparity
- 40 years or older women
- Delivery gap greater than 10 years;
- Body mass index (BMI) higher than 35 kg/m^2
- A family history of preeclampsia
- Multiple pregnancies

American College of Obstetricians and Gynecologists (ACOG), also proposed the same risk factors as proposed by NICE, the difference being the BMI of 30 kg/m^2. Moreover it categorised all the factors as high risk[1].

This concept allowed a detection rate of 37% and 28.9% of cases in early and late preeclampsia, respectively. The highest predictability was observed with previous history of preeclampsia.

The risk factors along with a relative risk index where the risk factor is relative to pregnant women without any risk factor, have been presented in the following Table 2-2.

Table 3-2. Risks associated with preeclampsia[24]

RISK FACTOR	RELATIVE RISK
Anti-phospholipid antibodies	10
Preeclampsia in a previous pregnancy (particularly if severe or before 32 weeks' gestation)	7
Diabetes mellitus (pre-existing)	3
Family history of preeclampsia (first-generation relative)	3
Multiple gestation	3
Nulliparity	3
Elevated body mass index	2
Maternal age > 40 years	1.6

3.4. PATHOGENESIS OF PREECLAMPSIA

Clinical observations like the fact that symptoms and abnormalities associated with preeclampsia usually recede after delivery, and that incidence of preeclampsia increases in multiple pregnancies as well as in cases of hydatiform moles provide undeniable proof of placental origin of preeclampsia, but the etiology of preeclampsia is still not crystal clear. Theories explaining the pathogenesis of preeclampsia include the following[26] :

Utero-placental origin. According to this hypothesis, trophoblast invasion is limited and the transformation of maternal spiral arteries fails, resulting in low placental perfusion, which leads to preeclampsia symptoms. The causes of abnormal placentation in women with preeclampsia are unknown, but they could include faulty extravillous trophoblast (EVT) differentiation with reduced invasive properties, as

well as changes in maternal decidual tissues that control cytokines/growth factors-mediated trophoblast behaviour. Reduced/inhibited maternal nitric oxide generation may lead to endothelial dysfunction or hinder implantation, resulting in cytotrophoblast differentiation defects. Furthermore, elevated hypoxic environment, oxidative stress, endothelial dysfunction, and abnormal systemic inflammation are known to cause decreased trophoblastic invasion and poor placental perfusion.

Angiogenic origin: The plasma from preeclamptic women affects the capacity of pre-constricted vasculature to relax, simulating preeclamptic vessels. Furthermore, endothelial cell dysfunction caused by an ischemic placenta during preeclampsia is linked to a shift in the balance of circulating angiogenic and anti-angiogenic growth factors, which can lead to hypertension. During preeclampsia, circulating levels of angiogenesis regulators like vascular endothelial growth factor (VEGF) and placental growth factor (PLGF) are lowered, which may be responsible for several of the clinical symptoms. By binding to VEGFR2 and VEGFR1 (also known as Flt-1) respectively, VEGF and PLGF stimulate angiogenesis, but soluble Flt-1 (sFlt-1) suppresses angiogenesis[31].

Immunogenic origin: According to this theory, inability of the maternal immune system to identify the feto-placenta may induce preeclampsia by causing defective vascular remodeling leading to hypertension and proteinuria. Preeclamptic women showed lower levels of histocompatibility antigen (HLA-G, E)[32]. Preeclampsia is thought to be a result of the ongoing immune-mediated inflammatory changes that occur throughout pregnancy, and it's possible that women who have a strong immune response to paternal HLA antigens are more likely to suffer endothelium damage, which precedes preeclampsia[33]. Several immune-related risk factors in pregnant women, including as pre-existing autoimmune illness, autoimmune antibodies to

angiotensin II type I (AT1) receptors and phospholipids, etc. enhance the risk of preeclampsia.

Genetic predisposition: Even while both hereditary and environmental variables enhance the risk of preeclampsia, having preeclampsia in first degree relatives raises a woman's risk of preeclampsia by 2–4 times. It's possible that genetic variables play a role in the angiogenic imbalance seen in preeclampsia patients.

Pathogenesis of preeclampsia is broadly divided into two phases:

- Abnormal placentation which occurs in the first trimester - which results in generation of an unidentified signal due to a combination of factors like endothelial dysfunction, placental perfusion deficiency, defective implantation, oxidative stress or high placental mass
- Development of maternal response, which develops in the 2^{nd} and 3^{rd} trimesters and results in clinical manifestation of preeclampsia characterized by hypertension and proteinuria[26]

The pathogenesis of preeclampsia is presented in Figure 2-1.

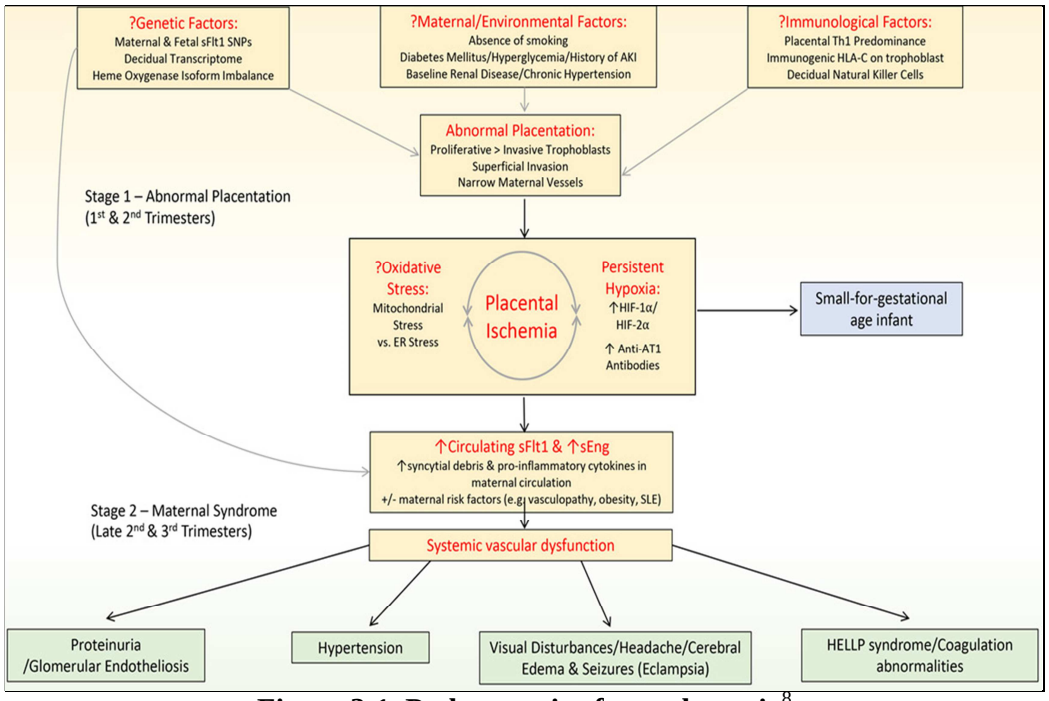

Figure 3-1. Pathogenesis of preeclampsia[8]

<u>Abnormal placentation</u>:

Faulty placentation and failure in transformation of uterine spiral arteries has emerged as a major explanation for the pathogenesis of pre-eclampsia. Proper fetal development requires higher uterine blood flow, which is achieved by remodelling of spiral arteries, consisting of 4 steps facilitated by a trophoblastic invasion of their walls. First the decidua is invaded, followed by migration of intra-arterial trophoblast and intramural invasion of the arteries with which the middle (muscular) layer is lost and replaced by connective tissue and fibrinoid material. The last stage is vascular reendothelialization and other maternal modifications caused by the procedure. These vessels develop a higher mean diameter than that seen in non-pregnant women and thereby provide intervillous space with adequate blood supply for maintenance of pregnancy. The radial and arching arteries have higher blood pressure in their walls as

a result of increased blood flow, which acts as a stressor and eventually causes the endothelium to secrete nitric oxide, resulting in overall uterine artery vasodilation. The spiral arteries are more prone to remodeling in the core region of the placenta[34-36].

However, in preeclampsia failure of cytotrophoblasts to transform from the proliferative epithelium to the invasive endothelial subtype, results in inadequate spiral artery remodelling and placental ischemia. Placental ischemia in humans is observed by using uterine artery Doppler tests. These showed robust systolic and diastolic uterine arterial flows during normal pregnancy; however, women with preeclampsia had severe diastolic flow limitation with a distinct notch in the waveform[8].

Additional evidence in support of the ischaemic placenta hypothesis was given by the observation that changes in the spiral arteries in preeclampsia were limited to the decidua, as compared to a proximal extension into the myometrium in normal pregnancies. Moreover, the average diameter of the spiral arteries was only 200 μm in pre-eclamptic samples when compared to 500 μm in the vessels of placenta from normotensive pregnancies[3].

Causes behind abnormal placentation:

Hypoxia: This was established by the upregulation of hypoxia-inducible transcription factors (TFs) and hypoxia-related gene signatures in the placenta. HIF1 and HIF2 are two proteins that are produced by the same oxygen-sensing mechanism and control the expression of hypoxia-induced genes such erythropoietin, vascular endothelial growth factor (VEGF), and NO synthase. When circulation and hence oxygenation to the fetus increase, HIF1 expression in human placentae increases in the first trimester and declines about 9 weeks. HIF1 levels that remain consistently high may signal

placental stress and the onset of pre-eclampsia. Pre-eclamptic placentas have been demonstrated to overexpress HIF1 and HIF2 and fail to downregulate their expression when exposed to oxygen[3,37].

Oxidative stress: In pre-eclampsia, antioxidant and pro-oxidant mechanisms are not balanced, which is proposed to be a result of defective spiral artery remodeling (which causes repetitive ischemia–reperfusion injuries)[38]. Pre-eclamptic placentas have an imbalance of reactive oxygen species (ROS)-generating enzymes and antioxidants at the molecular level. ROS inhibit the Wnt/-catenin signaling pathway, which enhances trophoblast invasiveness. The transcription of antiangiogenic factorslike SLFT1 may be aided by oxidative stress[39-41].

Role of Heme oxygenase and other enzymes: Heme oxygenase (HO), the heme degradation enzyme, has been shown to exert a significant effect on vascular function of mother and foetus, and in the development of placental and its functioning. HO has three isoforms, with HO-2 being involved in spiral artery invasion and HO-1 being strongly expressed in noninvasive trophoblastic phenotypes. In the reduced uterine perfusion pressure (RUPP) rat model, CoPP (cobalt protoporphyrin) an inducer of HO-1, reduced blood pressure and caused a pro-angiogenic shift in the VEGF (vascular endothelial growth factor) gene[8].

Maternal syndrome: The 2nd phase in the pathogenesis of preeclampsia caused by imbalance between proangiogenic and antiangiogenic factors, with formation of higher antiangiogenic factors leads to extensive maternal repercussions[42].

Imbalance in Circulating Angiogenic Factors: Increased levels of the anti-angiogenic protein sFLT1 in placentas preeclampsia patients have been observed. sFLT1 shows antiangiogenic effects by inhibiting activity of proangiogenic proteins VEGF and PlGF. VEGF is responsible for endothelial cell function, and PlGF is significant in angiogenesis and selectively binds to VEGFR1/sFLT1. Soluble endoglin (sENG), an endogenous TGF-1 (transforming growth factor 1) inhibitor, is another antiangiogenic protein that has been intensively researched in pre-eclampsia. sENG is raised in preeclamptic women's sera 2 months before the start of clinical indications of preeclampsia, corresponds with illness severity, and decreases after delivery[8].

The role of sFLT1 and sENG is shown in Figure 2-2[8].

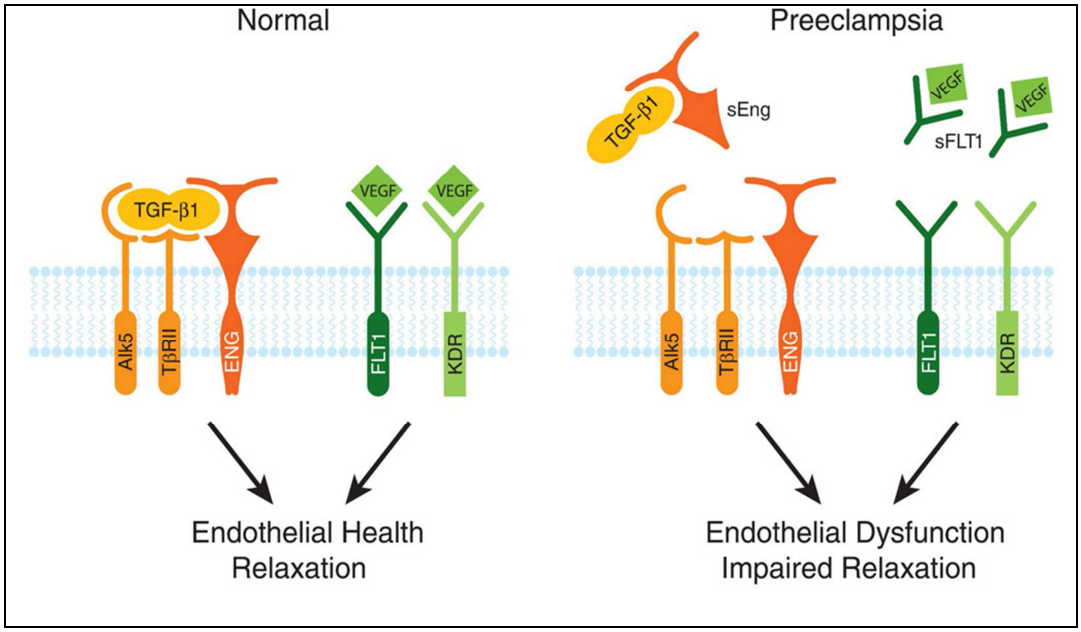

Figure 3-2: Endothelial dysfunction resulting due to sFLT-1 and sENG

Hypertension: Anti-angiogenic factors and agonistic autoantibodies that bind to the angiotensin II type 1 receptor (AT1-AAs) may produce the hypertension seen in preeclampsia, rather than the renin–angiotensin–aldosterone system (RAAS). These autoantibodies are produced in women with pre-eclampsia and are demonstrated to be

responsible for causing proteinuria, hypertension and glomerular endotheliosis. AT1-AA levels do not entirely decrease after delivery, which might contribute to the elevated cardiovascular risk reported in women who have had pre-eclampsia. Antioxidant NO, which affects the effects of PlGF and VEGF in vitro also plays a potent role in preeclampsia. Women with pre-eclampsia had lower NO levels in their blood. Additionally, an increase in Endothelin 1 (ET1) – a potent vasoconstrictor and a decrease in H2S which is involved in angiogenesis and vasodilation, is observed in pre-eclamspia[43-45].

Obesity, insulin resistance and other factors: Increased hyperinsulinemia, aberrant placental glycogen accumulation, and altered placental insulin signaling are all linked to pre-eclampsia. Insulin resistance appears to work in concert with reduced angiogenic factors to enhance the risk of pre-eclampsia[46].

2.5 BIOMARKERS FOR PREECLAMPSIA DIAGNOSIS

Due to the unavailability of adequate screening methods as well as the severe outcome of this condition, all women with risk of preeclampsia have to go through intensive testing, involving days long hospitalizations. The Fetal Medicine Foundation algorithm-based screening method, which incorporates maternal factors, mean arterial pressure, uterine-artery pulsatility index, and PlGF was found to be superior to the normally used screening methods recommended by National Institute for Health and Care Excellence and ACOG.

The ASPRE trial (aspirin for evidence-based preeclampsia prevention) proved the success of such a screening method using PAPP-A (Pregnancy associated plasma protein A), and involved screening of women in the first trimester followed by administration of 150 mg of aspirin or placebo daily till 36 weeks gestation to high-risk women. It resulted in a decreased incidence of preterm preeclampsia than placebo, and identification rate was 76.7% (138/180)—43.1% for term preeclampsia—with a false positive rate of 9.1%. This proves the effectiveness of early screening with plasma biomarkers and imaging studies in prevention of disease[47].

