

**“A PROSPECTIVE OBSERVATIONAL STUDY OF MATERNAL RISK  
FACTORS AND PERINATAL OUTCOME IN FETAL GROWTH  
RESTRICTION AT TERTIARY CARE HOSPITAL**

Dissertation submitted to  
The Tamil Nadu Dr. M.G.R Medical University  
in partial fulfilment of the requirement for the award of

MS BRANCH II  
OBSTETRICS AND GYNAECOLOGY

**REGISTER NO. 221916883**



**DEPARTMENT OF OBSTETRICS AND GYNAECOLOGY  
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**MAY 2022**

## **CERTIFICATE**

This is to certify that this dissertation titled — **“A PROSPECTIVE OBSERVATIONAL STUDY OF MATERNAL RISK FACTORS AND PERINATAL OUTCOME IN FETAL GROWTH RESTRICTION AT TERTIARY CARE HOSPITAL”** is a bonafide work of **DR. PUSHPALAKHSMI. R.** and has been prepared under my guidance, in partial fulfillment of regulations of The Tamilnadu Dr. M.G.R. Medical University, for the award of M.S. Degree in Obstetrics and Gynecology during the year 2019 - 2022.

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## **DECLARATION BY THE CANDIDATE**

I hereby declare that this dissertation / thesis entitled — **“A PROSPECTIVE OBSERVATIONAL STUDY OF MATERNAL RISK FACTORS AND PERINATAL OUTCOME IN FETAL GROWTH RESTRICTION AT TERTIARY CARE HOSPITAL”** is a bonafide and genuine research work carried out by me under the guidance of **PROF DR. N. HEMALATHA, MD., D.G.O.**, Department of Obstetrics and Gynecology, MADRAS MEDICAL COLLEGE, Chennai.

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## ACKNOWLEDGEMENT

On completion of this contribution of scientific document it gives me immense and deep pleasure to acknowledge the guidance provided by my distinguished mentors.

With privilege and respect I would like to express my gratitude to my guide, **PROF DR. N.HEMALATHA ,MD., D.G.O.**, Professor, Department of Obstetrics & Gynaecology, **Institute of Obstetrics and Gynaecology, Madras Medical College & Government General Hospital, Chennai** for her constant support & guidance in doing this study.

I am forever grateful and thankful to my beloved **DIRECTOR AND PROFESSOR DR. S.VIJAYA, M.D., D.G.O** , Director, Institute of Obstetrics and Gynaecology, Madras Medical College, Egmore, Chennai for her valuable and constant support & guidance in doing this study.

I owe by thanks to **PROF. DR. E. THERANIRAJAN, M.D., DCH., MRCPCH (UK),FRCPCH(UK)** , **DEAN** , Madras Medical College , for permitting me to conduct this study in this esteemed Hospital and Institution.

I thank all my Unit Chiefs, Assistant Professors and my Co-postgraduates in the Department of Obstetrics & Gynaecology for their timely advice and support.

I extend my thankfulness to all the patients and their families who agreed to participate in my study.

I am forever thankful to all my family members and my friends who extended their support and helped me to complete my research work.

**Dr. PUSHPALAKSHMI. R**

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STUDY TOPIC: A PROSPECTIVE OBSERVATIONAL STUDY OF  
MATERNAL RISK FACTORS AND PERINATAL OUTCOME IN FETAL  
GROWTH RESTRICTION IN TERTIARY CARE HOSPITAL

**AIMS AND OBJECTIVES:**

- To study maternal risk factors and perinatal outcome in fetal growth restriction.
- To correlate with perinatal morbidity and mortality associated with fetal growth restriction.
- To study outcome of labour in fetal growth restrictions

**BACKGROUND**

Every fetus has its own growth potential and its own growth rate” JAMESM.TANNER

Fetal growth restriction undoubtedly remains the most challenging areas of research for obstetricians today.it is a major contributor of perinatal morbidity and mortality and has been described as etiologically responsible of about a 50% of perinatal deaths occurring preterm and 20% at term.despite of marked progress

made over the past two decades in both diagnostic procedures and management strategies, the question of what causes fetal growth restriction remains unanswered still in 30-40% of all cases of FGR. There are so many causes contributing to FGR –Maternal, fetal and placental causes.

## **METHODOLOGY**

This study was conducted in Institute of Obstetrics and Gynaecology, EGMORE, a Tertiary care teaching hospital for a period of one year. The study population consisted of 100 women with singleton pregnancies who gave birth to neonates with birthweight less than the 10th percentile. {include from methods inclusion criteris, exclusion criteria}. It's a prospective observational study Carried out in all pregnant women admitted as IUGR between 28-40 weeks of gestation.

## **RESULTS**

Incidence of FGR was 9.8% in present study. The incidence of FGR according to national perinatal database was 9.65% among neonates. Majority of mothers were in 26-35 years age group (59%) similar to Satyavrathan and Singh A et al.

The rate of FGR in developing countries is six times higher comparing to developed countries.hypertensive disorders of pregnancy (64%),idiopathic causes(24%),hypothyroid,diabetes,APLA were noted in FGR mothers similar to sharma et al andsatyavarthan etal

Cesarean mode of delivery remains the primary one.24% of cases delivered vaginally comparing to 32% as in Seal et al.imminent eclampsia,HELLP syndrome and eclampsia accounted for IMMEDIATE TERMINATION.the most common indication of termination was fetal distress(29%),severe oligohydramnios(40%),doppler changes which is observed in Rajarajeswari et al study.

FGR diagnosed between 32-34 weeks majority as with Seal et al.in Lekshmietal, 60% of the FGR born to the mothers<37weeks and 29%<32 weeks,while in ours 44% in 35-37 weeks and 32% were bornin 30-35 weeks



## INTRODUCTION

“Every fetus has its own growth potential and its own growth rate”

- JAMES M.TANNER

Fetal growth restriction undoubtedly remains the most challenging areas of research for obstetricians today.it is a major contributor of perinatal morbidity and mortality and has been described as etiologically responsible of about a 50% of perinatal deaths occurring preterm and 20% at term. Despite of marked progress made over the past two decades in both diagnostic procedures and management strategies, the question of what causes fetal growth restriction remains unanswered still in 30-40% of all cases of FGR. There are so many causes contributing to FGR –Maternal, fetal and placental causes.

In 1963,Gruenwald said that among the infants with low birth weight, one third of babies were secondary to “ chronic placental insufficiency”

Usher and McLean projected that standards for fetal growth should be based on the limits defined by +- 2 SD from normal limits.it is seemed most appropriate ,since adverse fetal outcomes were most marked when

the birth weight was below the third percentile.

Owen and colleagues in 1997 and Khan and Owen in 1998 reported that decrease rate of fetal growth in serial biometry is proportionate to cesarean sections done for fetal distress and for significant fetal growth restriction.

Martin et al proved that 8.1% of newborn babies weighed less than 2.5kg at birth and nearly 8% weighed more than 4kg at birth.

The main aim of our study is analysing the maternal and placental risk factors and perinatal outcome in FGR.

#### INCIDENCE OF FGR-3-10%

While the incidence of SGA depends on the threshold of percentile used, the incidence of FGR varies greatly In literature,ranging from 1-12% Approximately only a 20-30% of all SGA fetuses are true growth restricted fetuses.

FGR can lead to increased perinatal morbidity and mortality and impaired neurodevelopment. There is high chance of intrauterine fetal demise, intrapartum fetal distress, and operative deliveries in FGR.

In early preterm FGR, iatrogenic prematurity remains a pertinent issue. FGR associated with doppler changes suggested hemodynamic redistribution as a reflection of the adaptation of the fetus to the undernutrition, hypoxia, placental insufficiency and higher a risk of preeclampsia.

Prediction of risk, timely detection of compromised fetus, strict surveillance, and optimising time of delivery remains the primary aim of our study.

As early antenatal detection, by choosing optimal time and the method of delivery and intervention when it is required could play a role in minimising adverse perinatal outcome significantly. Ultrasonogram is done frequently in antenatal period to assess fetal size with serial measurements of fetal biometry.

Biophysical profile(BPP) and AFI plays the role.the combination of these with obstetric doppler is the best available tool for identification of small fetuses at risk of adverse outcomes.

CPR is calculated by dividing the middle cerebral artery PI by the umbilical artery PI and reflects in a combined fashion mild increases in placental resistance with reductions in fetal brain vascular resistance.

Before 34 weeks,FGR associated with doppler abnormality affects Umbilical artery S/D RATIO>3.0 followed by worsening of doppler manifested as absent end diastolic flow (ADEF) and reversal of end diastolic flow (REDF)

Doppler velocimetry not only decides the optimum time of delivery but also the optimum mode of delivery.

The study was undertaken to evaluate the sociodemographic variables,maternal risk factors,diagnosis-delivery interval ,mode of delivery ,birth weight and indications of operative delivery in FGR.Perinatal morbidity was assessed in terms of NICU stay,need for resuscitation and morbidities.

## **AIMS AND OBJECTIVES**

- To study maternal risk factors and perinatal outcome in fetal growth restriction.
- To correlate with perinatal morbidity and mortality associated with fetal growth restriction.
- To study outcome of labour in fetal growth restrictions.

## MATERIALS AND METHODS

This study was conducted in Institute of Obstetrics and Gynaecology, Egmore, A Tertiary care teaching hospital for a period of one year. The study population consisted of 100 women with singleton pregnancies who gave birth to neonates with birth weight less than the 10th percentile. {include from methods inclusion criteris, exclusion criteria }

It's a prospective observational study

Carried out in all pregnant women admitted as IUGR between 28-40 weeks of gestation. The Gestational age was calculated from LMP ,if there is discrepancy of more than seven days between LMP and early weeks USG, then USG EDD should be taken.

For above mentioned patients we will do clinical per abdominal examination, calculate gestational age from each trimester scan to categorise either early or late onset FGR.

The above patients are closely monitored throughout the delivery, Close monitoring of all patients with FGR and perinatal outcome after delivery and upto discharge Name, Age, Unit, Registration number and Address of the

patients were noted. Detailed obstetric history including the history of pregnancy induced hypertension; gestational diabetes and chronic hypertension were obtained. History of previous pregnancies including birth weight of previous babies, perinatal deaths, and mode of delivery were elicited. Details of present pregnancy were asked, including the date of last menstrual period, details of scan in the first trimester and clinical examination noting, if available, were scrutinized.

A note was made of the maternal weight, blood pressure and obstetric examination findings of fundal height and various laboratory investigation results. Those with uterine fundal height less than 3cms from the expected height were clinically diagnosed as FGR and ultra sound examination was done with special emphasis on biometric measurements. Abdominal circumference less than 5th percentile and estimated foetal weight less than 10th percentile for that gestational age were selected for study. In cases with risk factors, serial sonography was done to identify fetal growth restriction. Initial dating scan followed by second ultra-sound examination was done at around 34 to 36 weeks.

Patients with irregular cycles, unknown dates, those with restricted growth from the 1st trimester onwards by ultrasound and pelvic examination were included in the study group as were those with history of viral

exanthematous fever, intake of drugs like antiepileptics, antipsychotics & anticoagulants.

An informed consent will be taken. Patients are selected according to inclusion and exclusion criteria

**INCLUSION CRITERIA:**

- All pregnant women with singleton FGR pregnancy between 28 to 40 weeks

**EXCLUSION CRITERIA:**

1. Less than 28 weeks of gestation
2. Women with multiple pregnancy
3. Pregnant women admitted in labour



## **REVIEW OF LITERATURE**

There is considerable debate on the definition of FGR. In accurately dated pregnancies approximately 80 – 85% of fetuses identified as being FGR are constitutionally small. True FGR cases are 10 – 15% and the remaining 5 – 10% of fetuses are having chronic intrauterine infections or chromosomal / structural anomalies.

### **Normal Fetal Growth**

Fetal growth is controlled by complex process confounded by multiple variable such as maternal weight, socio economic factors. It depends on two components genetic potential and substrate supply which is derived from the placenta dependent on the uterine and placental vascularity.

### **Fetal Growth**

14 – 15 weeks of gestation	- 5g / day
20 weeks	- 10g / day
32 – 34 weeks	- 30 – 35g / day

### **SFH**

14 – 32 weeks – 1 cm / week

Abdominal growth increases on an average by 1 inch per week after 30 weeks.

## **Classification of FGR**

Three categories of FGR depends on the time of onset/Pathological process

### **Type I / Symmetrical / Early onset FGR**

Any pathological insult at early phase of embryonic and fetal development from 4 – 20 weeks.