Angiogenic factors have established their efficacy as biomarkers in pre-eclampsia as imbalance of these factors plays an important role in preeclampsia. The levels of PlGF, sFLT1 and sENG, as well as the ratios of sFLT1 to PlGF and PlGF to sENG, vary for women with preeclampsia and normal pregnancies. Changes in sENG and sFLT1 levels in the first and second trimesters predicted preterm pre-eclampsia, whereas third-trimester levels identified women at risk of severe late disease and stillbirth. PlGF and sFLT1 levels have been found to change as early as 6–10 weeks

before clinical pre-eclampsia and these changes occurred sooner in women who had preterm pre-eclampsia[3].

Angiogenic biomarkers may also be useful in distinguishing pre-eclampsia from other diseases that manifest in pregnancy with similar signs and symptoms, such as CKD, gestational thrombocytopenia, and chronic hypertension, and thus may be able to replace invasive renal biopsy for diagnostic purposes[48]. In a largescale study conducted in UK, the plasma sFLT1:PlGF ratio assessed at 28 weeks showed a positive predictive value of 32% for preterm pre-eclampsia[49].

Hyper-uricemia also serves as a biomarker for predicting development of preeclampsia following gestational or chronic hypertension. Evidence about the contribution of uric acid to the pathogenesis of preeclampsia is mixed, though general consensus suggests that levels are elevated secondary to renal injury and decreased excretion[8].

The various screening tests which have been evaluated in the literature for diagnosing preeclampsia, have been extensively reviewed and presented in Figure 2-3[11]. These are grouped into four major categories.

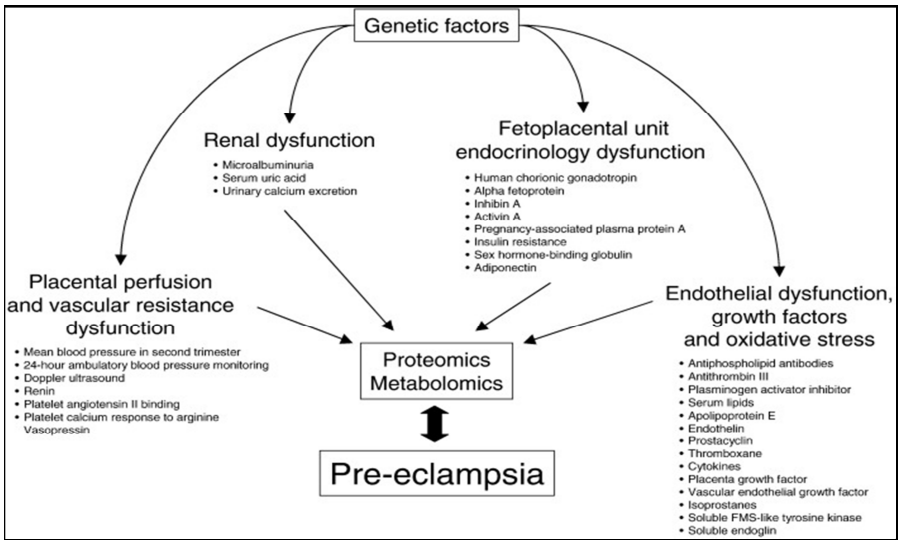

Figure 3-3. Biomarkers in preeclampsia[11]

3.6 MANAGEMENT OF PREECLAMPSIA

Depending on the underlying pathogenic mechanism, several strategies have been developed by researchers, which are presented in Table 2-3[50].

Table 3-3. Management techniques based on the pathology of preeclampsia

Pathology	**Management techniques**
1) Oxidative stress	Antioxidants like Silymarin
2) Micro-emboli in the small vascular bed	Aspirin, low molecular weight heparin, antithrombin infusion
3) Vasoconstriction	Vasodilators (sildenafil citrate), Nitric oxide donors (glycerol trinitrate, isosorbide mononitrate), nitric oxide precursors (L-arginine)
4) Increase in placental sFlt-1 and endoglin	statins for inducing heme-oxygenase pathway, gelsolin, esomeprazole to stop syncytio-trophoblast vesicle shedding, metformin for inhibition of HIF-1α, dextran sulfate apheresis for removal of circulating sFlt-1
5) Decrease in circulating VEGF/PLGF	Replacement of PLGF or VGEF
6) Systemic inflammatory response (excessive TNF-α, TLR4 receptors)	TNF-α antagonists, aspirin hydroxy-chloroquine, apolipoprotein. B) Anti-TLR4 receptors: Curcumin, Vitamin D

The treatment methods are intended to slow down the pathological process of preeclampsia. Along with well-established treatment methods for hypertension, aspirin and control of blood sugar and renal function; new techniques are also required. Currently used medications which have been effective in prolonging

pregnancy include Sildenafil as a vasodilator, esomeprazole as an inhibitor of vesicle shedding, metformin as an inhibitor of HIF-1α, hydroxy-chloroquine as an antagonist of TNF-α , and curcumin as an anti-TLR4 receptors. Other techniques include recombinant placental growth factor injections and dextran sulphate apheresis which due to their invasive nature are only employed in severe cases[50].

3.7 LACTATE LEVELS IN DIAGNOSIS OF PREECLAMPSIA

Glycolysis comprises of the initial step in metabolism of glucose resulting in the formation of pyruvate. Pyruvate in turn can participate in any one of these metabolic pathways. It results in energy production by taking part in the tricarboxylic acid pathway or be transformed to lactate via lactate dehydrogenase. Pyruvate acts as a substrate in gluconeogenesis for glucose formation, or undergoes transamination to alanine. Lactate formation from pyruvate results only when pyruvate concentration is high. Conversion of pyruvate to lactate is favored during hypoxic tissue conditions and several other clinically relevant conditions Lactate is generated by most tissues in the body and is created from pyruvate by lactate dehydrogenase (LDH) in the final phase of glycolysis as a substrate for gluconeogenesis. Lactate concentrations in the blood are less than 2 mmol/L, and it is continually created and consumed in typical aerobic circumstances[19]. Lactate is produced at a rate of roughly 1400 mmol per day, and though lactate may be produced by any tissue, it is principally produced by skeletal muscle (25%), skin (25%), brain (20%), intestine (10%), and red blood cells (20%).

In hypoxic conditions or when the rate of cellular glycolysis exceeds the mitochondrial capacity, pyruvate is converted to lactate for adenosine triphosphate synthesis, resulting in an increase in plasma lactate levels. Hypoperfusion,

mitochondrial malfunction, and the existence of a hypermetabolic state are all factors that contribute to increased lactate levels. The majority of situations that cause excessive lactate generation and decreased lactate clearance are pathologic and reflect tissue hypoxia or nonhypoxic tissue damage. The use of serum lactate as a biomarker in critically unwell patients is based on this discovery[19].

Since hypoxia plays a major role in pathogenesis of preeclampsia, elevated lactate levels can be expected in preeclamptic patients, hence lactate level measurement could help in prediction of preeclampsia. Moreover, rise in activity and gene expression of lactate dehydrogenase enzyme has been noted in placentas from preeclamptic patients, thereby leading to increase in concentrations of lactate. Lactate dehydrogenase an intracellular enzyme present in lungs, kidney, liver, heart, muscles and blood cells, catalyses the interconversion of NADH and NAD+, thereby transforming pyruvate, the final product of glycolysis to lactate. Because erythrocytes lack mitochondria, glycolysis invariably results in lactate generation, whereas other tissues such as the brain, renal medulla, retina, and skin rely heavily on glycolysis and oxidize lactate. Higher Serum LDH level has been detected in preeclamptic patients in various studies[51]. Hall et al., 2000 showed a higher LDH level before delivery in early onset of PE with severe features[52] and Jaiswar et al. 2011, observed high LDH level women with preeclampsia and eclampsia[53]. Thereby proving the association of high serum LDH level severity and poor outcomes in preeclampsia patients. Similarly, Qublan et al. 2005, calculated that average LDH level was 348±76 IU/l in patients with PE without severe features and 774±69.61 IU/l in those with PE with severe features[54]. Thereby proving the relationship between serum LDH level and severity of preeclampsia. Higher LDH level indicates increase in concentration of lactate, the end product. As a result, the concept that increased circulating lactate

suggests tissue hypoperfusion is used to measure lactate as a predictive tool in PE. The goal of this study is to see the association of serum lactate concentrations upon admission and the severity of PE in women.

4. MATERIALS AND METHODS

4.1 STUDY DESIGN

The present study was a hospital based observational study to evaluate the association of serum lactate concentration with severity of pre-eclampsia and maternal complications. This study was conducted at KAHER's Dr. Prabhakar Kore Hospital, Belagavi for a period of 12 months. Data and samples were obtained from pregnant mothers and neonates who had been informed about the study's purpose. Patients who expressed an interest in taking part in the trial were enrolled after signing a written informed consent form.

4.2 STUDY SETTING

The study was conducted at the Department of Obstetrics and Gynecology of KAHER's Dr. Prabhakar Kore Hospital, Belagavi, Karnataka. The hospital is a clinical training facility that provides free health care to the underprivileged in basic specialties. KAHER's Dr. Prabhakar Kore Hospital is recognized by the Medical Council of India, and Government of Goa along with industries in Maharashtra and Karnataka.

4.3 STUDY PERIOD

The study was conducted for a period of 12 months (from January, 2020 to December, 2020). The study period included enrolment of participants, data collection, analysis and reporting.

4.4 STUDY POPULATION

The study population consisted of antenatal women diagnosed with pre-eclampsia and eclampsia presented to the labour room at the Department of Obstetrics and Gynecology, KAHER'S Dr. Prabhakar Kore Hospital, Belagavi during the study period fulfilling the inclusion criteria and consenting to participate in the study.

4.5 SAMPLE SIZE

Sample size was obtained by the formula:

$n = Z^2pq/d^2$

Where:

n = sample size

Z = 1.96 corresponding to 95% confidence interval

p = proportion of participants (37% for this study, based on Peguero et al.[2]).

q= 100-p

d = margin of error set at 7.4%

The minimum sample size was calculated as follows: 54.76

$n = 1.96 \times 1.96 \times 37 \times 63 / (7.4)^2$

=163.52, rounded off to 164

Hence, the minimum sample size was taken to be 164.

4.6 SAMPLING METHODS

Universal sampling method was adopted for this study.

4.7 SELECTION CRITERIA

4.7.1 Inclusion criteria:

Antenatal women with pre-eclampsia or eclampsia, as per WHO guidelines, presenting to the labour ward of Department of Obstetrics and Gynecology at KAHER's Dr. Prabhakar Kore Hospital, Belagavi were included in the study.

4.7.2 Exclusion criteria:

- Patients with gestational hypertension and chronic hypertension
- Women with pre-existing renal or vascular diseases
- Women with multiple gestation
- Women not consenting to participate in the study

4.8 DATA COLLECTION AND SAMPLING TECHNIQUES

All antenatal women, with a gestational age of > 20 weeks, diagnosed with new onset hypertension were screened for the study. Pre-eclampsia without severe features was defined as a SBP ≥ 140 and/or DBP ≥ 90 mm Hg associated with proteinuria, measured by dipstick method. PE with severe features was defined as a SBP ≥ 140 and/or DBP ≥ 90 mm Hg with proteinuria with evidence of end organ damage OR SBP ≥ 160 and/or DBP ≥ 110 mm Hg in the absence of proteinuria. Participant was also classified as PE with severe features if any of the premonitory symptoms: occipital headache/visual disturbance/epigastric discomfort was present along with hypertension. Patients presenting with generalized-tonic clonic convulsions in the absence of other cause were diagnosed as eclampsia. After identifying study participant, written informed consent was obtained for enrollment in the study. Details of methods employed for sampling and investigations are as follows:

4.8.1 Socio-demographic characteristics

Data on socio-demographic like age, obstetric history and detailed history about other associated conditions were collected via interview and use of structured questionnaires prior to collection of blood samples for clinical investigations. Findings of clinical examination and subsequent systemic examination were recorded on a predesigned and pretested proforma.

4.8.2 Complaints and history of presenting complaint

Data on period of amenorrhea, last menstrual period, expected due date, duration of abdominal pain, duration of leak per vagina, duration of bleeding per vagina, perception of fetal movement, imminent signs of pre-eclampsia/eclampsia (headache, blurring of vision, epigastric discomfort), seizures (and details in case of seizures such as number of episodes, duration, loss of consciousness and lucid interval) and other associated medical history were collected by interview/examination of the study participants.

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

Menstrual history consisting of details on age of menarche, previous menstrual cycles and period of gestation were collected from the study participants.

Past medical and surgical history, family history of illnesses and personal history were also assimilated form the patient via interview.

4.8.3 Clinical examinations

(i) General physical examination

Height, weight, body mass index (BMI), pulse rate, blood pressure, pallor, icterus, pedal edema and SpO2 were noted for each of the study participants.

(ii) Systemic examination and other examinations

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

Per speculum examination for active vaginal bleeding and per vaginal examinations for consistency of cervix, position, effacement, dilatation and station were recorded.

4.8.4 Clinical diagnosis and clinical investigations

Clinical diagnosis of the patient was recorded based on the examination.

Clinical investigations included blood group/Rh typing, complete blood count, peripheral smear, urine routine examination, serological tests for Human Immunodeficiency Virus (HIV), Hepatitis B surface antigen (HBsAg), venereal disease research laboratory (VDRL) test, bedside clot observation test (COT), pregnancy induced hypertension (PIH) profile including platelet count, urea, serum creatinine, urine albumin, uric acid and LDH levels, liver function tests including liver enzymes (alanine aminotransferase [*SGPT]*), aspartate aminotransferase [*SGOT*] and *alkaline phosphatase* [ALP]) and Disseminated Intravascular Coagulation (DIC) profile including tests for D-Dimer, fibrinogen, activated partial thromboplastin time (aPTT), prothrombin time/international normalized ratio (PT/INR) and thrombin time (TT).

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

4.8.5 Management/Intervention

Details of anti-hypertensive used, MgSO4 regimen, other medications prescribed for the condition were collected via proforma.

4.8.6 Delivery related information

ata on date of delivery, mode of delivery, induction/augmentation, duration of labour, intrapartum complications, blood loss, indications for lower segment Cesarean section (LSCS) and intra-OP findings were recorded for the study participants.

The placenta was observed post-partum and information on weight of placenta, retroplacental clots, placental infusions and other features was assimilated.

4.8.7 Maternal outcomes

Information on the following peripartum complications was collected and recorded for the study participants:

- Central nervous system: Convulsions, Posterior reversible encephalopathy syndrome (PRES) and stroke
- Haematological complications:
    - DIC
- Respiratory system:
    - Respiratory distress
    - Pulmonary edema
- Hepatic dysfunction
- Renal complications
- HELLP (haemolysis, elevated liver enzymes, low platelet count) syndrome
- Abruptio placenta
- Cerebro-vascular complications

Other information related to admission to the intensive care unit (ICU), duration of hospital stay, condition at discharge, cause of death (if any), and intervention (if any) were also assimilated and recorded towards maternal outcomes.

4.8.9 Perinatal outcomes

Data on perinatal outcomes including condition at birth (intrauterine fetal demise [IUFD], stillbirth), term/pre-term birth, sex, weight, APGAR score at birth, admission in neonatal ICU (NICU), indication for NICU admission, condition of baby at discharge were collected and recorded.

4.9 METHOD OF ESTIMATION OF SERUM LACTATE

Serum lactate was estimated by standard techniques using an automated analyzer. 2 ml of venous blood was collected in EDTA bulb from patients and centrifuged to obtain plasma. Analysis of plasma lactate was carried out by the commercial kit supplied by the manufacturer in an automated analyzer. The test is based on the principle of 'Calorimetric assay'. The intensity of the color formed is directly proportional to Lactate concentration.

4.10 FOLLOW UP

Participants of the study were followed up from the time of admission till the delivery and beyond in order to evaluate possible development of complications.

4.11 STATISTICAL ANALYSIS

Analysis of collected data was done using descriptive statistics since the study was an observational study. The data obtained was coded and entered into Microsoft Excel Worksheet. Statistical Package for the Social Sciences (SPSS) for Windows version 20.0 was employed for statistical analysis and interpretation of collected data.

Continuous quantitative variables were represented by mean ± SD (minimum, maximum). Data was divided into two groups with respect to certain qualitative

characteristic for comparison. Krushal-Wallis one-way analysis of variance was used to compare distribution between the groups and pre and post treatment measures. Discrete variables were represented by median ± SD using non-parametric tests. Categorical data were expressed in terms of frequencies and percentages. Chi-square test was used to measure the strength of associations between the categorical data including outcomes, clinical and demographic characteristics. Suitable graphs were used to depict the comparisons. Probability values ('p' values) of <0.05 at 95% confidence interval were considered to be statistically significant.

4.12 ETHICAL ISSUE AND ETHICAL CLEARANCE

An informed choice was given by each participant based on the participant's full understanding of the method or procedure, including its characteristics, actions, and possible risks and benefits. The participants' consent was sought and obtained after adequate information about all aspects covered by the study. During the process of obtaining consent, the rights to decline participation or to withdraw participation at any time of the study should they wish to do so, were emphasized. Information regarding privacy and confidentiality of the patient was provided. It was also ensured that the participants were educated about warning signs and the need for follow- ups.

Ethical clearance for this study was obtained from the Institutional Ethics and Research Committee, KAHER's Dr. Prabhakar Kore Hospital, Belagavi, Karnataka in prescribed format.

5. RESULTS

The study was conducted at the Department of Obstetrics and Gynecology of KAHER's Dr. Prabhakar Kore Charitable Hospital, Belagavi, Karnataka for a period of one year and six months. This observational study was conducted among antenatal women with pre-eclampsia or eclampsia, admitted to the labor ward from January 2020 to December 2020, after the study was approved by the Institute Ethics Committee.

Data obtained from structured questionnaires was analyzed using Graph Pad Prism version 9**.0** and MS Excel. Continuous quantitative variables have been given as mean ± SD (minimum, maximum). Categorical data have been expressed in terms of frequencies and percentages and have been compared using Chi-square test. Krushal-Wallis one-way analysis of variance was used to compare distribution between the groups and pre and post treatment measures. P value<0.05 was considered as significant in all cases.

5.1 Recruitment of study participants

The total number of mothers screened and enrolled for this study was 170. Details of inclusion of study participants have been given in Figure 5-1. The age of the participants ranged from 18 years to 38 years with mean age 25.08 ± 4.7 years. Average body mass index of study participants was found to be 24.14 ± 4.3 (ranging from 17.85 to 39).

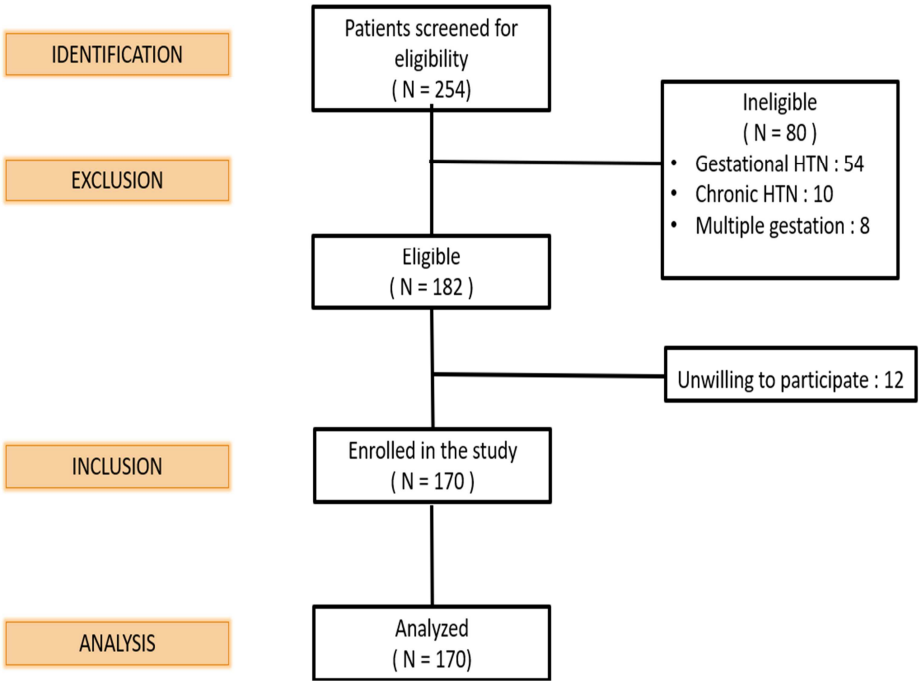

Figure 5-1: Enrolment of participants for the study

5.2 Distribution of study participants based on demographic and obstetric parameters

The distribution of study participants based on demographic and obstetric parameters has been given in Table 5-1

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

Sl.No	Parameter	Subgroup	No. of study participants	% of study population
1	Maternal age	≤ 20 years	32	18.8%
		21-25 years	67	39.4%
		26-30 years	41	24.1%
		≥ 30 years	30	17.6%
2	Body Mass Index	< 18.5	15	8.8%
		18.5 to 24.9	87	51.2%
		25 to 29.9	52	30.6%
		≥ 30	16	9.4%
3	Parity	Primigravida	91	53.5%
		Multigravida	79	46.5%
4	Gestational Age	≤ 37 weeks	99	58.2%
		> 37 weeks	71	41.7%

(i) **Age**: Maximum number study participants were in the age group of 21-25 years (39.4%), followed by the age group of 26-30 years (24.1%). There were almost similar number of study participants in the ≤ 20 years (18.8%) and ≥ 30 years (17.6%) (Figure 5-2).

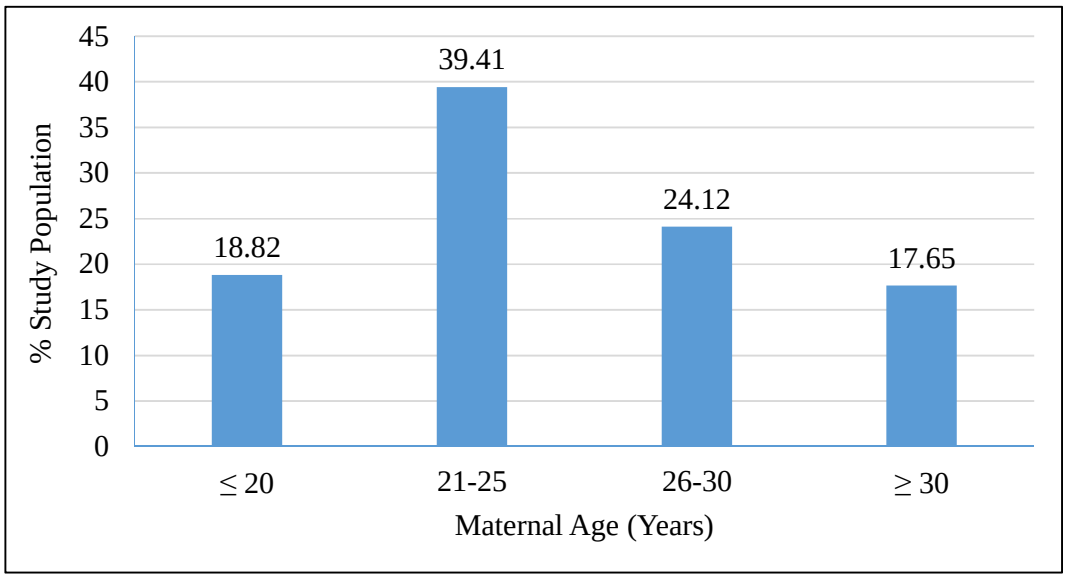

Figure 5-2: Distribution of study participants based on age

(ii) BMI: Of the study participants, 15 women (8.8%) were underweight (BMI < 18.5). 51.2%% (n=87) were observed to be in the BMI range of 18.5 to 24.9. Pre-pregnant weight (BMI) of 30.6% (n=52) of the study participants was in range of 25 to 29.9. 16 study participants were in the overweight range (9.4%) (Figure 5-3).

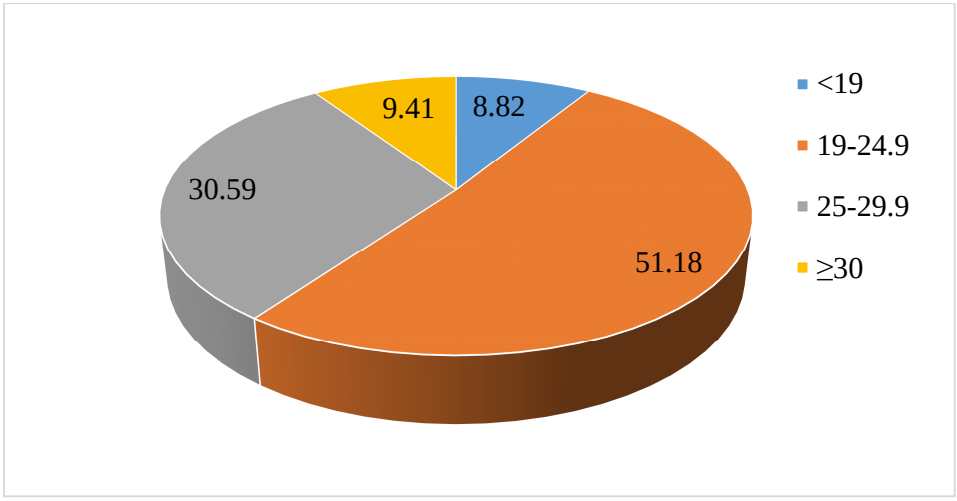

Figure 5-3: Distribution of study participants based on BMI

(iii) **Gravida:** Maximum number of study participants were primigravidae (53.5%). 46.5% of the participants were multigravida (n=79) (Table 1).

(iv) Gestational age: Average gestational age of study participants was observed to be 35.2 ± 4.02 (ranging between 26 weeks to 42.1 weeks). Gestational age of maximum proportion of study participants (58.2%) was ≤ 37 weeks. 41.7% of the women delivered after 37 weeks of pregnancy (Table 1).

5.3 Distribution of study participants based on clinical parameters

Both systolic and diastolic blood pressures were elevated for the study participants and average systolic and diastolic blood pressures in the range of 154.5 and 100 mm/Hg respectively (Table 5-2). Mean serum lactate levels for study participants was found to be 40.5 units. SpO2 levels were noted to be in the normal range. 42.3% of the study participants had 30-100 mg/dL proteinuria levels. Elevated proteinuria (> 300 mg/dL) was observed for around 30% of the study participants.

Table 5-2: Distribution of study participants based on clinical parameters

Sl. No.	Parameter	Mean ± SD	Median (IQR)
1	Systolic Blood Pressure	154.5 ± 16.9	150 (120, 200)
2	Diastolic Blood Pressure	100.1 ± 9.7	100 (80, 140)
3	Serum Lactate	40.5 ± 24.8	33.9 (7.6, 152)
4	SpO2	97.8 ± 2.1	98 (84,100)
5	Proteinuria	**Subgroup**	**N (%)**
		30 -100 mg/dL	72 (42.3%)
		100 -300 mg/dL	48 (28.2%)
		300-600 mg/dL	39 (22.9%)
		≥ 600 mg/dL	11 (6.5%)

SD-Standard deviation, IQR – Interquartile range

5.4 Distribution of study participants based on severity of condition

Table 5-3 shows the distribution of study participants based on case severity. Major proportion of study participants (61.1%) were observed to have PE with severe features. 20% of the women enrolled in the study were diagnosed with antepartum eclampsia. Pre-eclampsia without severe features was seen in 18.3% of the study participants respectively.

Table 5-3: Distribution of study participants based on severity of condition

Severity of condition	Number of study participants	Percentage (%)
PE without severe features	32	18.28%
PE with severe features	104	61.1%
Antepartum Eclampsia	34	20.0%

5.5 Correlation of serum lactate levels and severity of pre-eclampsia in study participants

Significant correlation (P=0.03) was established between the serum lactate levels and severity of pre-eclampsia in the study participants. In case of women with ante-partum eclampsia, serum lactate levels were in the range of 7.6 (minimum) to 152 (maximum) with average levels of 50.84. For PE without severe features and PE with severe features, the mean lactate levels were 39.8 and 37.2 respectively (Table 5-4 and Figure 5-4).

Table 5-4: Correlation of serum lactate levels and severity of Preeclampsia

Condition	Mean ± SD	Median (IQR)	P value
PE without severe features	39.8 ± 19.7	34.5 (16.0-88.3)	0.034* K-W
PE with severe features	37.2 ± 22.7	30.2 (9.3-132)	
Antepartum Eclampsia	50.84 ± 31.93	43.03 (7.6-152)	

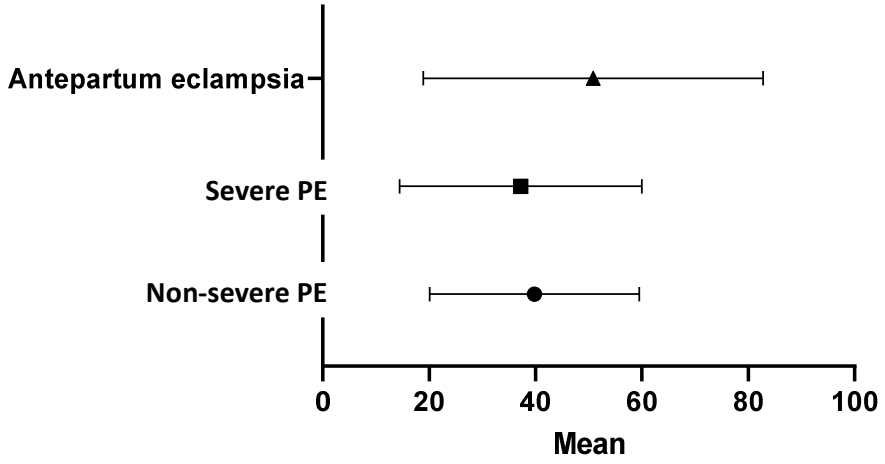

Figure 5-4. Mean lactate levels among different groups of study participants

5.6 Maternal complications and distribution based on serum lactate levels

(i) Overall study population

Overall, maternal complications were observed in 85 of the 170 study participants (50%). In all cases, majority of the study participants had high serum lactate levels (>27 mg/dL), establishing the association of raised serum lactate levels with maternal complication in pre-eclamptic and eclamptic women.

Most prevalent maternal complication was complications in the central nervous system (CNS), seen in 36 of the cases (Table 5-5). In this case, 77.7% of the women had lactate levels > 27 mg/dL. This was followed by HELLP, where 25 pre-eclamptic and eclamptic women reported this complication. In case of patients with HELLP, 80% of the women had high serum lactate levels and this association was statistically significant (P=0.03). Disseminated intravascular coagulation, pulmonary edema, renal dysfunction and abruption were some of the other maternal complications noted in the study participants and in all cases, serum lactate levels > 27 mg/dL were noted in > 60% of the patients.

Table 5-5: Maternal complications and distribution of total patient population based on serum lactate levels

Complication	Total Cases	Lactate Concentration			P Value
		<19.8 mg/dl	**19.8-27**	**>27 mg/dl**	
CNS	36	6 (16.6%)	2 (5.5%)	28 (77.7%)	0.06
DIC	5	2 (40%)	0	3 (60%)	0.25
Pulmonary edema	5	1 (20%)	1 (20%)	3 (60%)	0.97
HELLP syndrome	25	5 (20%)	0	20 (80%)	0.03*
Renal dysfunction	5	1 (20%)	0	4 (80%)	0.5
Abruption	9	2 (22.2%)	0	7 (77.7%)	0.3

(ii) PE with severe features

Maternal complications were observed in 42 women with severe pre-eclampsia. In all cases, majority of the study participants had high serum lactate levels (> 27 mg/dL), establishing the association of raised serum lactate levels with maternal complication in PE with severe features.

Most prevalent maternal complication in PE with severe features was HELLP, seen in 20 of the cases (Table 5-6). In this case, 80% of the women had lactate levels > 27 mg/dL. CNS complications (3 cases), disseminated intravascular coagulation (5 cases), renal dysfunction (5 cases) and abruption (9 cases) were some of the other maternal complications noted in case of PE with severe features and in all cases, serum lactate levels > 27 mg/dL were noted in > 60% of the patients. However, the association of lactate levels and maternal complications was not statistically significant for any of the complications. Pulmonary edema was not observed in any of the women with PE with severe features in this study.