#### **Causes**

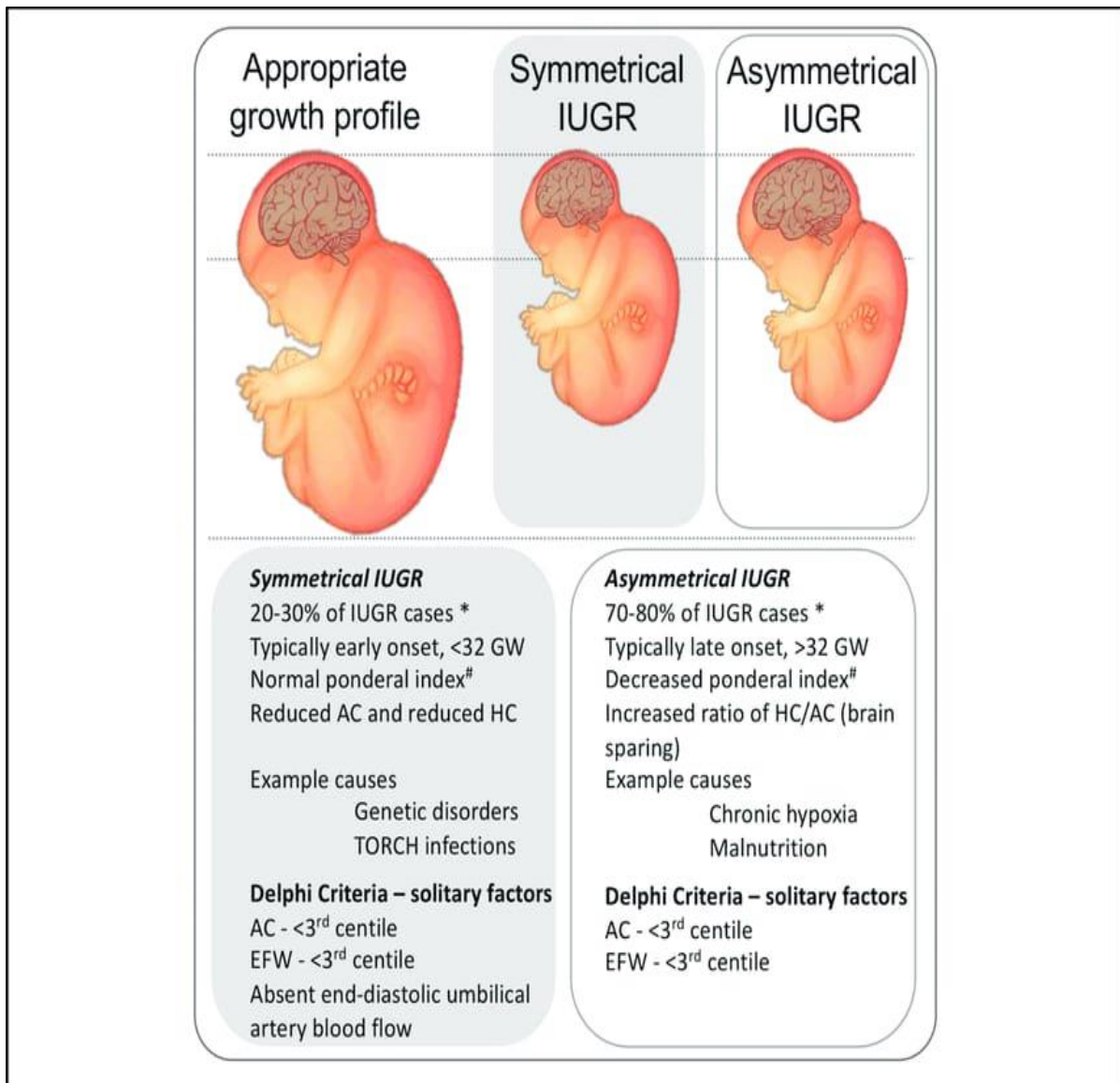
- TORCH
- Chromosomal Disorder
- Congenital Malformation

### **Type 2 / Asymmetric / Late Onset FGR**

Occur as a result of utero placental insufficiency. 70 – 80% of growth restricted fetus

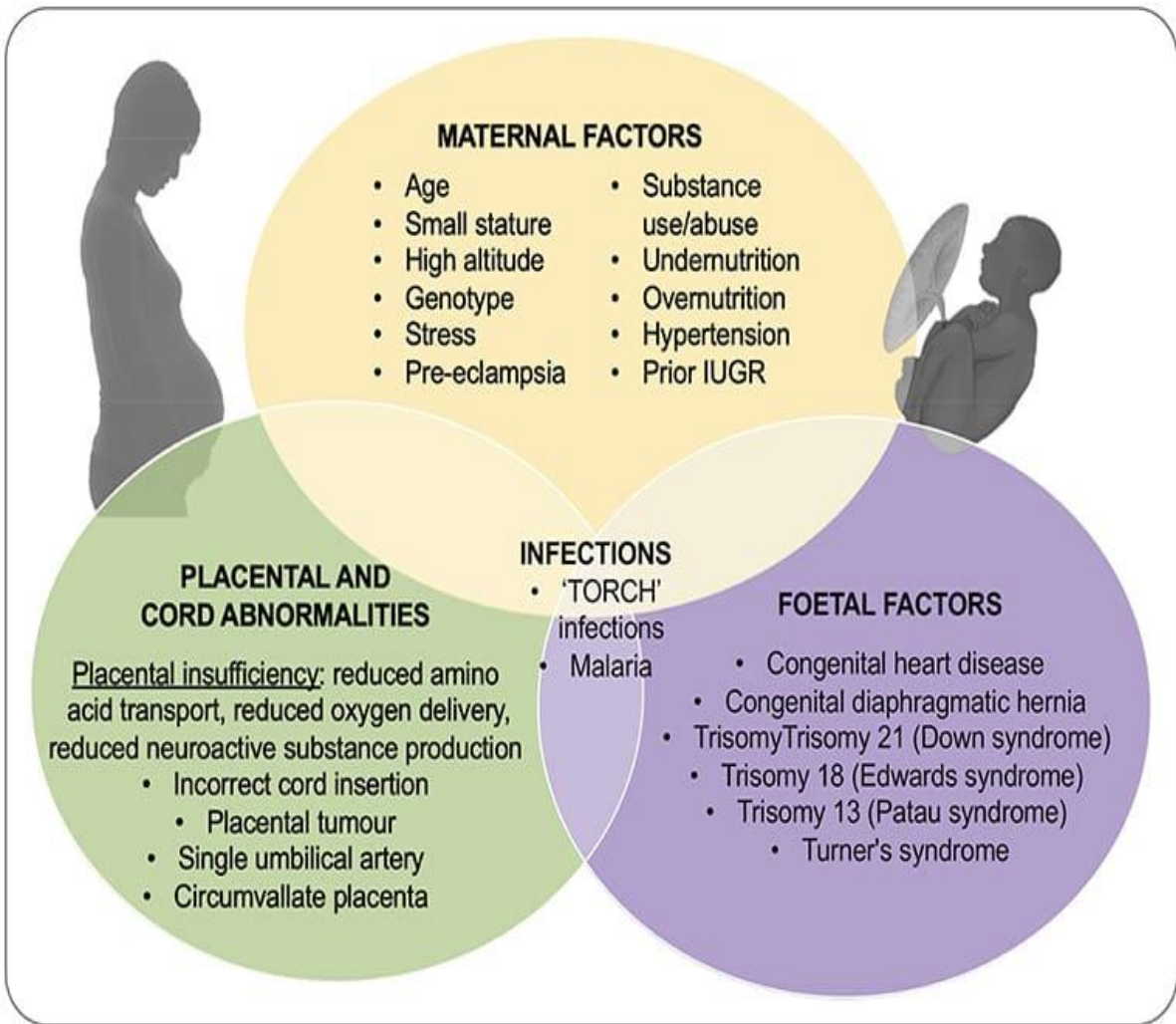
### **Intermediate FGR**

Combination of Type I and Type II FGR



## Aetiology

FGR is not a disease but is a manifestation of fetal, maternal and placental disorders that affect fetal growth. Fetal prognosis is largely dependent on the aetiology. It is therefore, important to ascertain the cause in order to counsel the patient and plan management.



## Fetal Causes

1. Chromosomal disorder – 5% of FGR, Trisomy 13, 18, 21, autosomal deletion
2. Structural Anomalies

All major structural defects involving CNS, Cardio Vascular, gastro intestinal, and musculo skeletal systems are associated with increased risk of fetal growth restriction.

## **Placental Causes**

Fetus is getting nutrition and oxygen supply from placenta single umbilical artery, abnormal placental implantation, velamentous umbilical and insertion, bilobed placenta associated with fetal growth restrictions.

## **Maternal Causes**

1. Extremes of maternal age
2. Nulliparity and grand multiparity

## **Previous history to IUGR**

Prepregnancy low maternal BMI

## **Maternal Diseases**

Medical complications such as

- Hypertension
- Renal disease
- Type I DM – Long standing
- Maternal congenital heart disease
- Maternal respiratory diseases such as cystic fibrosis, Bronchiectasis, Kyphoscolosis and Asthma

Results in uteroplacental insufficiency Thrombophilias

Nutritional Deficiency

### **Consequences of IUGR**

Comparing to normal infants, perinatal mortality and morbidity of IUGR is 3 – 20 times greater among the IUGR features. Increased incidence of still birth and oligohydramnios in antepartum period.

52% of unexplained still births and there is evidence of IUGR. Higher incidence of meconium aspiration, fetal distress and acidosis during labour in growth restricted fetuses. Long term implications on the fetus explanations for the increased risk of hypertension in adulthood and also the tendency to develop Chronic renal failure.

Therefore, importance is emphasized on optimization of timing of delivery, avoid intra partum hypoxia and provide skilled neo natal care at birth.

All these cases were kept under surveillance till confinement. A careful search for causes of IUGR like Smoking, Alcoholism and Hypertension were made. Anemia, if present, was corrected and gestational hypertension, if detected, was managed appropriately. The cases were monitored by Fetal Kick Count, Cardiotocography, Serial measurements of fetometry and Doppler

studies. Doppler studies were done on Umbilical artery, Middle Cerebral Artery and Ductus venosus with a real time color Doppler ultrasound machine. Umbilical cord was located in the pool of amniotic fluid and values were taken at mid cord or placental insertion. Middle cerebral artery was localized in transverse section of fetal skull, at the level of thalamus in the Sylvian fissure. The ductus venosus was sampled in the abdominal circumference section, where it joins the umbilical vein to IVC. The Doppler transducer was placed on the abdominal wall over the uterus and carefully manipulated till Doppler signals appropriate for those particular vessels were identified.

The signals were recorded for a minimum of 5 to 8 cycles with blood flow velocity waveforms of equal shape and amplitude and of satisfactory quality were obtained. The image was frozen and measurements taken. Doppler was considered as abnormal when there was absent or reverse diastolic flow in umbilical artery or PI values were above the 95th percentile for that gestational age. Cerebro placental ratio less than one was also taken as abnormal.

Those cases where fetal assessment was normal were monitored fortnightly till delivery. Those with absent and reverse flow were taken up for termination of pregnancy. In those cases with low diastolic flow in umbilical artery, where fetal maturity adequate for survival was present, the pregnancy

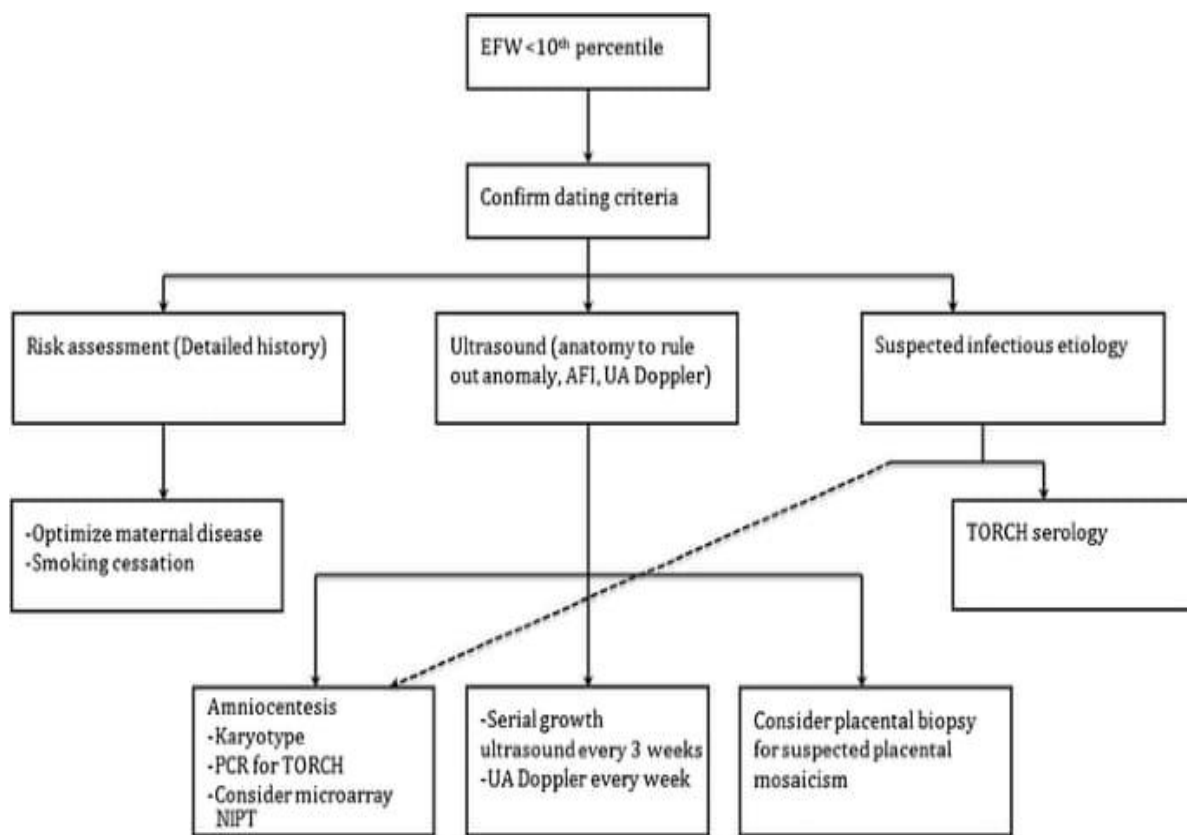
was terminated. In cases where fetal maturity was not reached monitoring was done with NST and BPP daily or twice weekly depending upon the severity of abnormality and associated complications. Pregnancy was terminated when there were abnormal readings from CTG or a low score on the bio-physical profile. In those cases where differential shunting of blood flow to fetal brain was present, termination was done even before NST or BPP were found to be abnormal. Mode of delivery was planned depending on the weight and gestational age and amount of liquor present. Outcome of pregnancy was recorded in detail including intrauterine demise, neonatal death, birth weight, Apgar score, development of neonatal complications and presence of congenital anomalies, placental weight and pathology. These details were entered in a proforma and the data was statistically analyzed and evaluated.

#### SCREENING TESTS FOR FGR

Screening the patients who are at high risk of developing FGR like preclampsia. poor maternal malnutrition, vascular insufficiency. Always confirm the gestational age according to the LMP, commonly wrong dates are told by patients and it is vital to calculate the proper gestational age prior branding as FGR . Menstrual history whether cycles are regular or if she is lactating any recent OC Pills intake 3 months prior to conception have to be elicited.



(i) **Weight Gain Diagnosis** of growth restriction initially by clinical examination  
 Increasing weight gain during pregnancy Normally 10-12 kg  
 weight gain during pregnancy: 0.5 kg/week ( or) 2 kg/month from 2<sup>nd</sup> trimester. Maternal weight gain is less than expected in pregnant women have restricted fetal growth

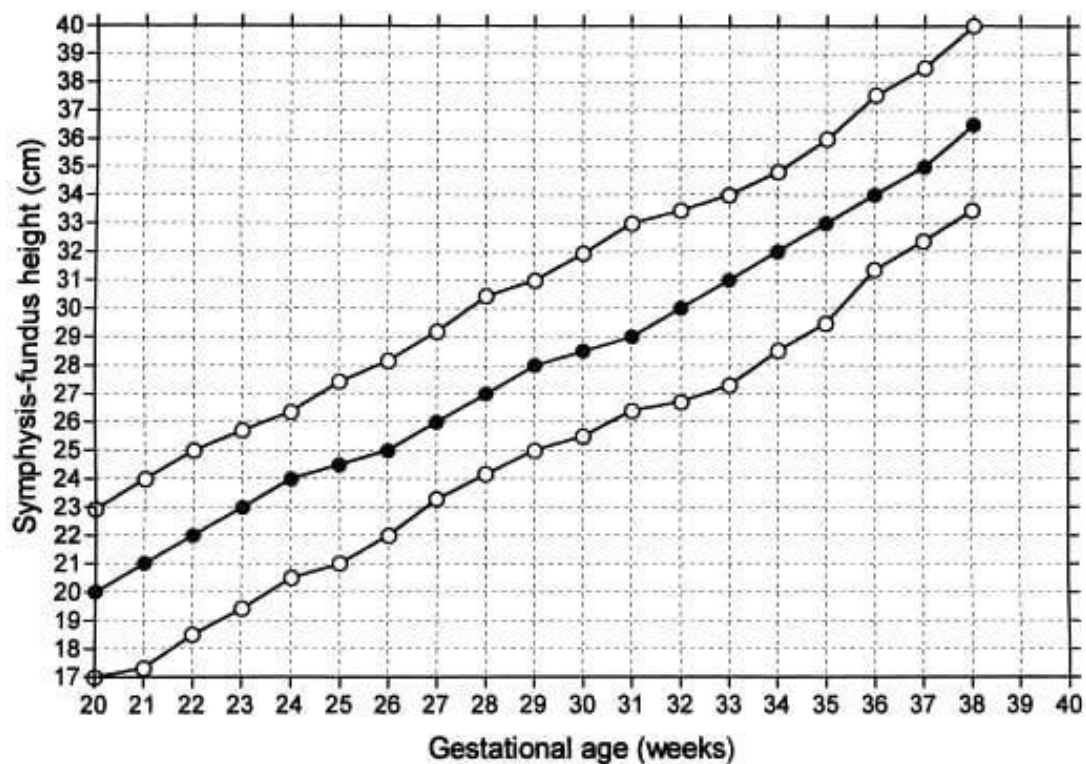


**Gravidogram - Symphysis Fundal Height measurement.**

On clinical examination symphysis fundal height increases by approximately 1 cm per week between 14 — 32 weeks of gestational age. A lag rise in fundal height of 4 weeks is suggestive of moderate FGR. lag of 6

weeks suggestive of severe FGR. Symphysis fundal height is measured from the upper border of symphysis pubis, to the level of uterine fundus. It increases approximately 1 cm/wk. A single symphysisfundal height measurement at 32 — 34 weeks of gestation have 65 — 85% sensitive and 96% specific for detecting fetal growth restriction. if Discrepancy between weeks of gestational age & height of fundus > 3 cm. Clinically reduced liquor. oligohydramnios, by easily palpable fetal parts. Obesity. fibroids. multiple pregnancy limit the accuracy of fundal height and abdomen girth measurement in assessing growth

A screening gravidogram plotting is necessary to easily identify growth lag. the gravidogram plots the symphysis-fundal height against the weeks of gestation.



USG :

In high risk and clinically suspicious pregnancy monitor growth by USG from 28 weeks onwards. Routine growth scan at 32 - 34 weeks can pick up many clinically unsuspected cases. In biometry most useful single parameter is Abdomen circumference.

1st Trimester - CRL accuracy  $\pm$  5 days, in 2<sup>nd</sup> Trimester upto 24 weeks  $\pm$  10 days accurate Gestational age can be calculated.

In USG we do fetal biometry and calculate estimated fetal weight, amniotic fluid volume, abdominal circumference and Doppler changes. Clinical diagnosis is unreliable ..

Tulli and colleagues analysed (2011) 151 second trimester USG is superior to first trimester for predicting FGR neonates . "**INTERGROWTH-21**" one recent ongoing project in 8 countries to define regional standards based on data from optimal maternal health and socioeconomic conditions of the population in 8 countries include: INDIA, CHINA, KENYA, OMAN, ITALY, BRAZIL, UNITED KINGDOM, UNITED STATES (Villar, 2014)

### **A. Biparietal Diameter:**

In normal fetus, BPD increases

Upto 28 weeks : 3 - 3.5 mm/wk

28 - 32 weeks . 2.5 mm/wk

> 32 weeks : 1.7 mm/wk

In FOR fetus. BPD increases

Between 13 — 34 weeks: < 2mm/wk 35 — 40 weeks < 1 mm/wk

### **B. Abdominal Circumference:**

Abdominal circumference have sensitivity of 96 — 100% for detecting fetal growth restriction. In normal growth fetus. Abdominal girth increases on an average by 1 inch / wk after 30 weeks. It is about 30 inches at 30 weeks in an average built women.



AC is the most useful single parameter. Sovio and colleagues (2015) recently demonstrated that growth velocity of the AC is the lowest decile distinguishes SGA babies who suffer increased morbidity. Increased hepatic blood flow is correlated with abdomen circumference and fetal growth velocity (ACOG 2016 C)7.

### **C. HC/AC**

HC/AC has the sensitivity of about 70%, HC/AC >1 after 36 weeks suggestive of FGR In the normal growing fetus Before 32 weeks gestation FIC/AC ratio > 1.0

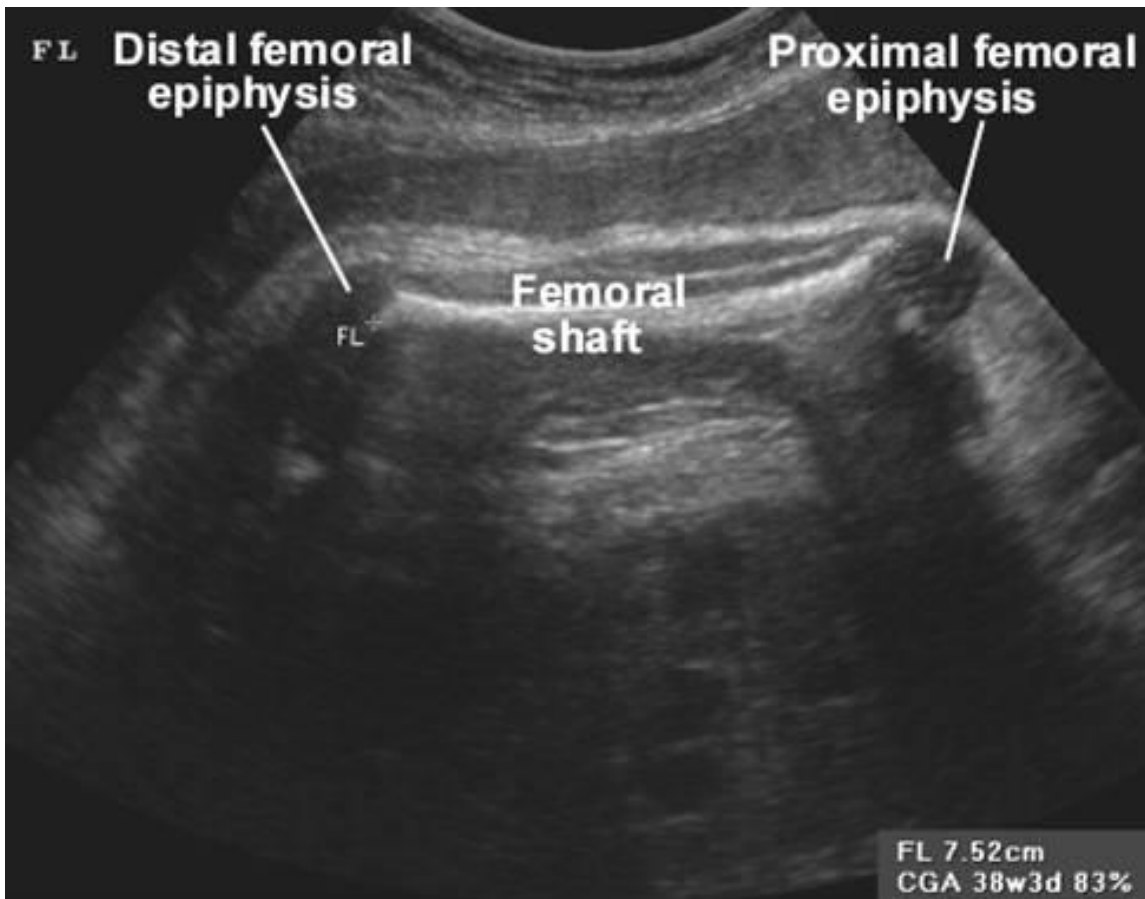
At 32 — 34 weeks gestational HC/AC ratio = 1.0 After 34 weeks gestational HC/AC ratio < 1.0

In asymmetric growth restriction, the head circumference remains larger than that of body and results in an elevated HC/AC Ratio.

### **D. FL/AC Ratio**

FL / AC ratio have the sensitivity of about 63%. Femur length is relatively spared in asymmetrical FOR. It is gestational age independent >23.5 suggestive of FGR.

In normal fetus FL/AC value  $\leq 22$  after 21 weeks of GA to term



### **E. PONDERAL INDEX**

Ponderal index is the way of determining the relationship of height to mass for an individual, it is a useful tool to detect FGR.

Normal Value  $-8.325 \pm 2.5$

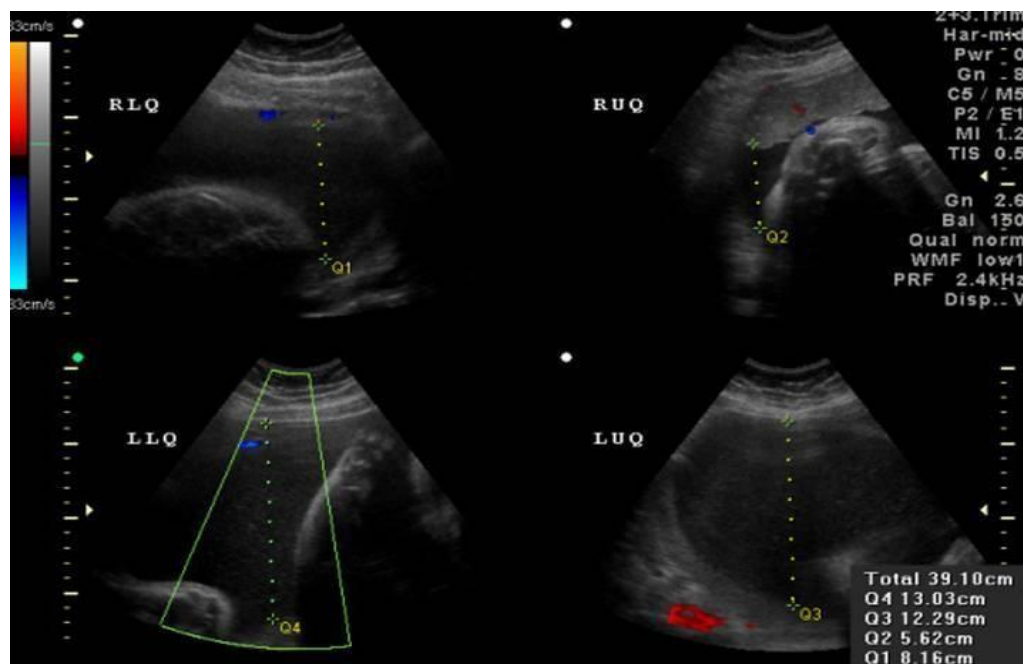
In FOR fetuses Ponderal Index is less than 7 In SGA Fetuses have normal ponderal index

## F. Fetal Transverse Cerebellar Diameter

Transverse Cerebellar Diameter is distance between outer borders of cerebellum. TCD is equal to Gestational Age in weeks till 32 weeks.

## G. Amniotic Fluid Index

Amniotic Fluid Index has been recognized as an important component of fetal wellbeing evaluation. It is the indicator of placental perfusion and related to fetal urine output. AFI is calculated by Summating vertical depth of amniotic fluid volume in all four quadrants. Hypoxia and diminished renal blood flow are the cause for oligohydraminos ( $AFI \leq 8$  cm) in FGR. Decreased amniotic fluid volume between 24-32 weeks gestation of pregnancy was significantly associated with malformation (Petrozelle et. al. 2011)



## **H.. Estimated Fetal Weight**

Ultrasound estimates of fetal weight and actual birth weight may be discordant by 20% or more. Estimated fetal weight have the sensitivity of about 87% to detect fetal growth restriction.