Table 5-6: Maternal complications and distribution of cases of PE with severe features based on serum lactate levels

Complication	Total cases	Lactate concentration			P value
		<19.8 mg/dL N (%)	19.8-27 mg/dL N (%)	>27 mg/dL N (%)	
CNS complication	3	0	0	3 (100%)	0.61
DIC	5	2(40%)	0	3(60%)	0.25
Pulmonary edema	0	0	0	0	-
HELLP	20	4(20%)	0	16(80%)	0.62
Renal dysfunction	5	1(20%)	0	4(80%)	0.9
Abruption	9	2(22.2%)	0	7(77.7%)	0.42

(iii) Antepartum eclampsia

Maternal complications were observed in 43 women with antepartum eclampsia. In all cases, majority of the study participants had high serum lactate levels (> 27 mg/dL), establishing the association of raised serum lactate levels with maternal complication in antepartum eclampsia also.

Most prevalent maternal complication in antepartum eclampsia were CNS complications, seen in 33 of the cases (Table 5-7). In this case, 75.5% of the women had lactate levels > 27 mg/dL. Pulmonary edema and HELLP were seen in 5 patients each. Disseminated intravascular coagulation, renal dysfunction and abruption were not observed in any of the women with antepartum eclampsia in this study. Serum lactate levels > 27 mg/dL were noted in > 60% of the patients. However, the association of lactate levels and maternal complications was not statistically significant for any of the complications.

Table 5-7: Maternal complications and distribution of cases of antepartum eclampsia based on serum lactate levels

Complication	Total cases	Lactate concentration			P value
		<19.8 mg/dL	19.8-27 mg/dL	>27 mg/dL	
CNS complication	33	6(18.18%)	2(6.06%)	25(75.75%)	0.6
DIC	0	0	0	0	-
Pulmonary edema	5	1(20%)	1(20%)	3(60%)	0.7
HELLP	5	1(20%)	0	4(80%)	0.7
Renal dysfunction	0	0	0	0	-
Abruption	0	0	0	0	-

5.7 Correlation of serum lactate levels and maternal complications among the different severity categories of pre-eclampsia

(i) Pre-eclampsia without severe features

For patients with PE without severe features, abnormal lactate levels were detected for 30 out of the 32 study participants and two women has normal lactate levels (Table 5-8). Maternal complications were not observed in any of the women with PE without severe features.

Table 5-8: Serum lactate levels and maternal complications among the women with PE without severe features

Maternal complications		**Lactate levels**	
		Abnormal	**Normal**
CNS	Absent	30 (93.75%)	2 (6.25%)
	Present	0	0
DIC	Absent	30 (93.75%)	2 (6.25%)
	Present	0	0
Pulmonary Edema	Absent	30 (93.75%)	2 (6.25%)
	Present	0	0
HELLP	Absent	30 (93.75%)	2 (6.25%)
	Present	0	0
Abruption	Absent	30 (93.75%)	2 (6.25%)
	Present	0	0

(ii) PE with severe features

Majority of the study participants with PE with severe features reported abnormal lactate levels (Table 5-9). Compared to women with normal lactate levels, participants with maternal complications reported abnormal levels of serum lactate. Pulmonary edema was not a reported maternal complication in case of PE with severe features.

Table 5-9: Serum lactate levels and maternal complications among the women with PE with severe features

Maternal complications		Lactate level		p-value
		Normal	**Abnormal**	
CNS	Absent	18 (20%)	71 (78.9%)	0.61
	Present	0	1 (1.1%)	
DIC	Absent	16 (17.8%)	69 (76.7%)	0.25
	Present	2 (2.2%)	3 (3.3%)	
Pulmonary Edema	Absent	18 (20%)	72 (80%)	
	Present	0	0	
HELLP	Absent	14 (15.6%)	58 (64.4%)	0.79
	Present	4 (4.4%)	14 (15.6%)	
Renal	Absent	17 (18.9%)	68 (75.6%)	0.9
	Present	1 (1.1%)	4 (4.4%)	
Abruption	Absent	17 (18.9%)	65 (72.2%)	0.57
	Present	1 (1.1%)	7 (7.8%)	

(iii) Antepartum eclampsia

Majority of the study participants with antepartum eclampsia reported abnormal lactate levels (Table 5-10). Compared to women with normal lactate levels, participants with maternal complications reported abnormal levels of serum lactate. However, the association of abnormal lactate levels and maternal complications in antepartum eclampsia was not statistically significant. DIC, renal complication and abruptions were not reported maternal complication in case of antepartum eclampsia.

Table 5-10: Serum lactate levels and maternal complications among the women with antepartum eclampsia

Maternal complications		Lactate level		p-value
		Normal	Abnormal	
CNS	Absent	0	1 (2.97%)	0.6
	Present	6 (17.6%)	27 (79.4%)	
DIC	Absent	6 (17.6%)	28 (82.4%)	
	Present	0	0	
Pulmonary Edema	Absent	5 (14.7%)	24 (70.6%)	0.7
	Present	1 (2.9%)	4 (11.8%)	
HELLP	Absent	5 (14.7%)	24 (70.6%)	0.7
	Present	1 (2.9%)	4 (11.8%)	
Renal	Absent	6 (17.6%)	28 (82.4%)	
	Present	0	0	
Abruption	Absent	6 (17.6%)	28 (82.4%)	
	Present	0	0	

5.8 Fetal outcomes of study participants with pre-eclampsia and eclampsia

Most of the neonates weighed less than 2.5 kg at birth (> 65%), indicating that neonates of women with pre-eclampsia and eclampsia are lower side of birth weight. Pre-term births were more in proportion (51.2%) compared to term births (48.8%). The number of still births were 32 in this study, which points to the fact still-births are one of the possible negative fetal outcomes of pre-eclampsia and eclampsia. In this study, a 1.2% perinatal mortality was noted. The rate of NICU admissions were 37.6% in this study (Table 5-11).

Table 5-11: Demographic characteristics of neonates of women with pre-eclampsia and eclampsia

Variable	Sub group	Number of neonates	Percentage
Birth weight	<1 Kg	22	12.9%
	1-1.5 Kg	41	24.1%
	1.5-2.5 Kg	51	30.0%
	> 2.5 Kg	56	32.9%
Delivery	Preterm	87	51.1%
	Term	83	48.8%
Status of the baby	Live birth	137	80.6%
	Still birth	31	18.2%
NICU admission		64	37.6%

6. DISCUSSION

Pre-eclampsia, defined as sudden onset hypertension with or without proteinuria and other organ complications after 20 weeks of pregnancy, is associated with serious adverse maternal and neonatal outcomes. Several studies have been conducted using a variety of biochemical indicators as predictors and prognosticators for preeclampsia in pregnant women. Of these biomarkers, serum lactate has been shown as an important marker for predicting the maternal and fetal outcomes of pregnant women with pre-eclampsia.

Lactate dehydrogenase, an intracellular enzyme is elevated in pre-eclamptic women due to cellular death. As a result, serum lactate levels can be used to determine the severity of disease as estimated degree of cellular death with the assumption that lactate levels indicate tissue hypoxia and hypoperfusion in preeclampsia. Therefore, it can be used as a reliable indicator for predicting the severity of this condition[54]. Accurate identification of at-risk women, early diagnosis, and appropriate treatment can be beneficial in improvement of maternal and fetal outcomes. The present study was taken up with the objective to evaluate association between serum lactate concentration with the severity of preeclampsia and eclampsia as well as the risk of developing maternal complications.

This study was a hospital based observational study conducted at KAHER's Dr. Prabhakar Kore Hospital, Belagavi for a period of 12 months. Data on demographic and clinical parameters was collected from 170 antenatal women, with a gestational age of > 20 weeks and diagnosed with new onset hypertension after obtaining written consent from the participants of the study. Statistical tools were employed to understand the correlation between serum lactate levels and severity of

pre-eclampsia and to assess maternal and perinatal outcomes of pre-eclampsia and eclampsia.

The age of the participants ranged from 18 years to 38 years with mean maternal age 25.08 ± 4.7 years. This is in correlation with previously reported study where average maternal age ranged between 25 years to 27 years[55]. Majority of pre-eclampsia patients in our study were primigravida with average BMI. These results correlate with findings of Qublan HS et al., who reported that patients with PE with severe features were younger in age and had low parity[54]. Gestational age of maximum proportion of study participants (58.2%) was ≤ 37 weeks, which indicates that before-term deliveries are more prevalent in pre-eclamptic women.

Both systolic and diastolic blood pressures were elevated in the study participants, along with elevated serum lactate levels. In their investigation, Jaiswar et al. discovered that patients with greater serum LDH levels had significantly higher systolic and diastolic blood pressure, which is similar to findings of this study[53]. Levels of proteinuria were elevated for most of the patients in this study. Although association between levels of proteinuria and severity of pre-eclampsia has not been found in previous studies[56] the likely hood of severity of pre-eclampsia increases once proteinuria is detected in the patients. Elevation of blood pressure is a hallmark of pre-eclampsia and in this study, elevation in systolic and diastolic blood pressure was around 25% which is considered as moderate increase. Similar findings have been reported in a recent study, where along with BP elevation, proteinuria has been also associated with pre-eclampsia[57].

In this study, > 60% of the patients reported PE with severe features. In a recent study, the prevalence of PE with severe features among postpartum mothers in Zanzibar was found to be 26.3%[58]. Since the present study was conducted in referral

hospitals, patients were admitted during later stages of pre-eclampsia and not during initial stages, which has contributed to the high number of severe cases. It is unknown as to how long these patients have spent at the primary care centers. When compared to prior studies on pre-eclampsia in the global south, which found prevalence rates ranging from 1.8 percent to only 16.7 percent[59,60], the current study's prevalence rate is rather high. Also, considering this to be the referral center, these results can be generalized for the whole region.

Serum lactate levels have come up as a reliable biomarker for pre-eclampsia in this study. Significant association between serum lactate levels and severity of pre-eclampsia has been establish from findings of this study (P=0.03). In their study, Rukhsana Afroz et al discovered that 91.1% of severe pre-eclamptic women and 82.9% of moderate pre-eclamptic women had elevated serum lactate levels[61]. However, in this study there was no direct correlation between a rise in serum lactate levels and an increase in the severity of preeclampsia. The mean lactate level in PE with severe features (37.2 mg/dL) was lower than in PE without severe features patients (39.8 mg/dL), which is contrary to findings of Qublan et al[54]. The mean lactate levels in antepartum eclampsia was the highest, at 50.8 mg/dL. This points to the importance of estimation serum lactate levels in hypertensive pregnant women, so that the emergence of pre-eclampsia or eclampsia can be well managed.

The prevalence of maternal complications observed in this study was at 50%, with 85 of the 170 study participants having some complications. Increased lactate production, slower clearance, or a combination of both can cause a rise in serum lactate concentration[62]. Many experimental tests have demonstrated the link between the development of lactate and tissue hypoxia by reducing the components of systemic oxygen delivery until the extraction of oxygen can no longer maintain

oxygen availability to the cells[63]. Preeclampsia causes cellular hypoxia due to microcirculatory or macro-circulatory malfunction, which culminates in an increase in serum lactate concentration and subsequent maternal complications. Indeed, it has been found that larger lactate levels are produced and sent to the placenta in pre-eclamptic patients. Impairment of end-organ is one of the direct effects.

In the study of Qublan et al.[54] severely pre-eclamptic women with elevated serum lactate levels had a significant increase in problems such as eclampsia, abruption placenta, and several other issues when compared to women with lower serum lactate levels. In a study by Martin et al., high lactate level was found to be a strong predictor for subsequent maternal morbidity[64]. Significant association with maternal complications such as HELLP syndrome and risk of maternal mortality has also been established in study by Catanzerite et al.[65], which is accordance with findings of this study.

In this study, the most prevalent maternal complication was complications in the central nervous system (CNS), seen in 36 of the cases, and 77.7% of the women with CNS had lactate levels > 27 mg/dL, which is elevated. However, significant association was established between elevated serum lactate levels and HELLP, where 25 pre-eclamptic and eclamptic women reported this complication in this study. In case of patients with HELLP, 80% of the women had high serum lactate levels and this association was statistically significant (P=0.03). Hemolysis, increased liver enzymes, and low platelet count syndrome (HELLP), a life-threatening thrombotic microangiopathy condition, are all typical symptoms of preeclampsia as established from previous studies [2,48,66,67].

HELLP syndrome affects about 0.6% of all pregnancies and is considered a milder form of PE with severe features. 70% of instances happen between weeks 27 and 37, and 20% happen within 48 hours following delivery. The majority of patients with HELLP syndrome have preeclampsia-like symptoms. HELLP develops in 10% to 20% percent of individuals with PE with severe features. Although liver involvement in preeclampsia is uncommon, it does indicate serious illness. Furthermore, it is well known that the liver (70%) and kidneys are the organs largely responsible for removal of excess serum lactate, and that lactate clearance may be hampered if these organs are dysfunctional[68]. This is consistent with our findings, as women with lactate concentrations had higher rates of HELLP (80% of the HELLP patients) and renal impairment (80% of the renal impairment patients) problems in our study.

Abruptions as a maternal complication was also observed in this study. Nine cases of abruptions were noted, which were all noted in case of patients with PE with severe features. This is similar to findings of Jaiswar et al.[53], who also observed abruptions as a maternal outcome in patients with PE with severe features. There were no complications in patients with pre-eclampsia without severe features in this study. This points to the fact that to avoid the most serious problems associated with a higher risk of maternal mortality and disability, early detection of preeclampsia and appropriate prevention are required.

On analysis of maternal complications and severity of pre-eclampsia, most of the patients showing maternal complications had abnormal levels of serum lactate. As an interesting observation, even in the group of women who did not have a problem, lactate concentrations were higher[23]. This was true for patients with PE without severe features, where patients with abnormal lactate levels did not have any complications.

Almost 20% of patients with severe and antepartum eclampsia had abnormal levels of serum lactate, but did not present any complications. It is possible that a small percentage of these cases did not develop a complication because they were delivered before the complication developed, in a competing risk environment. The elevated lactate concentrations in the general group may be due to the high lactate concentrations in these cases where the problem was avoided by early delivery.

In this study, maximum maternal complications were reported in the severe category, as compared to antepartum eclampsia or PE without severe features. Pregnancy complications such as PE with severe features/eclampsia or HELLP syndrome are linked to a higher risk of maternal morbidity and mortality. Severe bleeding from abruptions, pulmonary edema, cerebral haemorrhage, and liver rupture are among complications that can cause maternal morbidity. Women who acquire PE with severe features, eclampsia, or HELLP syndrome before 32 weeks of pregnancy are more likely to suffer these problems[69,70]. Women who have PE with severe features/eclampsia, those with HELLP syndrome have a higher risk of unfavorable maternal outcomes[31]. Similar outcomes were observed in this study.

In this study, the mean birth weights of neonates both to patients with pre-eclampsia and eclampsia were lower than 2.5 kg and this has been observed in previous studies[71]. Contrary to a previous study conducted in Canada where 61.2% neonates born to women with preeclampsia were delivered after 37 weeks, in this study 51.1% of the neonates were delivered before 37 weeks. However, there was no statistically significant difference in birth weight between neonates born to women with preeclampsia after 37 weeks and babies born to normotensive mothers at the same gestational age. As a result, most babies born at term to pre-eclamptic women have a normal birth weight for their gestational age.

Pre-term newborns are more susceptible to the effects of low birth weight. This could explain why prior epidemiologic investigations on the relationship between preeclampsia or prenatal hypertension and birth weight came up with inconsistent results[71–73]. The incidence of preterm deliveries among patients with pre-eclampsia was high (27.5% percent) in one study, where it was observed that babies born to women with pre-eclampsia had a substantially lower mean birth weight (2.9 kg)[71]. The total effect on birth weight seen in the study could be influenced by the proportions of full-term and preterm deliveries among preeclampsia patients. The fact that most babies born patients with pre-eclampsia at term have normal fetal growth cannot be reconciled with the widely held idea that preeclampsia is caused by a reduction in uteroplacental perfusion[74,75]. A growing body of evidence supports the idea that preeclampsia has significant pathophysiologic heterogeneity. In this study, significant association (P=0.03) was established bet wen birth weight and lactate levels of mother, making it an important predictor of fetal outcome in pre-eclamptic women.

Studies have shown that premature births, NICU admissions and neonatal morbidity, and neonatal fatalities are all higher among neonates of pre-eclamptic women[76]. It was observed in this study that 51% of the neonates were born pre-term and the rate of NICU admissions was around 38%, which is higher that literature reported findings. The small cohort and stringent inclusion criteria can be a possible reason for this high incidence of both prematurity and NICU admissions. Also, since this study was conducted at a tertiary referral hospital, so most patients were admitted for complicated or preterm deliveries.