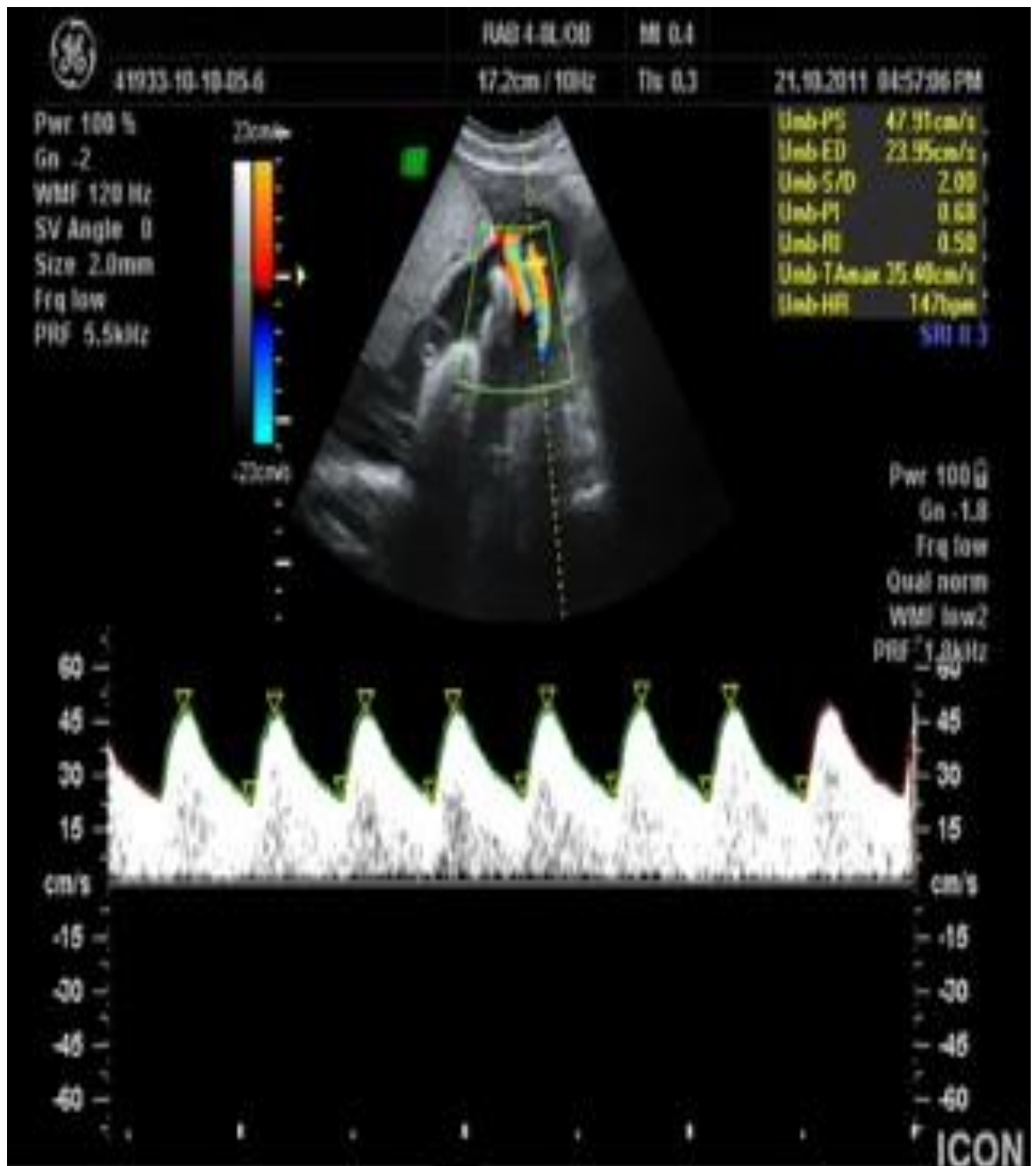
**Duryea (2014)(8)** plot, fetal growth curves birth weight against a gestational age, based on an obstetric estimate more accurately termed population reference rather than a standard reference. A population references include pregnancies of varying risk, and their outcomes both normal and abnormal. standard reference include only normal pregnancies with normal Outcomes.

Uterine Artery Doppler is abnormal in early onset FGR but it is normal in 1<sup>st</sup> trimester scan in late onset FGR.

## **B. Umbilical Artery**

Umbilical artery Doppler can assess the resistance to blood perfusion in the Fete placental unit.





There is low impedance continuous forward flow throughout cardiac cycle from 14 weeks onwards. Any conditions that obliterate small muscular arteries in the tertiary stem villi of placenta lead to progressive decrease in end diastolic flow. This progressive Decrease EDF lead to Absent EDF and then Reverse End Diastolic Flow.

Placental vascular resistance progressively increased leads to AEDF. REDF.[Pulsatility Index, Absent End Diastolic flow, Reverse End Diastolic Flow].

If resistance : 30% - lead to decrease in EDF. If resistance : 50% - lead to absent EDF. If resistance : 70% - lead to reversed EDF.

AEDF & REDF associated with severe IUGR. (EFW <3rd percentile) and oligohydramnios.

**Unterseheideri(9)** and associated reported that abnormal (UA) Umbilical Artery Doppler wave form velocimetry with an estimated fetal (EFW) weight <3rd percentile is strongly associated with poor obstetrical outcome.

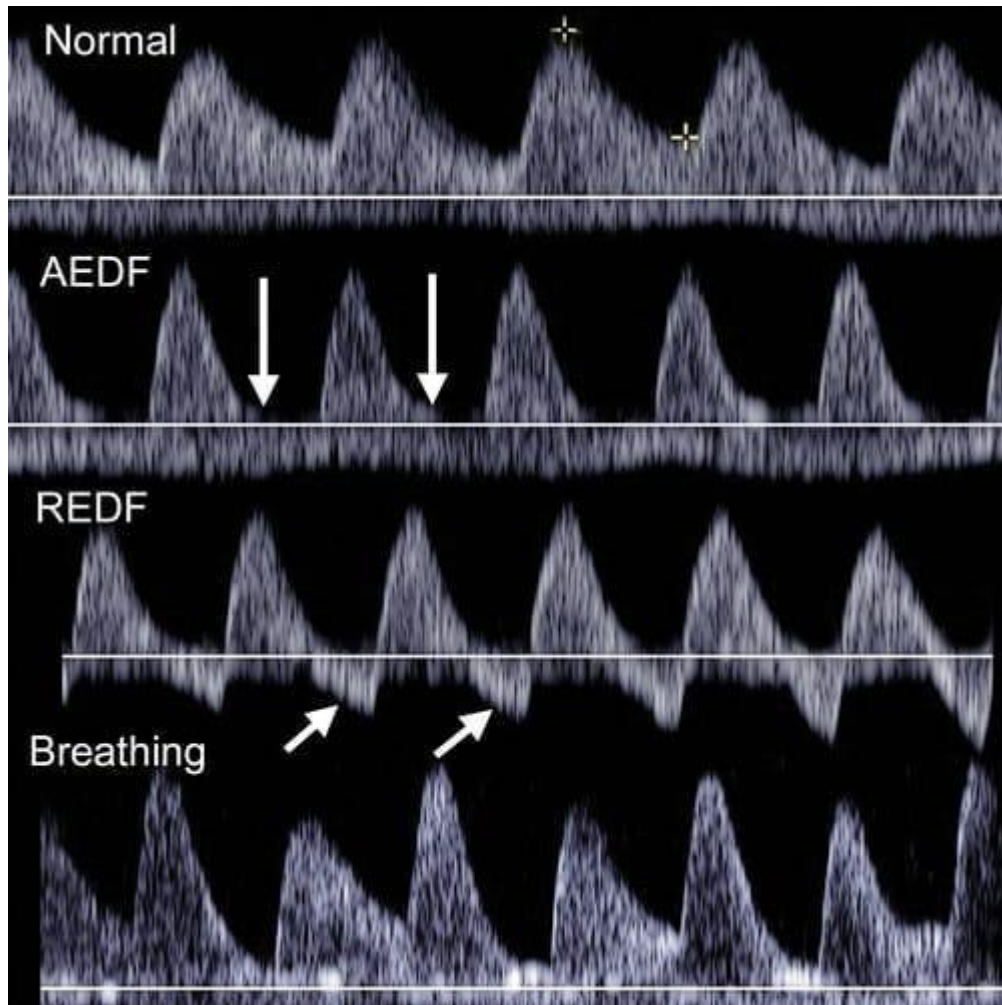
### C.Middle Cerebral Artery

Cerebral circulation is a high impedance. continuous flow Cerebral MCA contribute throughout cardiac cycle in normal pregnancy. 80% of cerebral circulation. It is a major branch of Circle of Willis.

MCA Doppler is to be done in the transverse plane of the fetal head in their longitudinal view. angle  $30^{\circ}$ . Proximal portion of MCA near the Circle of Willis have shown better reproducibility.

MCA peak systolic velocity may be a better predictor of perinatal mortality in preterm IUGR. During increased placental vascular resistance, fetal hypoxemia occurs that leads to central redistribution of blood flow to brain, heart and adrenal glands with redistribution of blood flow to the peripheries and renal flow. This redistribution of blood flow known as "brain sparing reflex" characterised by increased EDF (low PI) in the MCA. ROZA et. al., and associates (2018) found brain sparing effect due to redistribution of fetal circulation had a higher incidence of behavioural problems later.

Fetal brain sparing when this ratio is <5th percentile For gestational age.



## II. BIOPHYSICAL PROFILE

- Declining amniotic fluid volume related to renal blood flow and the degree of vascular redistribution,
- Decline in global fetal movements occurs with worsening of fetal hypoxemia.
- Further deepening of hypoxemia leads to cessation of fetal breathing movement.
- BPP score of  $\leq 4$  is associated with a fetal pH  $\leq 7.20$ . While a score of  $< 2$  has a sensitivity of 100% acidemia
- Loss of tone and movement is characteristic as the pH drops further.

### NON STRESS TEST

Non Stress Test is "NON REASSURING" when a mean pH between 7.10 and 7.20.

Spontaneous "decelerations" especially "late decelerations" associated with placental insufficiency. Late Deceleration start at the peak of uterine contraction and attain maximum decrease in heart rate at the end of uterine contraction. If its not corrected at this stage leads to fetal demise later.

Antenatal risk includes previous history of SGA or stillbirth. causes such as tobacco, alcohol and other drugs. fetal infections (CMV and Rubella are the most associated onset and maternal diseases (mainly renal and vascular). Other risk factors are preeclampsia related, such as thrombophilic conditions. obesity, and chronic hypertension. Although these risk factors are multiple and not always well defined. it remains a key step to select a population of high-risk on which a close follow-up may be warranted. Nevertheless. only use of this high-risk group will develop IUGR.

### **Fundal Height Measurement**

Both the fundal height measurement and the abdominal palpation have sensitivities of about 30% to detect SGA and. therefore. could not be recommended. Nevertheless. it has been reported that customized standards for fundal height. which adjust for parity. maternal height and weight. ethnicity and fetal gender. and a longitudinal evaluation allow sensitivities of about 30%.<sup>20</sup> comparable to the detection rate of routine third trimester fetal biometry in low-risk pregnancies. In settings where a policy of third trimester ultrasound is not in place. fundal height measurements remain common practice.

All these cases were kept under surveillance till confinement. A careful search for causes of IUGR like Smoking, Alcoholism and Hypertension were made. Anemia, if present, was corrected and PI1-1, if detected, was managed appropriately. The cases were monitored by Fetal Kick Count, Cardiotocography, Serial measurements of fetometry AR and Doppler studies. Doppler studies were done on Umbilical artery, Middle Cerebral Artery and Ductus venosus with a real time color Doppler ultra sound machine. Umbilical cord was located in the pool of amniotic fluid and values were taken at mid cord or placental insertion. Middle cerebral artery was localized in transverse section of fetal skull, at the level of thalamus in the Sylvian fissure. The ductus venosus was sampled in the abdominal circumference section, where it joins the umbilical vein to IVC. The Doppler transducer was placed on the abdominal wall over the uterus and carefully manipulated till Doppler signals appropriate for those particular vessels were identified.

The signals were recorded for a minimum of 5 to 8 cycles with blood flow velocity waveforms of equal shape and amplitude and of satisfactory quality were obtained. The image was frozen and measurements taken. Doppler was considered as abnormal when there was absent or reverse diastolic flow in umbilical artery or PI values were above the 95th percentile for that gestational age. Cerebro placental ratio less than one was also taken as abnormal.

Those cases where fetal assessment was normal were monitored fortnightly till delivery. Those with absent and reverse flow were taken up for termination of pregnancy. In those cases with low diastolic flow in umbilical artery, where fetal

maturity adequate for survival was present, the pregnancy was terminated. In cases where fetal maturity was not reached monitoring was done with NST and BPP daily or twice weekly depending upon the severity of abnormality and associated complications. Pregnancy was terminated when there were abnormal readings from CTG or a low score on the bio-physical profile. In those cases where differential shunting of blood flow to fetal brain was present, termination was done even before NST or BPP were found to be abnormal. Mode of delivery was planned depending on the weight and gestational age and amount of liquor present. Outcome of pregnancy was recorded in detail including intrauterine demise, neonatal death, birth weight, Apgar score, development of neonatal complications and presence of congenital anomalies, placental weight and pathology. These details were entered in a proforma and the data was statistically analyzed and evaluated.

### Pregnancy Dating

Pregnancy dating based upon the last menstrual period provides inaccurate estimates of the gestational age. since up to a 20% of women with regular cycles ovulate later than expected. Hence. in clinical settings where a policy of first or early second trimester scan is in place. it seems to be more appropriate to systematically use the fetal biometrics to date the pregnancy and ensure a reliable fetal age assessment for most purposes, for example Down's syndrome screening. There are several formulae to date the pregnancy from early biometry, with low systematic and random errors. Crown-rump length (CRL) is a biometric parameter that can be measured in the early stages of



gestation Fig. 3). Technically the main limitation is the progressive bending of the embryo which makes measurements less reliable beyond 12-13 weeks of gestation (or 60-70 mm). Normal reference ranges to date the pregnancy are published elsewhere. If possible, below the 14 weeks, all obstetric ultrasound units are currently recommended to adopt this method of assessing gestational age from crown rump length. From then on, it seems conceptually more appropriate to use cephalic (head circumference) and/or femur (femur length) biometrics. Series in which different formulas have been tested in pregnancies conceived with artificial reproductive techniques provide comprehensive recommendations on this matter. Once the pregnancy has been dated by an early scan, further adjustments must not be performed.