The number of still births were 32 in this study, which points to the fact still-births are one of the possible negative fetal outcomes of pre-eclampsia and eclampsia. In this study, a 1.2% perinatal mortality was noted. The rate of NICU admissions were 37.6% in this study. It is widely established that pregnancies complicated by PE with severe features or eclampsia, increase maternal and neonatal morbidity and mortality. In the literature, different prenatal morbidity and mortality rates are given[77–80]. Despite the fact that neonatal morbidity was similar in all categories in our study, perinatal mortality was higher in pregnancies complicated by HELLP syndrome or PE with severe features before 32 weeks of pregnancy due to a larger frequency of stillbirths. Interestingly, when early (32 weeks) foetal losses were considered, the eclampsia group performed slightly better. This was most likely attributable to the fact that the eclampsia group had more abdominal deliveries and fewer growth-restricted foetuses. The increased foetal and perinatal mortality may be explained by the high proportion of patients in the research cohort who did not have frequent prenatal follow-ups. In situations of PE with severe features, eclampsia, or HELLP syndrome, these findings show that neonatal morbidity and mortality are reliant on gestational age rather than illness. Magann et al. discovered that foetal morbidity and death are depending on gestational age, and found that HELLP syndrome, PE with severe features, and eclampsia had similar and nonsignificant associations[81]. However, in this study, no statistically significant association between serum lactate levels and fetal outcomes were observed

7. CONCLUSION

In obstetrics, determination of a biomarker that can predict development of maternal complications, could help in the successful management of PE with severe features, thus improving the perinatal outcome. Serum lactate levels could be used as a predictor for the severity of this condition considering the fact that lactate levels indicate tissue hypoxia and hypoperfusion in preeclampsia. However, a larger scale study with a larger sample size is required to strengthen the role of lactate as a predictor of PE. Furthermore, use of an additional biomarker along with lactate could provide more accurate results. Prediction of maternal and fetal outcomes in pre-eclampsia from a simple biochemical test can help prevent unfavorable events associated with pre-eclampsia and eclampsia.

The key conclusions of this study are as follows:

(i) Incidence of pre-eclampsia and eclampsia were higher in case on multigravida and women of younger age group.

(ii) There is a positive association of increasing serum lactate levels and severity of pre-eclampsia and eclampsia.

(iii) Higher incidences of maternal complications were likely in patients with PE with severe features compared to patients with PE without severe features.

(iv) There is no positive association between increased lactate concentration and development of maternal complications in pre-eclamptic and eclamptic women.

(v) Higher incidences of pre-term births, birth weights < 2.5 kgs and NICU admissions were likely in case of women with pre-eclampsia and eclampsia.

SUMMARY

The present study was an observational study conducted to evaluate association between serum lactate concentration with the severity of preeclampsia and eclampsia as well as the risk of developing maternal complications and to assess maternal and perinatal outcomes of pre-eclampsia and eclampsia. The study was carried out at the Department of Obstetrics and Gynaecology of KAHER's Dr. Prabhakar Kore Charitable Hospital, Belagavi, Karnataka for a period of one year and six months. Patient population consisted of antenatal women with Pre-eclampsia or eclampsia, admitted to the labor ward of Department of OBG at KAHER's Dr. Prabhakar Kore Hospital, Belagavi, from January 2020 to December 2020. A total of 170 women were selected for enrollment based on inclusion criteria. Data regarding sociodemographic and clinical characteristics was collected in form of structured questionnaires and analyzed statistically.

Key findings of this study have been summarized as follows:

- The age of the participants ranged from 18 years to 38 years with mean age 25.08 ± 4.7 years. Average body mass index of study participants was found to be 24.14 ± 4.3 (ranging from 17.85 to 39).
- Maximum number of study participants were primigravidae (53.5%). 46.5% of the participants were multigravida (n=79).
- Gestational age of maximum proportion of study participants (58.2%) was ≤ 37 weeks. Average gestational age of study participants was observed to be 35.2 ± 4.02 (ranging between 26 weeks to 42.1 weeks).
- Both systolic and diastolic blood pressures were elevated for the study participants and average systolic and diastolic blood pressures in the range of 154.5 and 100

mm/Hg respectively. Mean serum lactate levels for study participants was found to be 40.5 mg/dl.

- Major proportion of study participants (61.1%) were observed to have PE with severe features. 20% of the women enrolled in the study were diagnosed with antepartum eclampsia. PE without severe features was seen in 18.3% of the study participants.
- Serum lactate levels were one of the novel predictors of severity of pre-eclampsia. Significant correlation (P=0.03) was established between the serum lactate levels and severity of pre-eclampsia in the study participants.
- Maternal complications were seen in 50% of the study participants. Most prevalent maternal complication was complications in the central nervous system, seen in 36 women. This was followed by HELLP which was observed in 25 study participants. Disseminated intravascular coagulation, pulmonary edema, renal dysfunction and abruption were some of the other maternal complications.
- A raised serum lactate concentration was observed in women presenting with complications, although a statistical significance could not be established.
- 61.1% of the patients with maternal complications belonged to the PE with severe features group. Association of HELLP and serum lactate level was found to be statistically significant. None of the patients with PE without severe features had any maternal complications. Pulmonary edema and CNS complications were seen in case of antepartum eclampsia.
- > 65% of the neonates weighed less than 2.5 kg at birth, indicating that neonates of women with pre-eclampsia and eclampsia are born with low birth weight. Pre-term births were more in proportion (51.2%) compared to term births (48.8%).

- In this study, a 1.2% perinatal mortality was noted. The rate of NICU admissions were 37.6% in this study.

LIMITATIONS OF THE STUDY

Following are limitations of this study:

- This study was conducted in a tertiary center therefore the findings may not adequately reflect the entire Belagavi region.
- Conclusions and deductions from this study cannot be assertive because of the limitations of its small sample size.

The study did not take into account the participants' socioeconomic position, including as income, living standards, and nutritional status, which could be important factors of pre-eclampsia

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ANNEXURE - I - ETHICAL CLEARANCE

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

Ref: MDC/DOME/ 194 **Date: 24/12/2019**

To.

REG. NO. BJ0119004

PG student in Obstetrics and Gynecology,
J.N.Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"ASSOCIATION OF SERUM LACTATE CONCENTRATION WITH SEVERITY OF PRE-ECLAMPSIA AND MATERNAL COMPLICATIONS: AN OBSERVATIONAL STUDY ",** is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

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

(Dr. Roopa M Bellad)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

8

ANEXXURE II- CONSENT FORM

Purpose of the study:

I have been informed by **REG. NO. BJ0119004**, Post Graduate in M.S. Obstetrics and Gynaecology under the guidance of Dr._____, Department of Obstetrics and Gynaecology, J.N. Medical College, KAHER, Belagavi is conducting a study to determine 'ASSOCIATION OF SERUM LACTATE LEVEL WITH SEVERITY OF PRE-ECLAMPSIA AND MATERNAL COMPLICATIONS: AN OBSERVATIONAL STUDY' at KAHER's Dr. Prabhakar Kore Charitable Hospital, Belagavi.

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

As the incidence of PE and eclampsia is high, this study aims at assessing the incidence and demographic features of PE and eclampsia in our institution and consequently analyse incidence of individual outcomes.

Study procedure:

Once I have signed the informed consent form, the personal details like name, age, place, address, my education, my health, reproductive history and other information will be noted down. Blood sample will be withdrawn for lactate level. The reports will be noted and I will be followed up.

Potential Risks:

There are no observable risks associated with the study.

Financial incentive for participation:

I will not receive any payment for taking part in this research study.

Alternatives:

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

Privacy:

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

Authorisation to publish results:

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

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

Institutional Policy:

In case I have any questions related to the study, in future, I can contact **REG. NO. BJ0119004**, Department of Obstetrics and Gynaecology, KAHER, J.N Medical College, Ph. No. _____ or phone number: _____ or Dr._____, Dept. Of Obstetrics and Gynaecology, KAHER, J.N Medical College, Belagavi.

Voluntary Participation:

My participation in the study is voluntary. In case I need any further information regarding my rights as study participant, I may contact Dr. Roopa M Bellad, Professor of Paediatrics, as Chairman of J. N. Medical College Institutional Ethics Committee on Human Subjects Research, Phone No.0831 2473777 ext-1527 at J. N. Medical College, Belagavi. My doctor will take care of me during this pregnancy or in the future. I am free to stop participation in this study at any time and for any reason.

Signatures:

Person requesting consent, please check applicable boxes:

☐ Consent obtained (for adult respondent)

I have read the consent form or the consent form has been read to me. I understand the consent and the signature or sign below confirms that I agree to participate in this study.

Study identification number: ☐☐☐☐

_____ _____

Signature or thumbprint of participant Date

ANEXXURE III - SCREENING AND RECRUITMENT FORM

1) Screening number: ☐☐☐

2) Date of screening (dd-mm-yyyy): ☐☐ ☐☐ ☐☐☐☐

3) First name : _____ Middle name : _____ Last name: _____

4) Age (years): ☐☐

5) OP/IP number: ☐☐☐☐☐☐

6) Husband's name: _____

7) Address: _____

8) Phone number: _____

9) Gestational age:

1) Is the period of gestational above 20 weeks Yes ☐ ☐

2) History: (1=yes, 2=no)

a) Patients with gestational hypertension and chronic hypertension ☐

b) Women with pre-existing renal or vascular diseases. ☐

c) Multiple gestation ☐

The woman is eligible to consent only if answer to 1 is yes and 2 is no:

Eligible ☐

Consented ☐

ANEXXURE IV - DATA COLLECTION INSTRUMENT

ASSOCIATION OF SERUM LACTATE CONCENTRATION WITH SEVERITY OF PRE-ECLAMPSIA AND MATERNAL COMPLICATIONS: AN OBSERVATIONAL STUDY

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

COMPLAINTS AND HISTORY OF PRESENTING COMPLAINT:

Period of gestation	
Duration of pain abdomen	
Duration of leak per vagina	
Duration of bleeding per vagina	
Perception of fetal movements (Yes/No)	
Imminent signs – 1. Headache 2. Blurring of vision 3. Epigastric discomfort	
Seizures (Yes/No) If Yes: 1. Number of episodes 2. Duration 3. Loss of consciousness 4. Lucid interval	
Any associated medical history	

OBSTETRIC HISTORY:

Married Life / Consanguinity	
Obstetric Score 1. Gravida 2. Para 3. Living 4. Abortion 5. Death	
Last child birth	
History of previous pregnancy	

MENSTRUAL HISTORY:

Age of menarche	
Previous Menstrual cycles	
LMP	
EDD/CEDD	
Period of gestation	

PAST HISTORY:

Past medical history	
Past surgical history	

FAMILY HISTORY:

PERSONAL HISTORY:

GENERAL PHYSICAL EXAMINATION:

Height	

Weight	
BMI	
Pulse rate	
Blood pressure	
Pallor	
Icterus	
Pedal edema	
SPO2	

SYSTEMIC EXAMINATION:

Cardio-vascular System	
Respiratory System	
Per Abdomen : Size of uterus	
Contractions (present/absent)	
Tender/Tense	
Presentation	
Fetal Heart Sound	

PER SPECULUM EXAMINATION:

Active Bleeding	

PER VAGINAL EXAMINATION:

Consistency of cervix	
Position	
Effacement	
Dilatation	
Station	

CLINICAL DIAGNOSIS:

INVESTIGATIONS:

Blood group/ Rh typing	
Complete Blood Count	
Peripheral smear	
Urine Routine	
Serology: HIV/HbsAg/VDRL	
Bedside COT	
Serum Lactate	
PIH profile: 1. Platelet count	
2. Urea	
3. Serum creatinine	
4. Urine Albumin	
5. LDH	
6. Uric Acid	
7. LFT:	
- Enzymes: SGOT/SGPT/ALP	
- Total Bilirubin	
- Serum Albumin	
DIC profile: 1. D-DIMER	
2. Fibrinogen	
3. PT/INR	
4. aPTT	
5. TT	

OBSTETRIC ULTRASOUND:

Date of scan	
Doppler	

FUNDOSCOPY:

MANAGEMENT:

Anti-hypertensives used	
Mgso4 regimen	
Other drugs if used	
Mode of delivery	
Induction/Augmentation:	
Duration of Labour	
Intrapartum Complications	
Blood Loss	
Indication for LSCS	
Intra-OP findings:	

EXAMINATION OF PLACENTA:

Weight of Placenta	
Retroplacental Clots	
Placental Infarction	
Other features	

MATERNAL OUTCOME:

Peripartum Complications: 1) CNS: Convulsions 2) HEMATOLOGICAL: • DIC	

3) RESPIRATORY: • Respiratory Distress • Pulmonary edema 4) HEPATIC DYSFUNCTION 5) RENAL COMPLICATIONS 6) HELLP 7) Abruptio Placenta 8) Cerebro-vascular complications	
ICU admission (Yes/No)	
Duration of stay	
Condition at discharge	
Cause of death if so	
Intervention if any	

PERINATAL OUTCOME:

Condition at birth (Live/IUFD/Stillbirth)	
Term/Preterm	
Sex	
Weight	
APGAR	
NICU Admission	
Indication for NICU admission	
Condition of baby at discharge	

Sl. NO	IP NUMBER	AGE	OBSTETRIC SCORE	GESTATIONAL AGE	DIAGNOSIS/htn	ANTENATAL COMPLICATION	MEDICAL HISTORY	BMI	SYSTOLIC BP	DIASTOLIC BP	SPO2	PROTEINURIA	LACTATE	PLATELET	DOPPLER	FUNDOSCOPY	NO. OF ANTI-HYPERTENSIVES	MGSO4 PROHYLAXIS	MODE OF DELIVERY	INDICATION	INTRAPARTUM COMPLICATIONS	CNS COMPLICATION	DIC	PULMONARY EDEMA	HELLP	RENAL	ABRUPTIO	ICU	MATERNAL OUTCOME	NEONATAL OUTCOME	TERM/PRETERM	BIRTH WEIGHT	NICU
1)	996902	19 YRS	PRIMIGRAVIDA	34+4	SEVERE PE	FGR	NIL	23.23	150	110	100	2+	24.9	3.67	NORMAL	GRADE 1 HR	2	NIL	LSCS	FAILED INDUCTION	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	1.56 KG	YES
2)	997852	18 YRS	PRIMIGRAVIDA	32+4	SEVERE PE	NIL	NIL	32.46	150	100	99	1+	24.1	2.9	NORMAL	NORMAL	1	NIL	LSCS	FAILED INDUCTION	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	1.8 KG	YES
3)	998708	36 YRS	G4P2L2A1	27+6	SEVERE PE	FGR	HTN IN PREVIOUS PREGNANCY	23.8	180	100	98	1+	25.6	3.5	AEDF	NORMAL	2	YES	VAGINAL	INDUCED	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	STILLBIRTH	PRETERM	685 GMS	NA
4)	999557	27 YRS	PRIMIGRAVIDA	35+1	SEVERE PE	NIL	NIL	29.4	140	90	98	1+	52.8	1.84	INCREASED RESISTANCE	NORMAL	2	YES	LSCS	IMMINENT ECLAMPSIA	CONVULSIONS	YES	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	2.08 KG	YES
5)	1000353	24 YRS	G3P2L2	37+2	SEVERE PE	NIL	NIL	17.85	160	110	100	2+	24.8	3.6	NORMAL	NORMAL	2	NIL	VAGINAL	INDUCED	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2 KG	YES
6)	1007246	21 YRS	PRIMIGRAVIDA	26+6	SEVERE PE	NIL	NIL	35.5	150	90	98	3+	33.9	1.65	INCREASED RESISTANCE	NORMAL	1	NIL	VAGINAL	INDUCTION	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	STILLBIRTH	PRETERM	550 GMS	NA
7)	1011491	27 YRS	G2P1L1	38	NON SEVERE PE	NIL	NIL	27.3	140	90	98	1+	31.6	2.23	NORMAL	NORMAL	NIL	NIL	VAGINAL	SPONTANEOUS	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	3KG	NO
8)	1011418	21 YRS	PRIMIGRAVIDA	37	NON SEVERE PE	NIL	NIL	25.1	140	90	100	1+	26.3	2.2	NORMAL	NORMAL	1	NIL	VAGINAL	SPONTANEOUS	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2.9 KG	NO
9)	1011248	35 YRS	PRIMIGRAVIDA	33+2	SEVERE PE	NIL	NIL	36	180	110	97	2+	77.8	1.76	NORMAL/AEDF	GRADE 2 HR	2	YES	LSCS	SEVERE PE	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	1KG/1.1 KG	YES
10)	1011121	27 YRS	G2P1L1	38+2	SEVERE PE	NIL	NIL	25	150	100	98	2+	23.3	2.76	NORMAL	NORMAL	1	NIL	LSCS	PREVIOUS LSCS	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2.8 KG	NO
11)	1012239	20 YRS	PRIMIGRAVIDA	36+3	SEVERE PE	NIL	NIL	21	180	120	97	1+	43.7	2.1	NORMAL	NORMAL	2	YES	LSCS	IMMINENT ECLAMPSIA	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	2.8 KG	NO
12)	1012222	28 YRS	G2A1	34+5	NON SEVERE PE	FGR	NIL	21.3	140	90	100	1+	34.5	1.67	NORMAL/INCREASED RESISTANCE	NORMAL	1	NIL	LSCS	TWINS	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	1.5 KG	YES
13)	1011548	29 YRS	PRIMIGRAVIDA	30+5	ECLAMPSIA	NIL	NIL	25.1	160	100	94	2+	64.6	1.51	NORMAL	NORMAL	2	YES	LSCS	ANTEPARTUM ECLAMPSIA	NIL	YES	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	1.2 KG	YES
14)	1012091	28 YRS	PRIMIGRAVIDA	28+6	SEVERE PE	FGR	NIL	21.9	140	100	96	3+	15.7	52,500	AEDF	NORMAL	1	NIL	VAGINAL	INDUCTION	NIL	NO	NO	NO	YES	NO	NO	NO	STABLE	STILLBIRTH	PRETERM	670 GMS	NA
15)	1012972	36 YRS	G3P1L1A1	29+1	SEVERE PE	NIL	HTN IN PREVIOUS PREGNANCY	23.8	160	100	94	4+	28.4	96,000	REVERSAL	GRADE 1 HR	2	YES	LSCS	SEVERE PE	NIL	NO	NO	NO	YES	NO	NO	NO	STABLE	LIVE	PRETERM	880 GMS	YES
16)	1016742	29 YRS	G4P2L2A1	28+2	SEVERE PE	IUFD	NIL	31	160	90	96	3+	19.1	1.1	NIL	NORMAL	2	NIL	VAGINAL	SPONTANEOUS	ATONIC PPH	NO	YES	NO	NO	NO	YES	NO	STABLE	STILLBIRTH	PRETERM	1.1 KG	NA
17)	1013195	18 YRS	PRIMIGRAVIDA	36+3	ECLAMPSIA	NIL	NIL	21.9	160	110	90	2+	44.4	2.8	NORMAL	GRADE 2 HR	2	YES	LSCS	ANTEPARTUM ECLAMPSIA	NIL	YES	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	2.8 KG	NO
18)	1013527	27 YRS	G2P1L1	37	NON SEVERE PE	NIL	NIL	25	130	100	98	1+	35.1	2.98	NORMAL	NORMAL	1	NIL	LSCS	PREVIOUS LSCS	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2.2 KG	NO
19)	1013834	26 YRS	G2P1L1	38+5	SEVERE PE	NIL	NIL	24.3	200	110	98	2+	25.5	1.87	NORMAL	NORMAL	3	NIL	LSCS	FETAL DISTRESS	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2.5 KG	NO
20)	1013956	20 YRS	PRIMIGRAVIDA	34+6	SEVERE PE	NIL	NIL	28	170	130	96	1+	32.3	2.02	NORMAL	NORMAL	1	NIL	LSCS	FETAL DISTRESS	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	1.8 KG	YES
21)	1014128	24 YRS	G2P1L1	27	SEVERE PE	FGR	NIL	24.2	170	100	97	4+	75.8	2.69	NORMAL	GRADE 1 HR	2	YES	VAGINAL	INDUCED	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	STILLBIRTH	PRETERM	780 GMS	NA
22)	1014750	19 YRS	PRIMIGRAVIDA	32+2	ECLAMPSIA	NIL	NIL	28.5	150	110	84	NIL	22.6	4.68	NIL	GRADE 1 HR	2	YES	LSCS	ANTEPARTUM ECLAMPSIA	NIL	YES	NO	YES	NO	NO	NO	YES	STABLE	LIVE	PRETERM	1.4KG	YES
23)	1014876	22 YRS	PRIMIGRAVIDA	40+3	SEVERE PE	NIL	NIL	25.9	170	110	98	1+	26.8	3.19	NORMAL	NORMAL	1	NIL	VAGINAL	INDUCED	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	3 KG	NO
24)	1015127	24 YRS	PRIMIGRAVIDA	40+5	NON SEVERE PE	NIL	NIL	32.8	140	100	99	1+	30.7	1.85	NIL	NORMAL	1	NIL	LSCS	MSL	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	PERINATAL MORTALITY	TERM	2.8 KG	YES
25)	1015124	29 YRS	G3P1L1A1	39	SEVERE PE	NIL	NIL	26.1	170	110	98	1+	20.8	1.78	NORMAL	NIL	1	NIL	VAGINAL	INDUCTION	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2.5 KG	NO
26)	1016594	19 YRS	PRIMIGRAVIDA	36+5	SEVERE PE	IUFD	NIL	26.4	190	100	97	3+	47	67,000	NIL	GRADE 1 HR	2	NIL	LSCS	ABRUPTION	ATONIC PPH	NO	YES	NO	NO	YES	YES	NO	STABLE	STILLBIRTH	PRETERM	2.1 KG	NA
27)	1017587	24 YRS	G2E1	38+4	ECLAMPSIA	FGR	NIL	34.6	180	130	96	1+	56.8	3.37	NIL	NORMAL	2	YES	LSCS	ANTEPARTUM ECLAMPSIA	NIL	YES	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	1.5 KG	YES
28)	1017511	22 YRS	G2P1L1	38+1	SEVERE PE	NIL	RHD	26.4	146	90	98	3+	58	1.27	NORMAL	NORMAL	NIL	NIL	LSCS	SEVERE PE	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2.4 KG	NO
29)	1018545	22 YRS	PRIMIGRAVIDA	39+5	SEVERE PE	NIL	NIL	30	180	110	96	1+	58.3	98,000	NIL	NORMAL	1	NIL	VAGINAL	SPONTANEOUS	ATONIC PPH	NO	NO	NO	YES	NO	NO	NO	STABLE	LIVE	TERM	2.9 KG	NO
30)	1018633	27 YRS	G2P1L1	36+3	SEVERE PE	NIL	NIL	29.5	150	100	98	1+	27.6	1.78	NORMAL	NORMAL	2	NIL	LSCS	SEVERE PE	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	2.5 KG	NO
31)	1019053	27 YRS	G2P1L1	37+5	SEVERE PE	IUFD	NIL	27.5	140	100	92	2+	29.1	98,000	NORMAL	NORMAL	1	NIL	VAGINAL	INDUCED	ATONIC PPH	NO	YES	NO	YES	NO	YES	NO	STABLE	STILLBIRTH	TERM	2.7 KG	NA
32)	1019112	35 YRS	G4P2L2A1	38	SEVERE PE	NIL	NIL	35	160	100	96	1+	132	3.78	NORMAL	NORMAL	1	YES	VAGINAL	SPONTANEOUS	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2.6 KG	NO
33)	1019127	20 YRS	PRIMIGRAVIDA	37+5	SEVERE PE	NIL	NIL	29.3	170	110	97	1+	92.2	2	NORMAL	NORMAL	2	NIL	VAGINAL	SPONTANEOUS	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	1.4 KG	YES
34)	1019208	22 YRS	PRIMIGRAVIDA	36+2	NON SEVERE PE	FGR	NIL	25	140	100	98	1+	39.7	1.6	AEDF	NORMAL	NIL	NIL	LSCS	FGR WITH AEDF	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	1.6 KG	YES
35)	1019295	21 YRS	PRIMIGRAVIDA	29+1	ECLAMPSIA	FGR	NIL	26.1	170	120	96	4+	41.7	3	NIL	NORMAL	2	YES	VAGINAL	INDUCTION	NIL	YES	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	680 GMS	YES
36)	1019464	27 YRS	PRIMIGRAVIDA	39+6	NON SEVERE PE	NIL	NIL	29.7	150	100	98	1+	41.7	2.96	NORMAL	NORMAL	1	NIL	LSCS	FETAL DISTRESS	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2.6 KG	NO
37)	1019410	20 YRS	PRIMIGRAVIDA	37+1	NON SEVERE PE	NIL	NIL	24.5	140	100	98	1+	21.1	2	NORMAL	NORMAL	1	NIL	LSCS	MSL	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2.7 KG	NO
38)	1019544	25 YRS	G2A1	39+1	SEVERE PE	NIL	NIL	34.9	170	100	98	2+	55.1	3.59	NORMAL	NORMAL	2	YES	LSCS	MSL	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	4 KG	NO
39)	1019659	30 YRS	G5P4L2	34+5	ECLAMPSIA	FGR	NIL	27.1	120	90	98	1+	66.9	1	NIL	GRADE 1 HR	2	YES	LSCS	ANTEPARTUM ECLAMPSIA	NIL	YES	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	1.7 KG	YES
40)	1019635	35 YRS	G3P2L2	33+1	ECLAMPSIA	IUFD	NIL	18.2	180	140	92	3+	91.1	96,000	NA	NORMAL	3	YES	VAGINAL	INDUCED	CARDIAC ARREST	YES	NO	YES	YES	NO	NO	YES	CARDIAC ARREST	STILLBIRTH	PRETERM	1.2 KG	NA
41)	1017518	23 YRS	PRIMIGRAVIDA	37+4	NON SEVERE PE	NIL	NIL	22.4	140	90	100	1+	43	3	NORMAL	NORMAL	NIL	NIL	VAGINAL	SPONTANEOUS	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2.8 KG	NO
42)	1017891	19 YRS	PRIMIGRAVIDA	37+6	SEVERE PE	NIL	NIL	20.6	150	110	98	2+	48.7	1.52	NORMAL	NORMAL	1	NIL	LSCS	FETAL DISTRESS	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2.3 KG	NO
43)	1018180	21 YRS	PRIMIGRAVIDA	26	ECLAMPSIA	FGR	NIL	28	150	90	96	3+	19	16,000	NIL	NORMAL	2	YES	LSCS	ANTEPARTUM ECLAMPSIA	NIL	YES	NO	YES	YES	NO	NO	NO	STABLE	STILLBIRTH	PRETERM	400 GMS	NA
44)	5775643	20 YRS	PRIMIGRAVIDA	38+4	SEVERE PE	NIL	NIL	26.4	180	100	98	1+	18.6	2.06	NORMAL	NORMAL	1	NIL	LSCS	SEVERE PE	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2.9 KG	NO
45)	1020096	27 YRS	G4P2L2A1	37+3	SEVERE PE	NIL	HTN IN PREVIOUS PREGNANCY	30	140	90	99	2+	42.7	2.19	NORMAL	NORMAL	1	NIL	VAGINAL	SPONTANEOUS	NIL	NO	NO	NO	YES	NO	NO	NO	STABLE	LIVE	TERM	2.4 KG	NO
46)	1020174	21 YRS	PRIMIGRAVIDA	31	ECLAMPSIA	NIL	NIL	25.2	190	120	96	2+	17.1	4.3	NORMAL	NORMAL	2	YES	LSCS	ANTEPARTUM ECLAMPSIA	NIL	YES	NO	NO	NO	NO	NO	YES	HEMIPLEGIA	LIVE	PRETERM	1 KG	YES
47)	1020210	25 YRS	PRIMIGRAVIDA	29+5	ECLAMPSIA	NIL	NIL	25.1	150	90	97	3+	30.2	1.3	NIL	NORMAL	2	YES	LSCS	ANTEPARTUM ECLAMPSIA	NIL	YES	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	820 GMS	YES