## BIOMETRIC EVALUATION

Initially, and still in many places, the biparietal diameter was the only measurement that was routinely used for the assessment of fetal growth,. When pregnancy is normal, this parameter falls within the normal range, can be consider a representative Indicator of the other fetal organ growth, and tissues. but when pregnancy is abnormal , still fall within the normal range (head an is rarely affected in many cases.. On the other hand, misdiagnoses have been on many occasions in fetuses with marked brachycephalism or dolicocephalism in association with normal development. In addition, measurement of the BPD does not permit determination of fetal weight with acceptable reliability. The

substitution of BPD by head circumference or cephalic area does not substantially improve the Sensitivity of the method. With the purpose of improving the screening method measurement of the length of the femur is introduced. It has the advantage that it measures a component of fetal longitudinal growth and does not suffer the sudden flattening out characteristic of cephalic parameters as term, although it has the disadvantage of not being a useful parameter for establishing the diagnosis of FGR early stages. Abdominal circumference (AC) is the most accurate single biometry to predict SGA at birth. In high-risk women, AC at less than the tenth centile has sensitivities of 72.9-94.3% and specificities of 50.6 in prediction of fetuses with birthweight less than the tenth centile. The use of Cross-sectional reference charts for each biometry with the closest distribution to that of the screened population remains the gold standard and some studies are alert to the impact of choice of reference charts in the assessment of fetal biometry. In that sense they order to choose the most recommended is Z-score. Many charts require three repeat measurements in order to control random error. By increasing the number of measurements to four, the 95% error span is reduced to half. fetal biometrics could be used for estimated fetal weight (FEW) as it Predicts the occurrence of SGA birth with sensitivities of 33.3-89.2% and specificities of 53.7-90.91%. A prospective study<sup>o</sup> comparing several formulas concluded that Shepard formula have the best interclass correlation coefficient, with smallest mean difference from actual birthweight.

for fetuses weighing less than 2000 gm. this formula has not been validated. The Hadlock formula will be more appropriate when the fetus is expected to be very small. Controversy exists regarding using AC or EFW for the antenatal assessment. Whereas AC, the antenatal assessment of fetal growth is the simplest method and in high-pregnancies has higher sensitivities, EFW has a stronger association with birthweight below the 10th centile. We prefer using EFW since is more consistent with the neonatal assessment, which is mainly performed by weight. In addition, the accuracy of the individual fetal parameters cannot be checked as there is no gold standard. On the contrary, estimated fetal weight could be assessed against birthweight and has a random error of about 8%.

Since growth is a dynamic process, it seems logical that its quantification requires the evaluation of serial measurements. In fact, serial measurements of AC and EFW are superior to single estimates in the prediction of neonatal growth restriction defined by ponderal index or skinfold thickness and in the prediction of adverse outcome.

**STATISTICAL ANALYSIS:** All the data was entered into microsoft excel software and analysed using SPSS16 for windows. descriptive analysis was done. Chi-square/fischer exact has been used to find significance of study parameters and P value of <0.05 was considered significant

## OBSERVATIONS

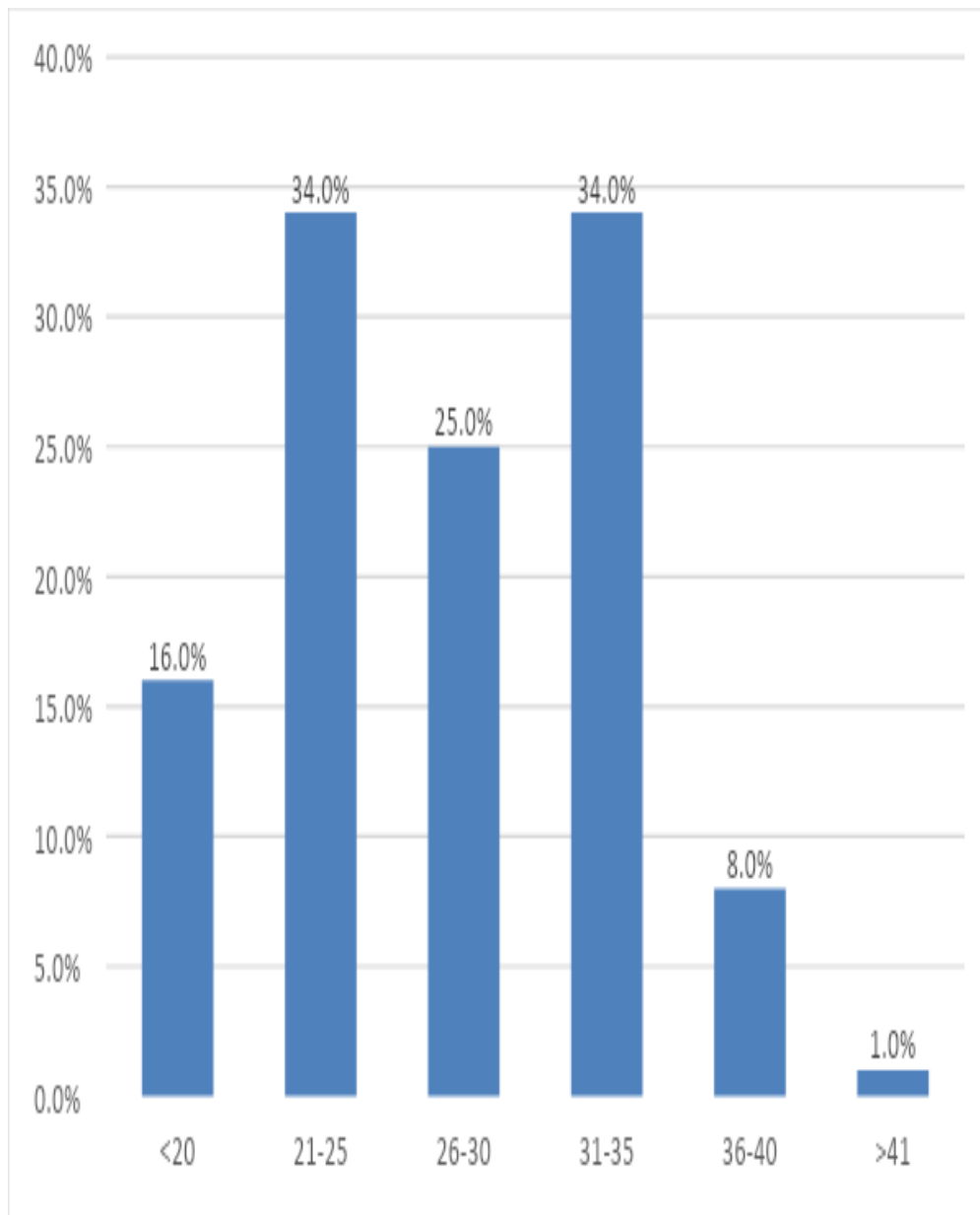
### SOCIO-DEMOGRAPHIC PROFILE AND FGR:

Majority of mothers were in 31-35 years(34%)age group with 60% among primigravidae.46% of women belongs to upper middle class socio economic status .

Only 5% of mother had previous history of FGR.All Cases are booked and immunised.

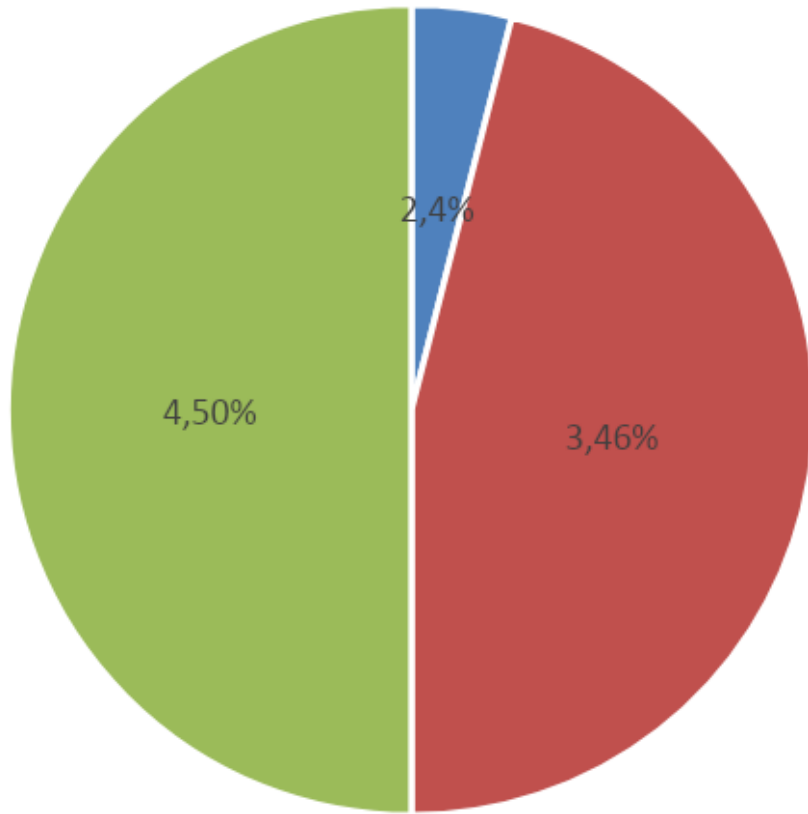
### AGE GROUP

Age group	Frequency	Percent
<20	16	16.0%
21-25	34	34.0%
26-30	25	25.0%
31-35	34	34.0%
36-40	8	8.0%
>41	1	1.0%
Total	100	100.0%



## SOCIO ECONOMIC CLASS

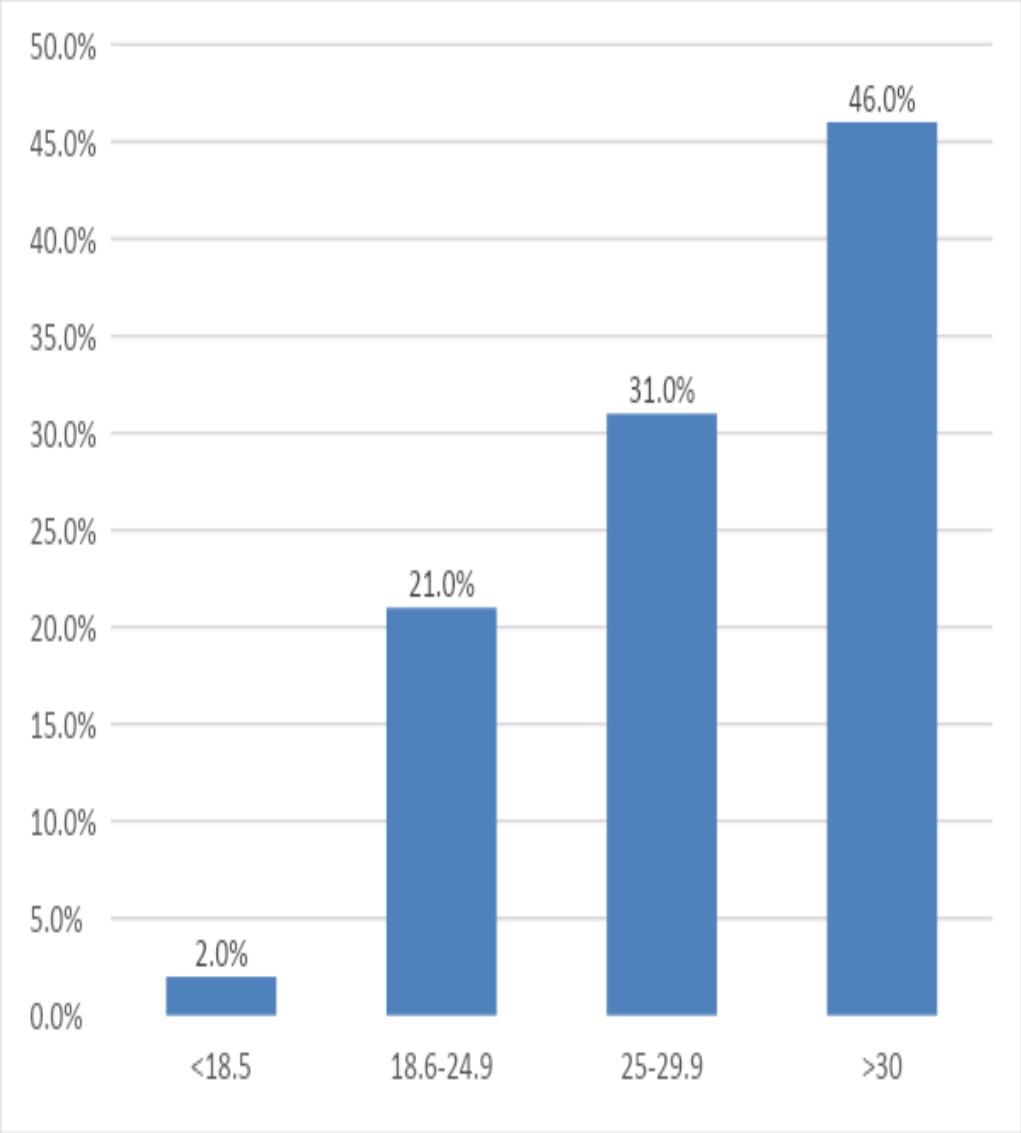
SE class	Frequency	Percent
2	4	4.0%
3	46	46.0%
4	50	50.0%
Total	100	100.0%



## BMI

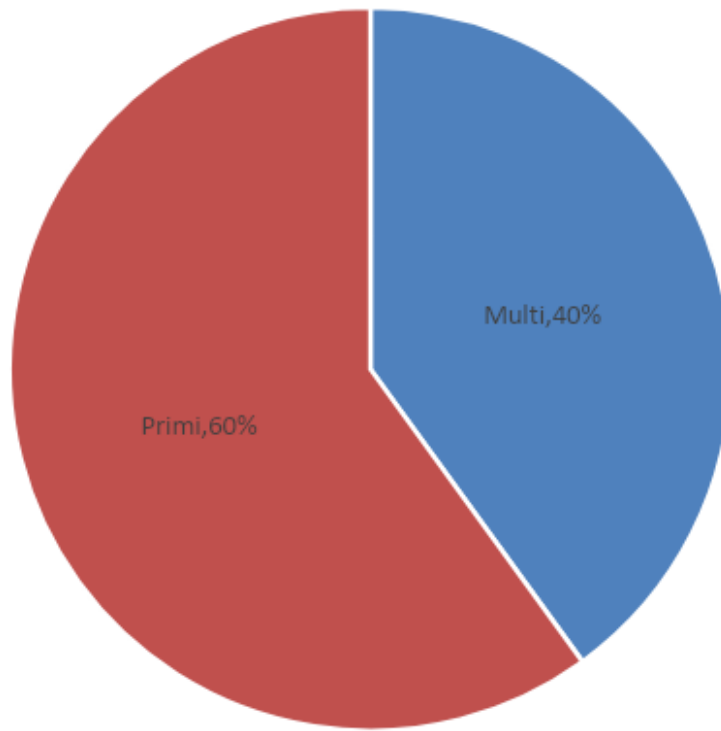
BMI	Frequency	Percent
<18.5	2	2.0%
18.6-24.9	21	21.0%
25-29.9	31	31.0%
>30	46	46.0%
Total	100	100.0%