48)	1020229	34 YRS	G3P1L1A1	38+5	SEVERE PE	FGR	NIL	30	160	90	99	2+	29.3	1.75	NORMAL	NORMAL	2	NIL	LSCS	PREVIOUS LSCS	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2.5 KG	NO
49)	1020331	21 YRS	PRIMIGRAVIDA	34	ECLAMPSIA	NIL	NIL	22	150	100	98	1+	38.2	2.34	NIL	GRADE 1 HR	2	YES	LSCS	ANTEPARTUM ECLAMPSIA	NIL	YES	NO	NO	NO	NO	NO	NO	PRES	LIVE	PRETERM	980 GMS	YES
50)	1020381	23 YRS	G3P2L1	29+4	NON SEVERE PE	NIL	NIL	23.4	140	90	100	1+	25.6	1.85	NORMAL	NORMAL	NIL	NIL	VAGINAL	INDUCED	ATONIC PPH	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	1.5 KG	YES
51)	1020521	20 YRS	PRIMIGRAVIDA	31+6	ECLAMPSIA	FGR	NIL	29.2	140	100	94	4+	37.5	2.11	AEDF	NORMAL	3	YES	VAGINAL	INDUCED	NIL	YES	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	970 GMS	YES
52)	1020661	32 YRS	G2P1L1	34+5	SEVERE PE	NIL	NIL	25.4	160	110	99	3+	28.2	96,000	NORMAL	NORMAL	1	NIL	LSCS	HELLP	NIL	NO	NO	NO	YES	NO	NO	NO	STABLE	LIVE	PRETERM	1.9 KG	NO
53)	1020714	26 YRS	G2P1L0	29+2	SEVERE PE	FGR	NIL	22	130	100	100	3+	32	52,000	NORMAL	NORMAL	2	NIL	LSCS	HELLP	NIL	YES	NO	NO	YES	NO	NO	NO	STABLE	LIVE	PRETERM	800 GMS	YES
54)	1020745	21 YRS	G4P1L0A2	36+2	ECLAMPSIA	NIL	NIL	32	180	110	97	2+	40	2.74	NORMAL	GRADE 1 HR	2	YES	LSCS	ANTEPARTUM ECLAMPSIA	NIL	YES	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	1.8 KG	YES
55)	1020829	20 YRS	PRIMIGRAVIDA	40+1	ECLAMPSIA	FGR	NIL	22	160	100	98	3+	52.6	1.61	NORMAL	GRADE 1 HR	2	YES	LSCS	ANTEPARTUM ECLAMPSIA	NIL	YES	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2.1 KG	NO
56)	1021054	20 YRS	PRIMIGRAVIDA	39+6	SEVERE PE	NIL	NIL	29.6	150	100	100	2+	38	2.23	NIL	NORMAL	1	NIL	LSCS	MSL	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2.7 KG	NO
57)	1021063	20 YRS	G2P1L1	37	SEVERE PE	NIL	NIL	25	140	100	96	2+	26	1	NIL	NORMAL	1	NIL	LSCS	PREVIOUS LSCS	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2.4 KG	NO
58)	1021066	24 YRS	PRIMIGRAVIDA	41+1	SEVERE PE	NIL	NIL	24	150	100	98	3+	23.3	2.28	NORMAL	NORMAL	1	NIL	LSCS	SEVERE PE	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	3.4 KG	NO
59)	1021069	23 YRS	PRIMIGRAVIDA	28+6	ECLAMPSIA	IUFD	NIL	19	150	100	93	3+	38.6	1.37	NA	NORMAL	2	YES	LSCS	ANTEPARTUM ECLAMPSIA	INTUBATION	YES	NO	YES	NO	NO	NO	YES	INTUBATED	STILLBIRTH	PRETERM	820 GMS	NA
60)	1021093	28 YRS	G4P2L2A1	37+2	SEVERE PE	FGR	NIL	24	160	96	99	4+	17.4	1.65	NORMAL	NORMAL	2	YES	LSCS	IMMINENT ECLAMPSIA	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	1.3 KG	YES
61)	1021052	38 YRS	G4P3L3	28+6	SEVERE PE	NIL	NIL	27.5	160	110	100	2+	26	1.56	NIL	NORMAL	2	YES	VAGINAL	INDUCED	ATONIC PPH	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	1.16 KG	YES
62)	1021135	36 YRS	G3P2L2	39+3	NON SEVERE PE	NIL	NIL	20.1	140	100	100	1+	25.7	1.96	NORMAL	NORMAL	1	NIL	LSCS	PREVIOUS LSCS	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	3 KG	NO
63)	1021175	21 YRS	PRIMIGRAVIDA	35+1	SEVERE PE	IUFD	NIL	18.2	150	90	99	1+	36	84,000	NORMAL	GRADE 1 HR	1	NIL	LSCS	ABRUPTION	ATONIC PPH	NO	NO	NO	YES	NO	YES	NO	STABLE	STILLBIRTH	PRETERM	1.5 KG	NA
64)	1021181	26 YRS	G2P1L1	33+1	SEVERE PE	IUFD	NIL	19.2	160	110	98	1+	17.9	4.3	NA	NORMAL	2	NIL	VAGINAL	INDUCED	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	STILLBIRTH	PRETERM	1.07 KG	NA
65)	1020904	23 YRS	G2P1L1	29+3	SEVERE PE	NIL	NIL	26.2	160	90	100	1+	23.6	2.46	AEDF	NORMAL	3	NIL	VAGINAL	INDUCED	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	1 KG	YES
66)	1021263	25 YRS	PRIMIGRAVIDA	31	SEVERE PE	NIL	NIL	20.5	200	110	98	4+	46	2.59	NORMAL	NORMAL	2	NIL	LSCS	SEVERE PE	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	1.28 KG	YES
67)	1021496	28 YRS	G3P2L2	29+2	SEVERE PE	FGR	NIL	29	180	120	99	2+	20.2	2.45	NORMAL	GRADE 1 HR	3	NIL	LSCS	PREVIOUS LSCS	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	750 GMS	YES
68)	1021799	34 YRS	G4P2L2A1	33+6	SEVERE PE	IUFD	NIL	24.6	130	94	99	2+	46	76,000	NA	NORMAL	1	NIL	VAGINAL	INDUCED	SHOCK	NO	YES	NO	YES	NO	YES	YES	SHOCK	STILLBIRTH	PRETERM	1.9 KG	NA
69)	1022209	30 YRS	PRIMIGRAVIDA	28+1	ECLAMPSIA	NIL	DIABETES	29.2	140	90	98	2+	120.9	2.3	NIL	NORMAL	3	NIL	LSCS	ANTEPARTUM ECLAMPSIA	ATONIC PPH	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	1.1 KG	YES
70)	1022646	20 YRS	PRIMIGRAVIDA	37+1	SEVERE PE	FGR	NIL	19.4	150	110	100	2+	19.6	3.9	NORMAL	NORMAL	1	NIL	LSCS	FETAL DISTRESS	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	1.35 KG	YES
71)	1023183	28 YRS	G3P1L1A1	33	SEVERE PE	NIL	NIL	18.4	150	110	96	3+	12.6	2.28	NORMAL	NORMAL	2	YES	LSCS	IMMINENT ECLAMPSIA	NIL	NO	NO	NO	NO	NO	YES	NO	STABLE	LIVE	PRETERM	1.4 KG	YES
72)	1023192	20 YRS	PRIMIGRAVIDA	37+5	ECLAMPSIA	FGR	NIL	22.6	130	90	99	1+	7.6	1.1	AEDF	GRADE 2 HR	1	YES	LSCS	ANTEPARTUM ECLAMPSIA	NIL	YES	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	1.48 KG	YES
73)	1023194	20 YRS	PRIMIGRAVIDA	37+5	SEVERE PE	NIL	NIL	20.6	140	90	99	3+	9.3	28,000	NORMAL	NORMAL	1	NIL	LSCS	HELLP	NIL	NO	NO	NO	YES	NO	NO	NO	STABLE	LIVE	TERM	2.4 KG	NO
74)	1023285	34 YRS	G4P2L2A1	33+3	SEVERE PE	IUFD	NIL	22	150	100	100	2+	13	20,000	NIL	NORMAL	1	NIL	VAGINAL	INDUCED	NIL	NO	YES	NO	YES	YES	NO	YES	DIALYSIS	STILLBIRTH	PRETERM	1.46 KG	NA
75)	1023351	30 YRS	G4P3L2	31+1	SEVERE PE	IUFD	NIL	20.4	140	90	98	3+	20	1.12	NA	NORMAL	1	NIL	VAGINAL	INDUCED	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	STILLBIRTH	PRETERM	1.2 KG	NA
76)	1023431	30 YRS	G3P1L1A1	30+3	NON SEVERE PE	IUFD	NIL	24.4	140	90	99	1+	26	2.9	NA	NORMAL	NIL	NIL	VAGINAL	INDUCED	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	STILLBIRTH	PRETERM	480 GMS	NA
77)	1023588	19 YRS	PRIMIGRAVIDA	40+1	SEVERE PE	NIL	NIL	30	140	100	99	4+	34	3.02	NORMAL	NORMAL	1	YES	LSCS	IMMINENT ECLAMPSIA	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	3.9 KG	NO
78)	1023559	27 YRS	G2P1L1	40+2	SEVERE PE	NIL	NIL	22.4	150	100	98	2+	17.1	1.09	NORMAL	NORMAL	1	NIL	LSCS	NON-PROGRESS OF LABOUR	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	3.3 KG	NO
79)	1023718	27 YRS	PRIMIGRAVIDA	38+2	SEVERE PE	NIL	NIL	19.8	150	100	100	3+	42	74,000	NORMAL	NORMAL	2	NIL	LSCS	HELLP	NIL	NO	NO	NO	YES	NO	NO	NO	STABLE	LIVE	TERM	2.3 KG	NO
80)	1023814	23 YRS	PRIMIGRAVIDA	35+$	SEVERE PE	NIL	NIL	26.4	180	110	96	1+	14.7	3.12	NORMAL	NORMAL	1	NIL	VAGINAL	SPONTANEOUS	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	2.4 KG	NO
81)	1023497	20 YRS	PRIMIGRAVIDA	36+3	SEVERE PE	FGR	NIL	28	160	100	98	1+	14.5	1.5	NORMAL	GRADE 2 HR	1	NIL	LSCS	SEVERE PE	ATONIC PPH	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	2.3KG/2.5KG	NO
82)	1024110	23 YRS	PRIMIGRAVIDA	33	SEVERE PE	NIL	NIL	22.4	140	100	100	3+	28.2	2.74	NORMAL	NORMAL	1	NIL	LSCS	IMMINENT ECLAMPSIA	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	1.4 KG	YES
83)	1024323	23 YRS	PRIMIGRAVIDA	38+3	SEVERE PE	FGR	NIL	18.6	160	100	99	2+	34	1.7	INCREASED RESISTANCE	NORMAL	1	NIL	LSCS	BREECH	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2 KG	NO
84)	1024335	26 YRS	PRIMIGRAVIDA	32+6	SEVERE PE	NIL	NIL	24.2	160	110	98	1+	38	1.5	NORMAL	NORMAL	2	YES	LSCS	IMMINENT ECLAMPSIA	NIL	NO	NO	NO	YES	NO	NO	NO	STABLE	LIVE	PRETERM	1.9 KG	YES
85)	1024315	31 YRS	G2P1L1	34+3	NON SEVERE PE	FGR	NIL	20.2	130	90	100	1+	21.8	1.5	NORMAL	NORMAL	1	NIL	LSCS	SEVERE PE	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	1.6 KG	YES
86)	1024603	24 YRS	PRIMIGRAVIDA	37+5	ECLAMPSIA	NIL	NIL	18	160	100	98	3+	152	3.5	NIL	GRADE 1 HR	2	YES	LSCS	ANTEPARTUM ECLAMPSIA	NIL	YES	NO	YES	NO	NO	NO	NO	STABLE	LIVE	TERM	2.7 KG	NO
87)	1024751	28 YRS	G2P1L1	41+5	NON SEVERE PE	NIL	NIL	22.6	140	90	100	1+	21.5	2.46	NORMAL	NORMAL	1	NIL	LSCS	FETAL DISTRESS	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	3.2 KG	NO
88)	1024944	21 YRS	G2A1	39	SEVERE PE	NIL	NIL	20.4	150	100	99	3+	25.5	1.75	NORMAL	NORMAL	1	YES	VAGINAL	INDUCTION	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2.5 KG	NO
89)	1025773	26 YRS	PRIMIGRAVIDA	35+3	SEVERE PE	FGR	DIABETES	28.2	150	100	98	2+	13.2	2.26	INCREASED RESISTANCE	NORMAL	2	NIL	LSCS	FETAL DISTRESS	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	1.3 KG	YES
90)	1025909	30 YRS	G2P1L1	38+2	NON SEVERE PE	NIL	NIL	21	150	90	100	1+	23.9	1.91	NORMAL	NORMAL	1	NIL	LSCS	PREVIOUS LSCS	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2.5 KG	NO
91)	1026794	26 YRS	G2P1L1	38+4	MILD PE	NIL	NIL	19.8	150	100	99	1+	23.9	2.24	NORMAL	NORMAL	1	NIL	LSCS	PREVIOUS LSCS	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	3.9 KG	NO
92)	1026899	25 YRS	PRIMIGRAVIDA	38+5	MILD PE	NIL	NIL	22.4	140	100	100	1+	16	3.42	INCREASED RESISTANCE	NORMAL	1	NIL	VAGINAL	INDUCED	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2.7 KG	NO
93)	1027130	27 YRS	G4P2L2A1	38+6	MILD PE	FGR	NIL	26.2	130	90	100	1+	16	1.66	NORMAL	NORMAL	1	NIL	LSCS	PREVIOUS LSCS	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	1.3 KG	YES
94)	1027026	34 YRS	G3P1L1A1	37+4	SEVERE PE	NIL	NIL	20.4	140	100	98	2+	26.1	1.64	NIL	NORMAL	2	NIL	LSCS	IMMINENT ECLAMPSIA	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LLVE	TERM	2.7 KG	NO
95)	1027093	23 YRS	G2A1	37	SEVERE PE	IUFD	HIV	24.2	180	110	98	2+	30.2	3.13	NIL	NORMAL	1	NIL	VAGINAL	INDUCED	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	STILLBIRTH	TERM	2.4 KG	NA
96)	1026769	23 YRS	G3P1L1A1	33+3	SEVERE PE	NIL	NIL	20.6	150	100	98	1+	28.4	1.36	NORMAL	GRADE 1	1	NIL	LSCS	SEVERE PE	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	1.8 KG	NO
97)	1027286	26 YRS	G3A2	29+6	SEVERE PE	IUFD	NIL	26.4	160	100	98	2+	16.1	2.97	NIL	NORMAL	1	NIL	VAGINAL	INDUCED	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	STILLBIRTH	PRETERM	1.5 KG	NA
98)	1027771	28 YRS	G2P1L1	38+6	SEVERE PE	NIL	NIL	20.2	170	100	98	2+	12.2	1.79	NORMAL	NORMAL	2	NIL	LSCS	PREVIOUS LSCS	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2.6 KG	NO

99)	1027767	20 YRS	PRIMIGRAVIDA	34+4	ECLAMPSIA	NIL	NIL	26.8	160	100	97	2+	17.8	3.19	NORMAL	NORMAL	2	YES	LSCS	ECLAMPSIA	NIL	YES	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	1.3 KG	YES
100)	1027923	24 YRS	G3A2	37	SEVERE PE	FGR	NIL	20.8	170	100	98	2+	21.5	1.91	INCREASED RESISTANCE	GRADE 2	2	NIL	LSCS	BREECH	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	1.6 KG	YES
101)	1027138	34 YRS	G3A2	32+3	SEVERE PE	NIL	RHEUMATOID ARTHRITIS	22.6	160	100	98	1+	10.4	2.96	NORMAL	GRADE 1	2	NIL	LSCS	SEVERE PE	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	1.4 KG	NO
102)	1028055	21 YRS	PRIMIGRAVIDA	39	SEVERE PE	NIL	NIL	18.2	140	80	99	2+	41.3	3.63	NORMAL	NORMAL	1	NIL	LSCS	SEVERE PE	NIL	NO	NO	NO	NO	YES	NO	NO	STABLE	LIVE	TERM	2.5 KG	NO
103)	1028063	25 YRS	G3P2L2	32	SEVERE PE	FGR	NIL	20.4	200	100	96	3+	48.4	2.69	NORMAL	GARDE 1	2	NIL	LSCS	PREVIOUS LSCS	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	1.2 KG	YES
104)	1028401	23 YRS	PRIMIGRAVIDA	39+5	SEVERE PE	NIL	NIL	26.4	150	100	99	3+	30.2	2.38	NORMAL	NORMAL	2	NIL	VAGINAL	SPONTANEOUS	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2.4 KG	NO
105)	1028533	32 YRS	G4P3L3	35	SEVERE PE	NIL	NIL	20.4	170	110	98	3+	48.6	63000	NORMAL	NORMAL	1	NIL	LSCS	HELLP SYNDROME	NIL	NO	NO	NO	YES	NO	NO	NO	STABLE	LIVE	PRETERM	1.68 KG	YES
106)	1028534	23 YRS	PRIMIGRAVIDA	32	SEVERE PE	IUFD	NIL	19.4	140	110	96	1+	28.6	1.63	NIL	NORMAL	1	NIL	VAGINAL	INDUCED	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	STILLBIRTH	PRETERM	700 GMS	NA
107)	1028784	20 YRS	PRIMIGRAVIDA	36+4	SEVERE PE	NIL	NIL	20	130	90	100	1+	30.2	23000	NORMAL	NORMAL	1	NIL	LSCS	CEPHALOPELVIC DISPROPORTION	ATONIC PPH	NO	NO	NO	YES	NO	NO	NO	STABLE	LIVE	PRETERM	2.1 KG	YES
108)	1028685	24 YRS	PRIMIGRAVIDA	31+4	SEVERE PE	NIL	NIL	22.4	170	110	98	4+	50.4	1.82	NORMAL	GRADE 2	2	YES	LSCS	IMMINENT ECLAMPSIA	NIL	YES	NO	NO	NO	NO	NO	NO	STABLE	LIVE- PERINATAL	PRETERM	600 GMS	YES
109)	1031142	28 YRS	G3P2L2	40	SEVERE PE	NIL	NIL	18.4	180	110	94	1+	28.6	2.45	NORMAL	NORMAL	2	YES	LSCS	IMMINENT ECLAMPSIA	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2.8 KG	NO
110)	1031740	20 YRS	PRIMIGRAVIDA	39	SEVERE PE	NIL	NIL	20	150	90	100	1+	23.8	1.8	NORMAL	NORMAL	1	NIL	LSCS	NON-PROGRESS OF LABOUR	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	3.1 KG	NO
111)	1031819	22 YRS	PRIMIGRAVIDA	38	SEVERE PE	IUFD	NIL	24.6	170	110	98	1+	30.7	76000	NIL	NORMAL	2	NIL	VAGINAL	INDUCED	NIL	NO	NO	NO	YES	NO	NO	NO	STABLE	STILLBIRTH	TERM	1.8 KG	NA
112)	1032428	28 YRS	G2P1L1	40+2	SEVERE PE	NIL	NIL	20.2	170	110	98	1+	17.6	1.99	NORMAL	NORMAL	1	NIL	LSCS	SEVERE PE	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	3.5 KG	NO
113)	1028957	29 YRS	PRIMIGRAVIDA	37+6	MILD PE	NIL	NIL	18.8	140	90	100	1+	40.7	2.44	NORMAL	NORMAL	1	NIL	LSCS	CDMR	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2.4 KG	NO
114)	1029258	30 YRS	PRIMIGRAVIDA	35+6	ECLAMPSIA	FGR	NIL	24.8	140	100	98	2+	61.4	96,000	INCREASED RESISTANCE	NORMAL	2	YES	LSCS	ANTEPARTUM ECLAMPSIA	METABOLIC ACIDOSIS	YES	NO	NO	YES	NO	NO	NO	VENTILATOR	LIVE	PRETERM	1.6 KG	YES
115)	1036439	27 YRS	G3P2L1	34+6	MILD PE	NIL	HISTORY OF PE	20.3	150	100	100	1+	81.1	2.23	NORMAL	NORMAL	1	NIL	LSCS	PREVIOUS LSCS	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	2.1 KG	YES
116)	1036166	23 YRS	PRIMIGRAVIDA	26+3	SEVERE PE	NIL	NIL	20	140	100	99	1+	54.6	2.41	NIL	GRADE 1	1	NIL	VAGINAL	INDUCED	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	STILLBIRTH	PRETERM	700 GMS	NA
117)	1037814	30 YRS	PRIMIGRAVIDA	37+1	MILD PE	NIL	DIABETES	28.6	130	90	100	1+	52.4	2.6	NORMAL	NORMAL	1	NIL	LSCS	FETAL DISTRESS	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2.9 KG	NO
118)	1039152	22 YRS	G3A2	39+2	MILD PE	NIL	NIL	20.4	140	90	100	1+	25.5	2.34	NORMAL	NORMAL	0	NIL	LSCS	FETAL DISTRESS	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	3.1 KG	NO
119)	1039984	24 YRS	PRIMIGRAVIDA	34	SEVERE PE	NIL	NIL	24.8	150	100	98	2+	48.6	2.2	NORMAL	GRADE 1	1	NIL	LSCS	SEVERE PE	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	1.8 KG	NO
120)	1040176	22 YRS	PRIMIGRAVIDA	26+4	ECLAMPSIA	NIL	NIL	19.4	170	100	96	3+	19.2	3.04	NIL	GRADE 2	1	YES	VAGINAL	INDUCED	NIL	YES	NO	NO	NO	NO	NO	NO	STABLE	STILLBIRTH	PRETERM	750 GMS	NA
121)	1039768	22 YRS	G2A1	36	MILD PE	NIL	NIL	20	140	90	100	1+	39.3	2.04	NORMAL	NORMAL	1	NIL	LSCS	CDMR	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2.9 KG	NO
122)	1044036	22 YRS	G2P1L1	27+5	ECLAMPSIA	NIL	NIL	24.6	140	90	100	4+	18.5	1.64	NIL	NORMAL	1	YES	VAGINAL	INDUCED	NIL	YES	NO	NO	NO	NO	NO	NO	STABLE	STILLBIRTH	PRETERM	900 GMS	NA
123)	1045372	28 YRS	PRIMIGRAVIDA	30+2	SEVERE PE	FGR	NIL	19.8	160	100	98	2+	30.1	3.03	AEDF	NORMAL	2	YES	VAGINAL	INDUCED	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	STILLBIRTH	PRETERM	810 GMS	NA
124)	1047604	29 YRS	N	35+6	MILD PE	FGR	NIL	30	140	90	98	1+	30.1	1.5	INCREASED RESISTANCE	NORMAL	2	YES	LSCS	FGR	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	1.4 KG	YES
125)	1048216	30 YRS	G2P1L1	28+3	SEVERE PE	NIL	NIL	24.2	120	80	99	1+	50.2	2.93	AEDF	NORMAL	2	NIL	VAGINAL	INDUCED	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	810 GMS	YES
126)	1048683	24 YRS	PRIMIGRAVIDA	31+1	SEVERE PE	IUFD	NIL	26.2	140	90	96	3+	90	43000	NORMAL	NORMAL	1	NIL	LSCS	ABRUPTION	ATONIC PPH	NO	NO	NO	NO	YES	YES	YES	STABLE	STILLBIRTH	PRETERM	1.5 KG	NA
127)	1048794	21 YRS	PRIMIGRAVIDA	31+3	ECLAMPSIA	NIL	NIL	30.2	130	80	92	3+	30.2	1.58	NORMAL	NORMAL	1	YES	LSCS	ECLAMPSIA	NIL	YES	NO	NO	NO	NO	NO	NO	STABLE	DIED ON DAY 2	PRETERM	1.4 KG	YES
128)	1048821	20 YRS	PRIMIGRAVIDA	27+6	SEVERE PE	NIL	NIL	24.2	140	100	98	2+	62.4	2.3	NORMAL	NORMAL	1	NIL	LSCS	SEVERE PE	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	1 KG	YES
129)	1049593	36 YRS	G3P1L1A1	34+1	SEVERE PE	NIL	NIL	20.4	160	100	99	3+	18.7	73,000	NORMAL	NORMAL	1	NIL	LSCS	HELLP SYNDROME	NIL	NO	NO	NO	YES	NO	NO	NO	STABLE	LIVE	PRETERM	1.3 KG	YES
130)	1050975	21 YRS	PRIMIGRAVIDA	41+3	SEVERE PE	NIL	NIL	24.6	180	100	98	3+	34.6	2.8	NORMAL	NORMAL	1	NIL	LSCS	SEVERE PE	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2.9 KG	NO
131)	1051174	25 YRS	G2P1L1	35+3	SEVERE PE	NIL	NIL	26.4	170	100	98	3+	98.3	1.44	NORMAL	GRADE 1	1	YES	LSCS	SEVERE PE	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2 KG	NO
132)	1051147	20 YRS	PRIMIGRAVIDA	33+2	MILD PE	FGR	NIL	22.6	150	90	99	1+	88.3	2.97	NORMAL	NORMAL	1	NIL	VAGINAL	INDUCED	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	1 KG	YES
133)	1051269	22 YRS	PRIMIGRAVIDA	38+2	MILD PE	NIL	NIL	20.4	140	90	98	1+	68.2	2.29	NORMAL	NORMAL	1	NIL	VAGINAL	INDUCED	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2.9 KG	NO
134)	1051563	20 YRS	PRIMIGRAVIDA	32+5	ECLAMPSIA	NIL	NIL	24.6	140	82	98	2+	48.1	2.17	NORMAL	NORMAL	1	NIL	VAGINAL	INDUCED	NIL	YES	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	1.36 KG	YES
135)	1050473	32 YRS	PRIMIGRAVIDA	33+3	SEVERE PE	NIL	NIL	28.6	140	90	98	1+	60.4	2.44	NORMAL	NORMAL	2	NIL	LSCS	FETAL DISTRESS	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	2.2 KG	YES
136)	1052678	20 YRS	G2P1L1	40+2	MILD PE	NIL	NIL	18.8	150	90	99	1+	34.9	2.46	NIL	NORMAL	1	NIL	VAGINAL	SPONTAENOUS	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2.8 KG	NO
137)	1052775	27 YRS	PRIMIGRAVIDA	39+6	MILD PE	NIL	NIL	26.4	150	100	98	1+	59.3	2.49	NIL	NORMAL	1	NIL	LSCS	DEEP TRANSVERSE ARREST	ATONIC PPH	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	3 KG	NO
138)	1053068	19 YRS	PRIMIGRAVIDA	40+5	MILD PE	NIL	NIL	21.4	120	80	99	1+	65.9	2.49	NORMAL	NORMAL	1	NIL	VAGINAL	SPONTANEOUS	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	3 KG	NO
139)	1053528	22 YRS	PRIMIGRAVIDA	40+5	SEVERE PE	FGR	NIL	28.4	150	110	96	2+	90.2	78000	NORMAL	GRADE 1	1	NIL	LSCS	FGR	NIL	NO	NO	NO	YES	NO	NO	NO	STABLE	LIVE	TERM	1.7 KG	YES
140)	1053552	24 YRS	G3P2L1	31+2	SEVERE PE	FGR	NIL	30.2	130	90	98	2+	88.6	2.71	AEDF	NORMAL	1	NIL	LSCS	IMMINENT ECLAMPSIA	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	1.3 KG	YES
141)	1053591	21 YRS	PRIMIGRAVIDA	36+3	SEVERE PE	FGR	NIL	22.8	150	100	98	3+	90.2	2.52	NORMAL	NORMAL	1	NIL	LSCS	ABRUPTION	NIL	NO	NO	NO	NO	NO	YES	NO	STABLE	LIVE	PRETERM	1.9 KG	YES
142)	1053797	30 YRS	G3A2	32	SEVERE PE	FGR	NIL	20.2	180	100	99	3+	100.2	2.47	NORMAL	NORMAL	1	YES	LSCS	ABRUPTION	NIL	NO	NO	NO	NO	NO	YES	NO	STABLE	LIVE	PRETERM	1.2 KG	YES
143)	1053909	26 YRS	G2P1L1	37+6	ECLAMPSIA	NIL	NIL	26.4	180	120	96	3+	98.4	67000	NORMAL	GRaDE 2	2	YES	LSCS	ANTEPARTUM ECLAMPSIA	NIL	YES	NO	NO	YES	NO	NO	NO	STABLE	LIVE	TERM	1.4 KG	YES
144)	1053880	22 YRS	PRIMIGRAVIDA	40+3	MILD PE	NIL	DIABETES	27.5	150	90	99	1+	43.2	3.13	NORMAL	NORMAL	1	NIL	VAGINAL	SPONTANEOUYS	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2.3 KG	NO
145)	1054131	38 YRS	G2P1L1	40+3	SEVERE PE	NIL	NIL	20.4	160	100	100	2+	11.5	1.7	NORMAL	NORMAL	1	NIL	LSCS	SEVERE PE	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	3.3 KG	NO
146)	1054185	24 YRS	PRIMIGRAVIDA	38	ECLAMPSIA	NIL	NIL	18.8	150	100	98	2+	80.8	2.01	NORMAL	NORMAL	2	YES	LSCS	ANTEPARTUM ECLAMPSIA	NIL	YES	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2.7 KG	NO
147)	1054393	26 YRS	G3A2	33+6	ECLAMPSIA	NIL	NIL	24.4	200	110	92	2+	68.2	2.67	NORMAL	NORMAL	1	YES	LSCS	ANTEPARTUM ECLAMPSIA	NIL	YES	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	2.6 KG	NO
148)	1054464	30 YRS	G4P2L2A1	36+3	SEVERE PE	FGR	NIL	20	160	110	98	3+	17	2.71	NORMAL	NORMAL	2	YES	LSCS	SEVERE PE	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	1.9 KG	YES
149)	1054539	20 YRS	PRIMIGRAVIDA	39	SEVERE PE	NIL	NIL	19.8	150	90	100	1+	36.3	1.8	NORMAL	NORMAL	1	NIL	LSCS	CEPHALOPELVIC DISPROPORTION	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	3.1 KG	NO

150)	1054807	22 YRS	PRIMIGRAVIDA	38	SEVERE PE	IUFD	NIL	28.6	170	110	96	1+	40.4	35000	NIL	GRADE 1	2	NIL	VAGINAL	INDUCED	NIL	NO	NO	NO	YES	NO	NO	NO	STABLE	STILLBIRTH	TERM	1.8 KG	NA
151)	1055036	30 YRS	G2A1	30+4	ECLAMPSIA	FGR	NIL	20.4	140	100	98	2+	48.6	65000	NIL	GRADE 2	2	YES	LSCS	ECLAMPSIA	NIL	YES	NO	NO	YES	NO	NO	NO	STABLE	LIVE	PRETERM	1.2 KG	YES
152)	1055390	21 YRS	G2P1L1	38+3	MILD PE	NIL	NIL	26.4	130	90	100	1+	78.1	2.66	NORMAL	NORMAL	1	NIL	VAGINAL	SPONTAENOUS	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2.8 KG	NO
153)	1055925	27 YRS	PRIMIGRAVIDA	40	ECLAMPSIA	NIL	NIL	18.2	150	90	94	3+	94.2	1.66	NORMAL	NORMAL	2	YES	LSCS	ECLAMPSIA	CONVULSIONS	YES	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2.8 KG	NO
154)	1055993	22 YRS	PRIMIGRAVIDA	37	SEVERE PE	FGR	NIL	23.4	170	120	98	2+	51.5	2.33	NORMAL	NORMAL	2	YES	LSCS	SEVERE PE	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	1.3 KG	YES
155)	1055848	21 YRS	PRIMIGRAVIDA	35+5	MILD PE	FGR	NIL	20.2	160	100	100	1+	62.9	2.08	NORMAL	NORMAL	1	NIL	LSCS	BREECH	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2 KG	NO
156)	1056288	20 YRS	PRIMIGRAVIDA	39+1	SEVERE PE	NIL	NIL	18.6	170	110	99	4+	40.2	2.97	NORMAL	NORMAL	1	YES	LSCS	IMMINENT ECLAMPSIA	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2.6 KG	NO
157)	1056805	26 YRS	G3P2L2	35+5	ECLAMPSIA	FGR	NIL	28.6	140	100	98	1+	26.2	2.86	NORMAL	GRADE 2	1	YES	LSCS	ECLAMPSIA	NIL	YES	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2.2 KG	NO
158)	1056888	19 YRS	PRIMIGRAVIDA	37+6	ECLAMPSIA	NIL	NIL	26.2	170	100	100	2+	36	2.26	NORMAL	GRADE 1	1	YES	LSCS	ECLAMPSIA	NIL	YES	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2.1 KG	NO
159)	1056871	24 YRS	G2P1L1	38+6	SEVERE PE	FGR	NIL	18.8	150	100	100	2+	46	93000	INCREASED RESISTANCE	NORMAL	1	NIL	LSCS	FETAL DISTRESS	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	1.7 KG	YES
160)	1056850	23 YRS	PRIMIGRAVIDA	28+6	SEVERE PE	IUFD	NIL	18.8	170	100	100	2+	36	2.39	NIL	GARDE 1	1	NIL	VAGINAL	INDUCED	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	STILLBIRTH	PRETERM	1 KG	NA
161)	1057224	22 YRS	G2P1L1	33+4	SEVERE PE	NIL	NIL	22.4	150	100	99	2+	46	46000	NORMAL	NORMAL	1	NIL	VAGINAL	INDUCED	NIL	NO	NO	NO	YES	NO	NO	NO	STABLE	LIVE	PRETERM	2.2 KG	NO
162)	1059084	24 YRS	PRIMIGRAVIDA	28	ECLAMPSIA	NIL	NIL	20	150	90	100	1+	28.8	2.95	NIL	NORMAL	1	YES	VAGINAL	INDUCED	NIL	YES	NO	NO	NO	NO	NO	NO	STABLE	STILLBIRTH	PRETERM	1 KG	NA
163)	1059512	27 YRS	PRIMIGRAVIDA	42+1	ECLAMPSIA	NIL	NIL	28.6	160	110	94	1+	60.4	1.48	NIL	NORMAL	2	YES	VAGINAL	SPONTANEOUS	ATONIC PPH	YES	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2.5 KG	NO
164)	1059500	24 YRS	PRIMIGRAVIDA	30	SEVERE PE	NIL	NIL	20.4	180	110	98	2+	46.2	2.79	NORMAL	NORMAL	3	YES	LSCS	SEVERE PE	ATONIC PPH	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	1.4 KG	YES
165)	1060665	21 YRS	G2P1L1	32	ECLAMPSIA	IUFD	NIL	20.2	140	90	94	3+	50.2	2.76	NIL	NORMAL	2	YES	VAGINAL	INDUCED	NIL	YES	NO	NO	NO	NO	NO	NO	STABLE	STILLBIRTH	PRETERM	1.2 KG	NA
166)	1060887	25 YRS	PRIMIGRAVIDA	38+2	SEVERE PE	NIL	NIL	22.8	180	110	98	3+	28.6	3.19	NORMAL	NORMAL	1	NIL	LSCS	SEVERE PE	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2.7 KG	NO
167)	1062450	30 YRS	PRIMIGRAVIDA	32+2	SEVERE PE	FGR	NIL	28.6	170	110	96	3+	40.8	1.32	AEDF	GRADE 1	1	NIL	VAGINAL	INDUCED	NIL	NO	NO	NO	NO	YES	NO	NO	STABLE	STILLBIRTH	PRETERM	1 KG	NA
168)	1063269	20 YRS	PRIMIGRAVIDA	38+1	SEVERE PE	NIL	NIL	20.2	140	90	100	1+	30.4	1.93	NORMAL	GARDE 1	1	YES	LSCS	IMMINENT ECLAMPSIA	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	TERM	2.6 KG	NO
169)	1063286	36 YRS	G2P1L1	28+4	SEVERE PE	NIL	NIL	20.4	160	100	100	3+	25.1	2.99	AEDF	GRADE 2	3	NIL	LSCS	SEVERE PE	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	LIVE	PRETERM	920 GMS	YES
170)	1066485	25 YRS	G2P1L1	34+5	SEVERE PE	IUFD	NIL	22.8	170	110	100	1+	21	2.66	NIL	NORMAL	1	NIL	VAGINAL	INDUCED	NIL	NO	NO	NO	NO	NO	NO	NO	STABLE	STILLBIRTH	PRETERM	1.4 KG	NO