## PARITY

Parity	Frequency	Percent
Multi	40	40.0%
Primi	60	60.0%
Total	100	100.0%



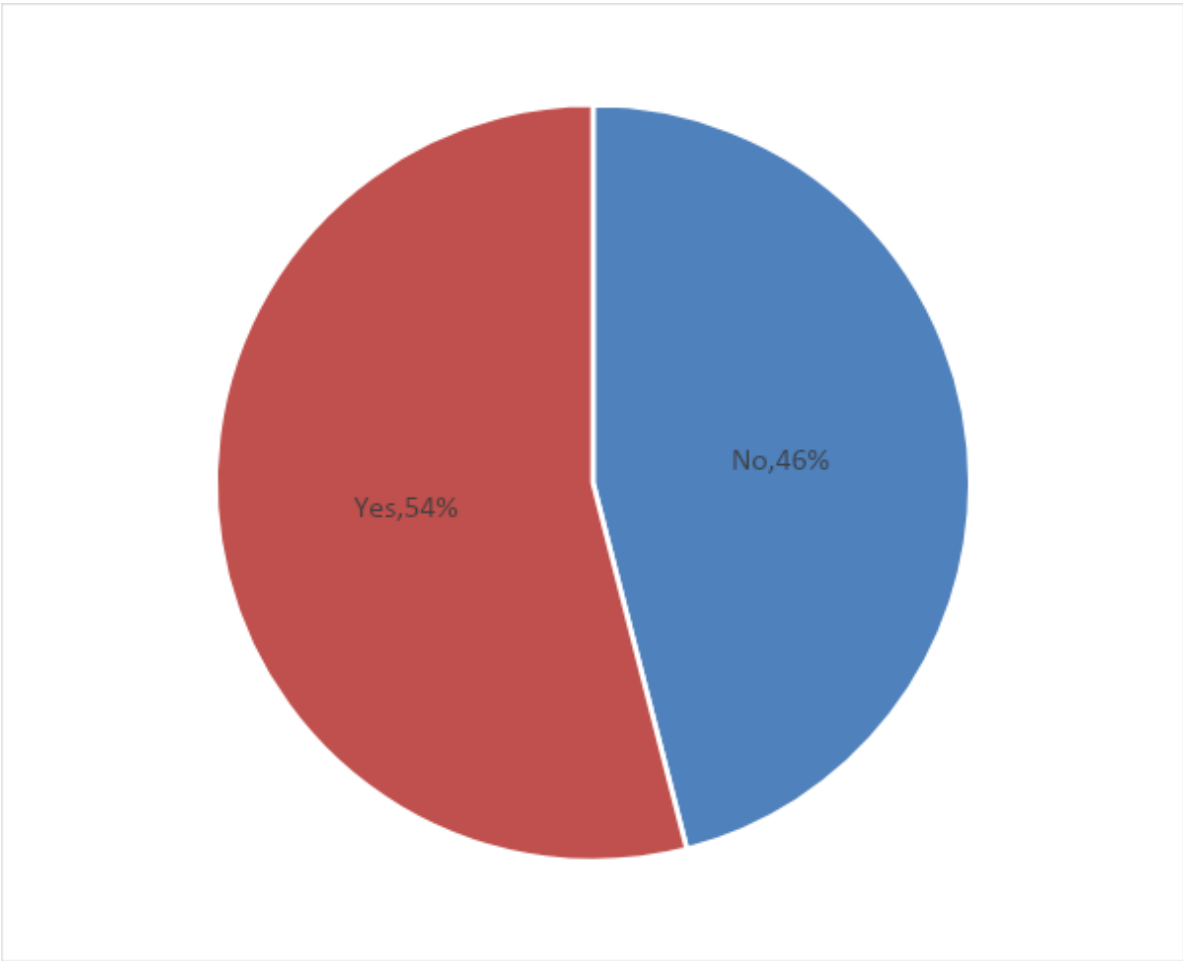
## MATERNAL RISK FACTORS AN FGR:

Coming to maternal morbidities leading to FGR,64% were hypertensive disorders of pregnancy presented as severe preeclampsia followed closely by Idiopathic causes(25%).Eclampsia/imminent eclampsia and HELLP required magnesium sulphate and antenatal corticosteroids.three cases of APLA presented with FGR.

Eleven cases of anemia presented with FGR requires blood transfusion.hypothyroidism accounts for 7percentage of cases.Gestational diabetes mellitus patients presented with FGR when patient is under high dose of insulin and among overt diabetes patients comparing to GDM on meal plan patients.

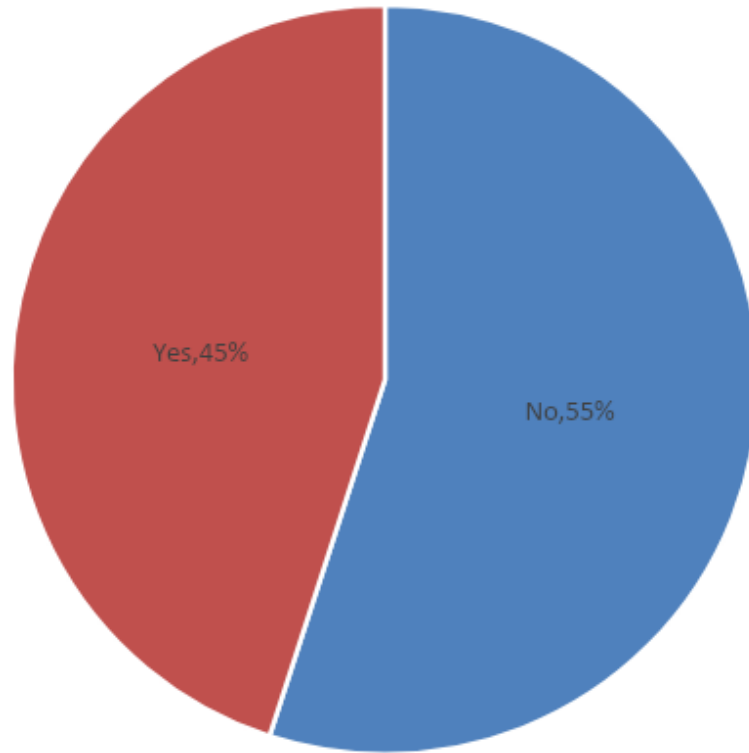
## SEVERE PREECLAMPSIA CHART

severe preeclampsia	Frequency	Percent
No	36	36.0%
Yes	54	54.0%
Total	100	100.0%



## IDIOPATHIC

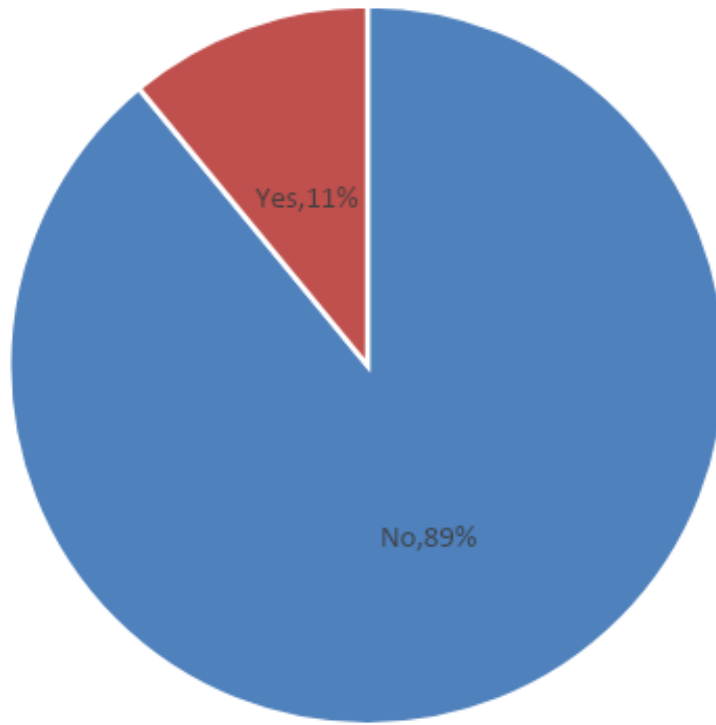
Idiopathic	Frequency	Percent
No	55	55.0%
Yes	45	45.0%
Total	100	100.0%



## ANEMIA

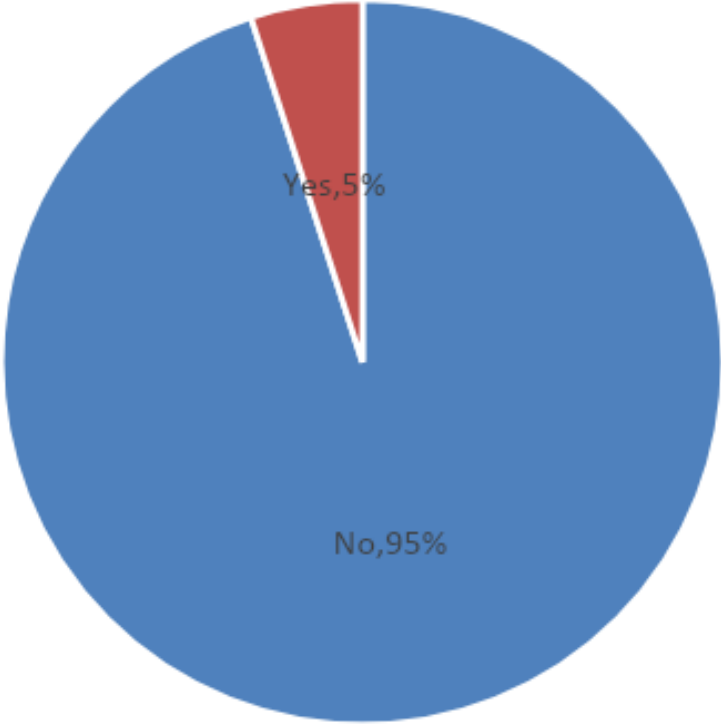
ANEMIA	Frequency	Percent
No	89	89.0%
Yes	11	11.0%
Total	100	100.0%





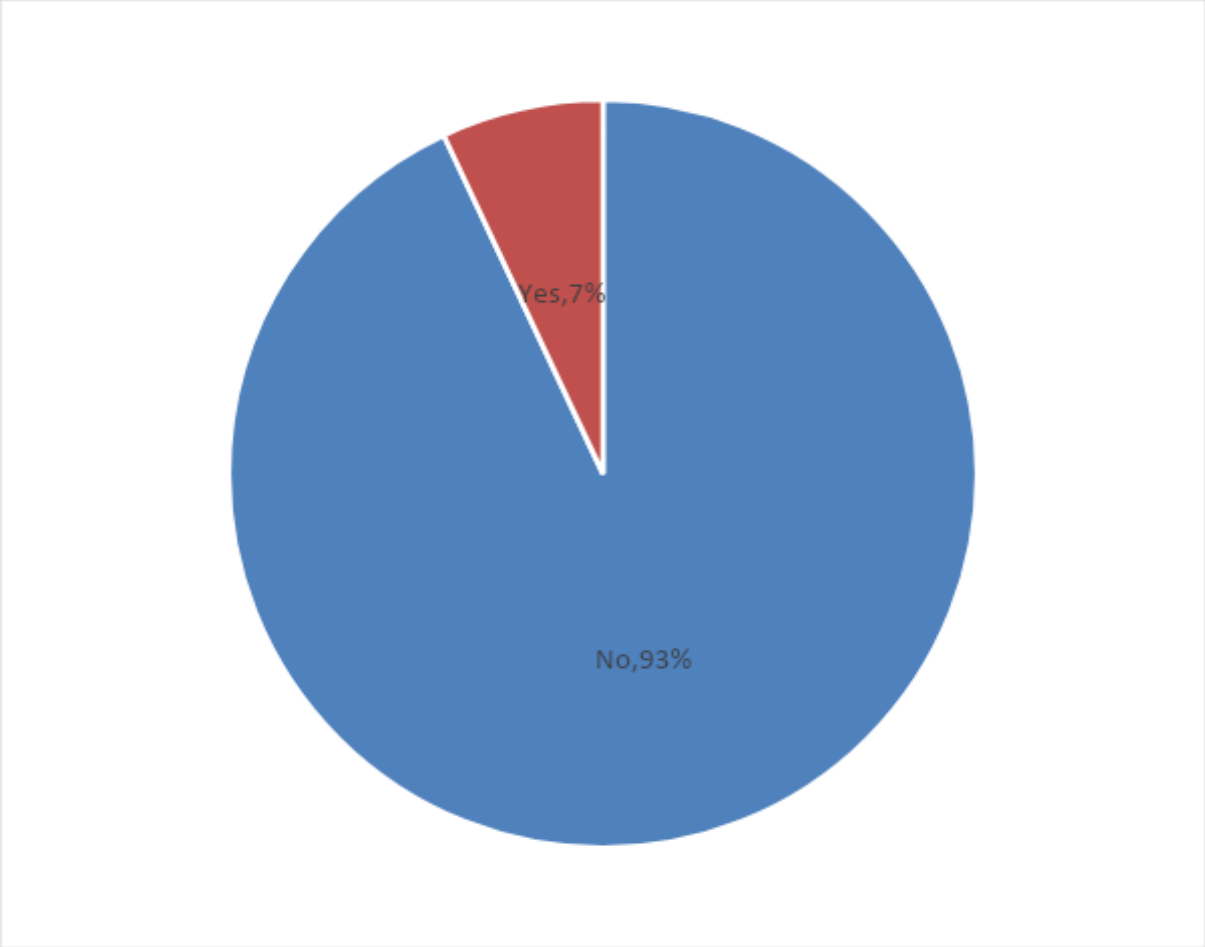
## PREVIOUS HISTORY OF FGR

Prev h/o IUGR	Frequency	Percent
No	95	95.0%
Yes	5	5.0%
Total	100	100.0%



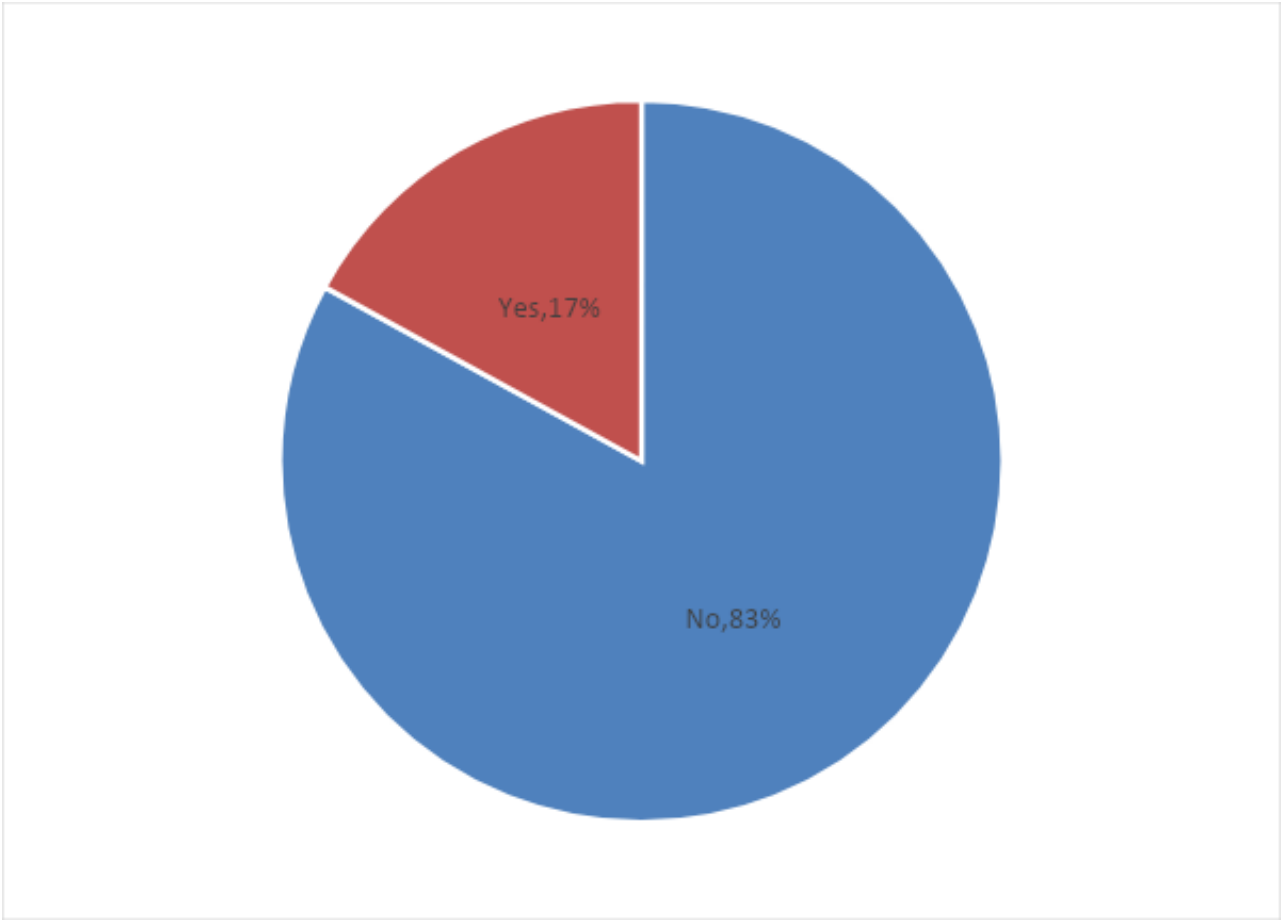
## Hypothyroid

Hypothyroid	Frequency	Percent
No	93	93.0%
Yes	7	7.0%
Total	100	100.0%



## GESTATIONAL DIABETES MELLITUS

DM	Frequency	Percent
No	83	83.0%
Yes	17	17.0%
Total	100	100.0%



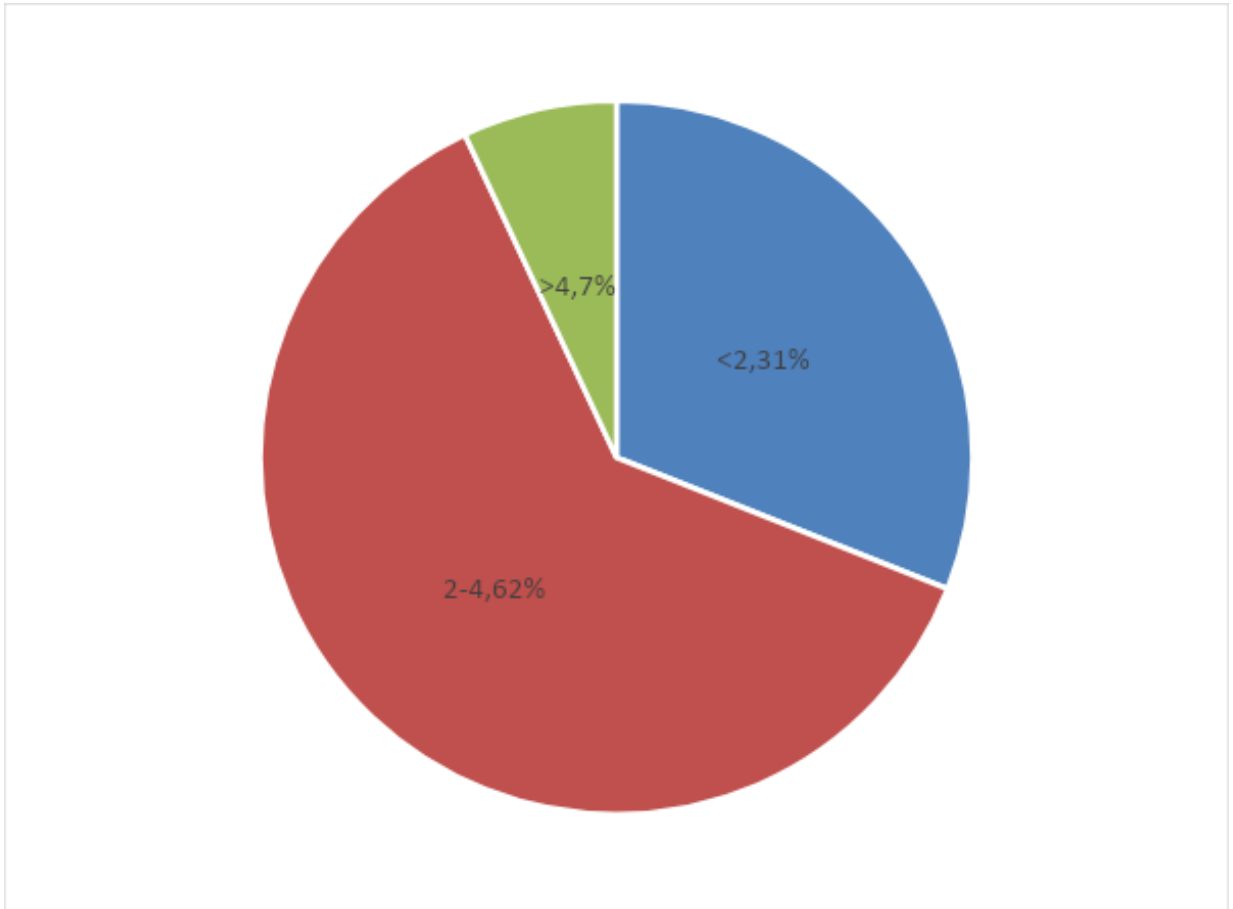
## **DIAGNOSIS-DELIVERY DELAY AND FGR**

FGR were diagnosed in 44% during 30-35 weeks of gestation followed by 32% as early preterm.23% FGR diagnose > 37 weeks group.13 cases delivered as emergency without induction due to reversal and absent end diastolic flow.39% cases delivered in 3-7 days. Hence diagnosis-delivery interval of < 48 hrs was noted as magnesium sulphate for neuroprotection as well as for prevention pre eclampsia and antenatal corticosteroids for fetal lung maturity delays the delivery which favours good perinatal outcome.

Decision was delayed in view of normal doppler parameters and good in utero surveillance.

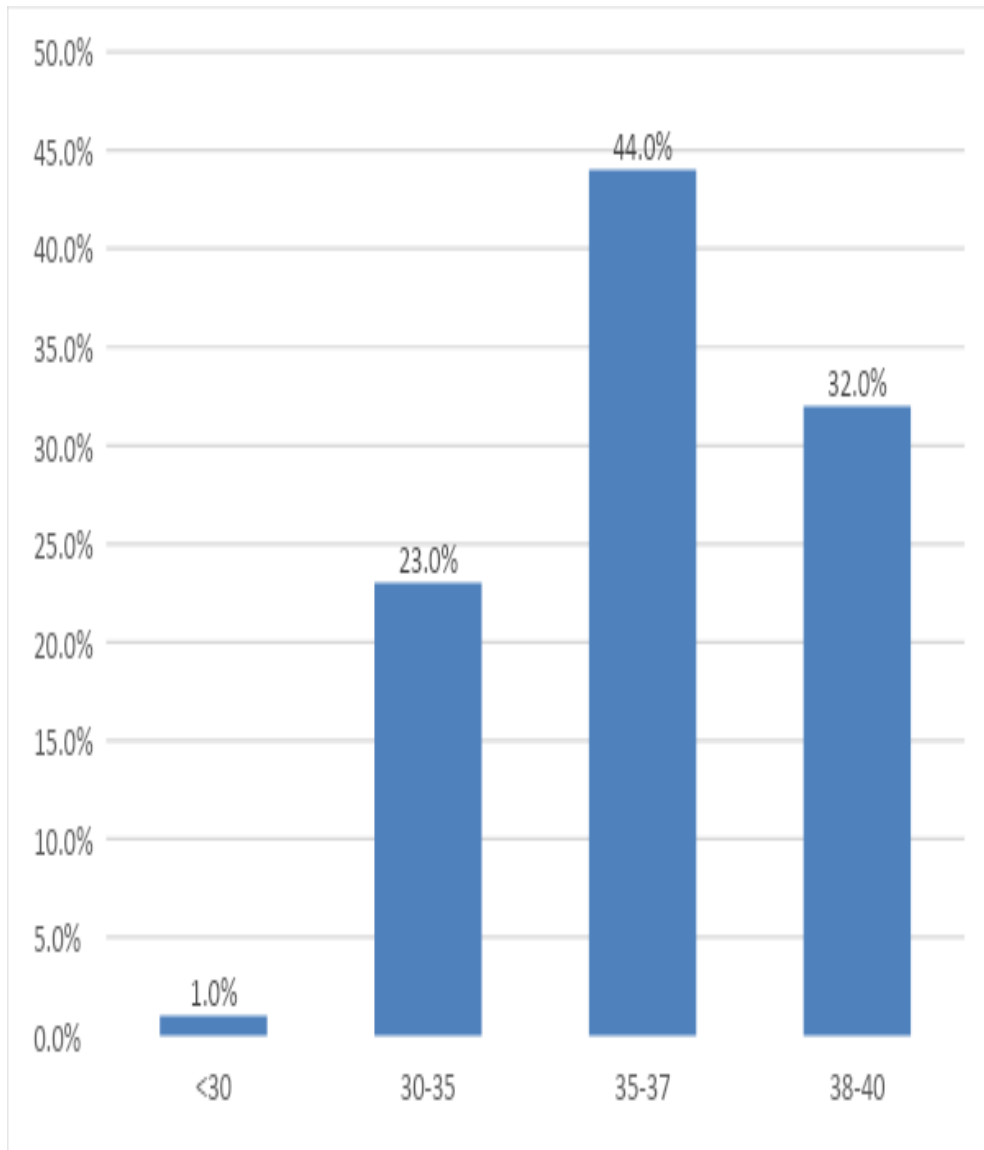


SFH	Frequency	Percent
<2	31	31.0%
2-4	62	62.0%
>4	7	7.0%
Total	100	100.0%



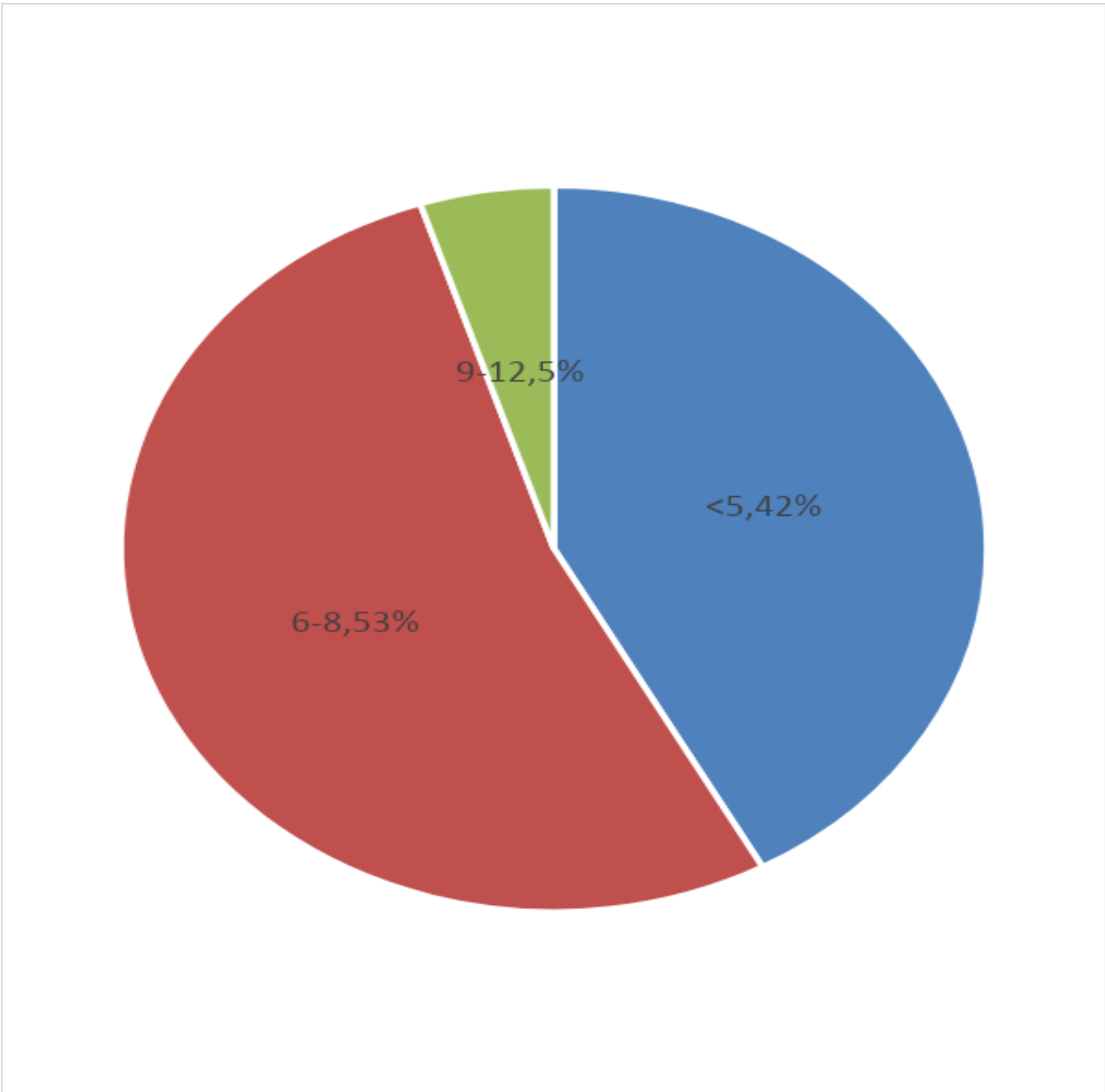
POG

POG at delivery	Frequency	Percent
<30	1	1.0%
30-35	23	23.0%
35-37	44	44.0%
38-40	32	32.0%
Total	100	100.0%



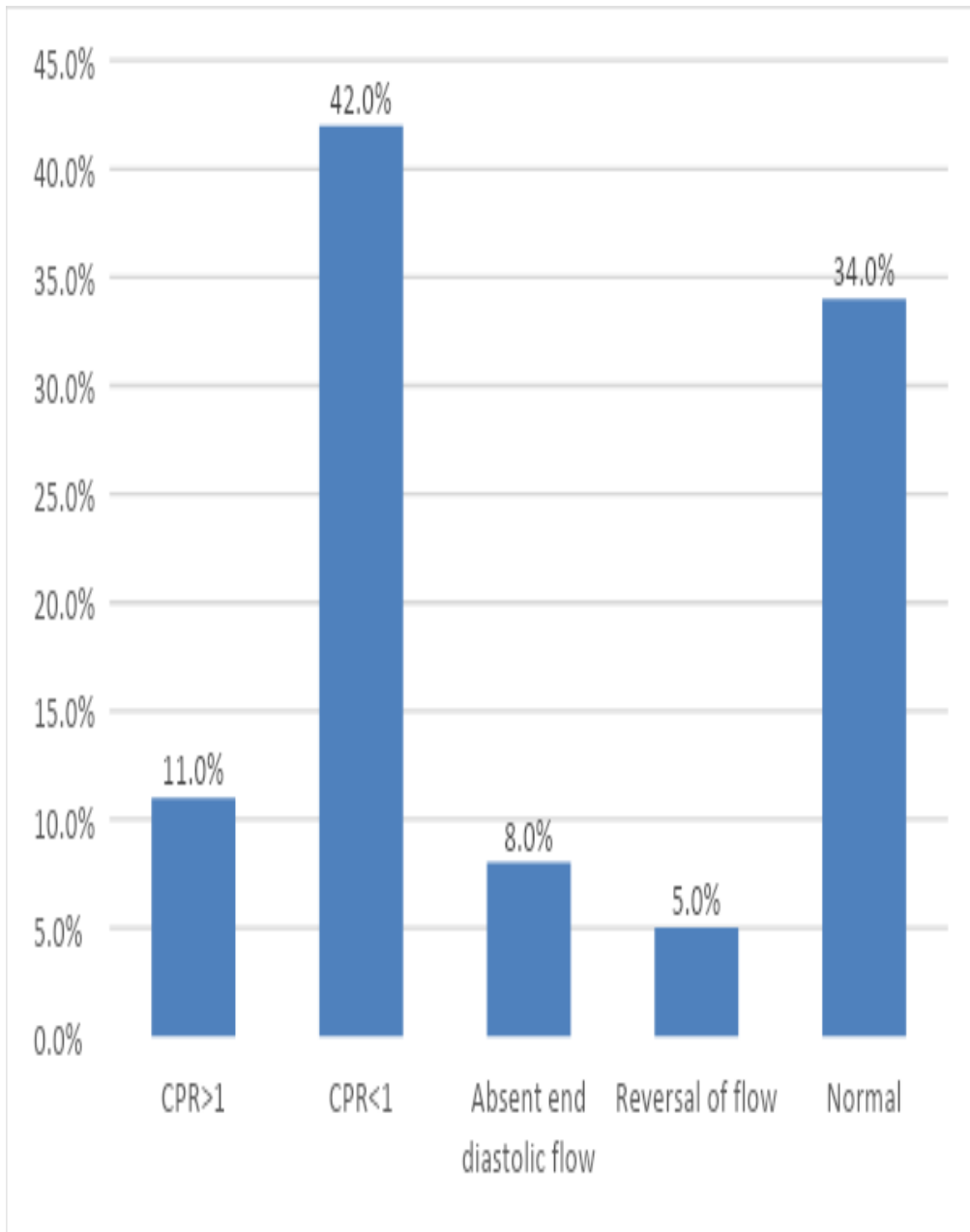
AFI

AFI	Frequency	Percent
<5	42	42.0%
6-8	53	53.0%
9-12	5	5.0%
Total	100	100.0%



## DOPPLER

DOPPLER	Frequency	Percent
CPR>1	11	11.0%
CPR<1	42	42.0%
Absent end diastolic flow	8	8.0%
Reversal of flow	5	5.0%
Normal	34	34.0%
Total	100	100.0%



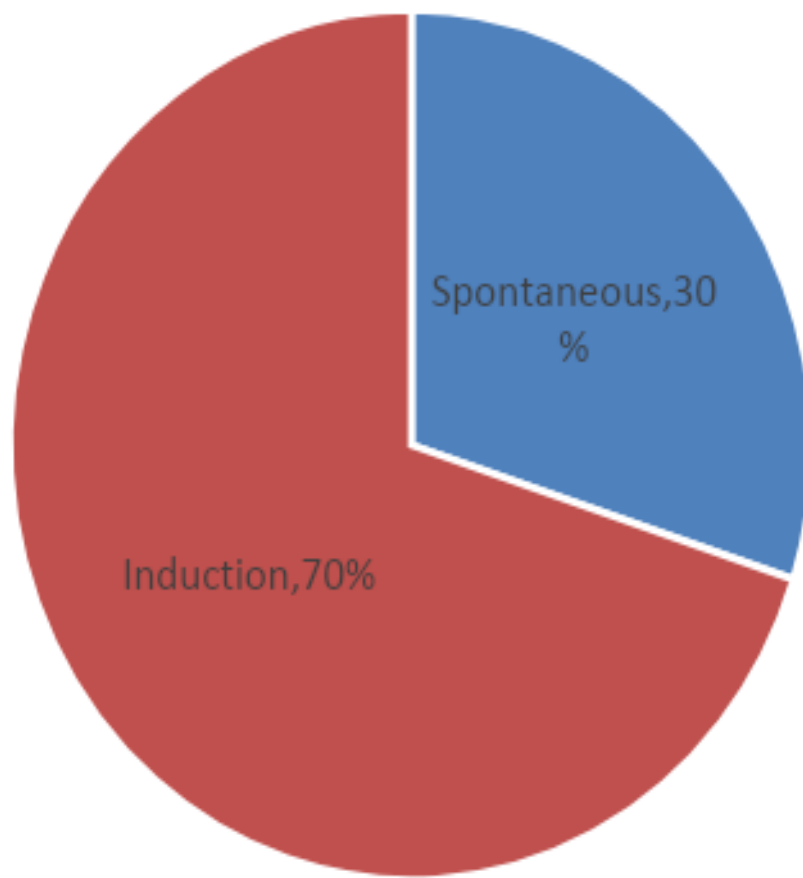


## MODE OF OUTCOMES AND FGR:

Fetal causes as a reason to deliver was noted in 59% cases due to fetal distress, non reassuring fetal heart rate, severe oligohydramnios, doppler abnormality  $CPR < 1$ , meconium stained liquor. 28% cases were terminated for maternal reasons with severe preeclampsia, imminent eclampsia, failed induction and unfavourable cervix and previous cesarean delivery added to burden of operative deliveries. 80% of cases undergo induction except previous LSCS cases and cases with AEDF and Reversal.

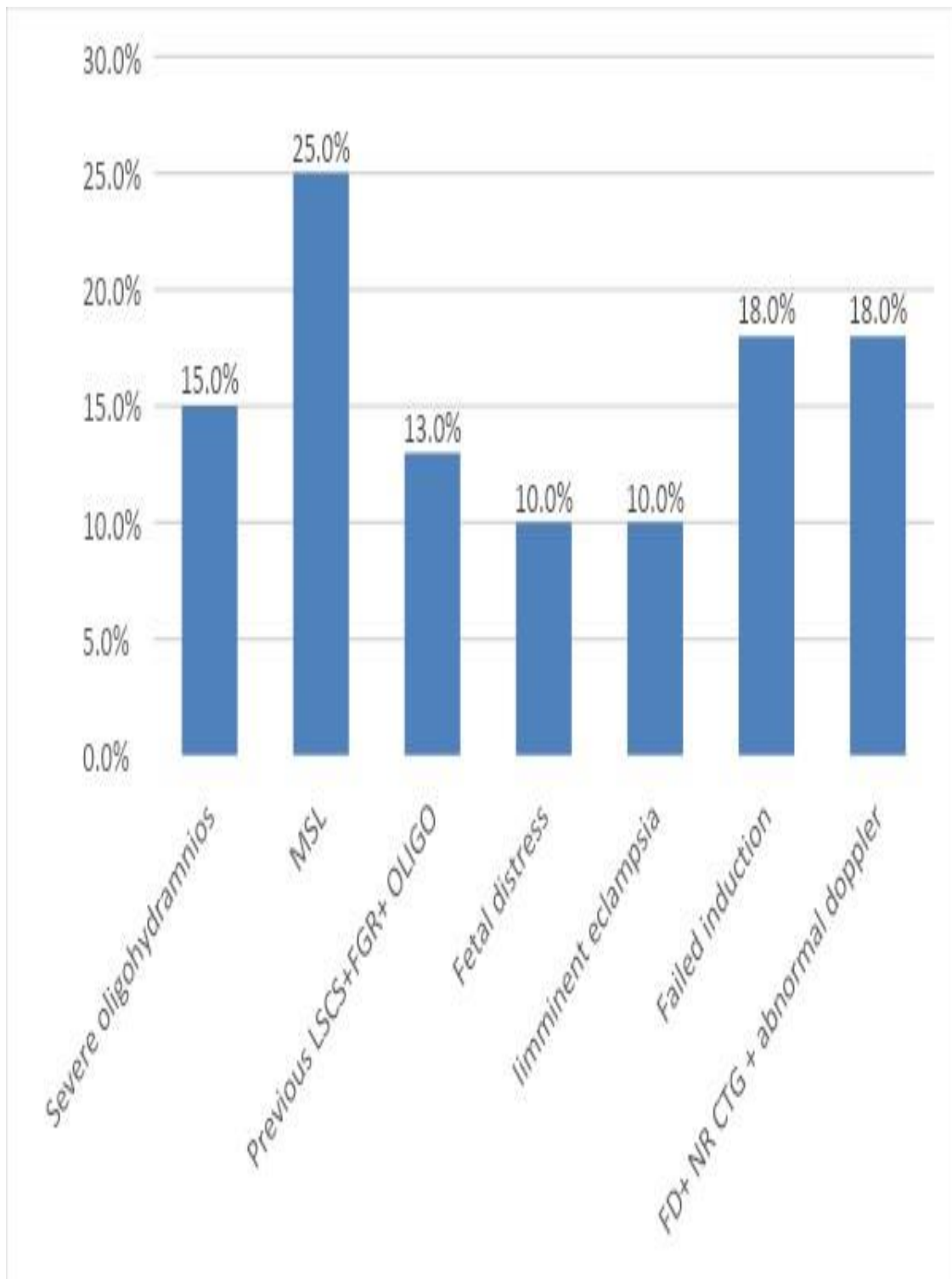
Cesarean delivery accounted for the majority of the cases (63%). Indications are given below

Mode of Termination	Frequency	Percent
Spontaneous	30	30.0%
Induction	70	70.0%
Total	9	9.0%



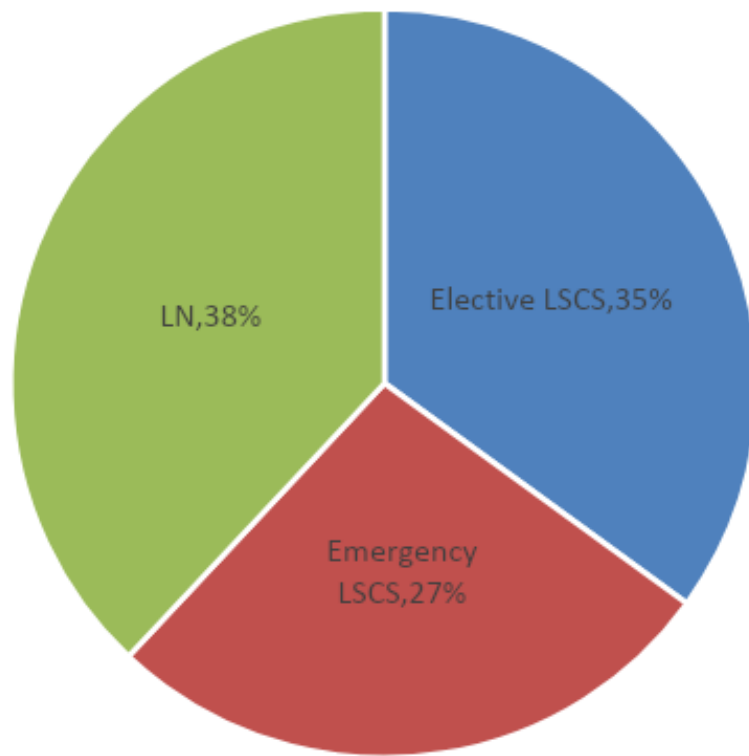
## INDUCTION OF TERMINATION

Indication of Termination	Frequency	Percent
Severe oligohydramnios	15	15.0%
MSL	25	25.0%
Previous LSCS+FGR+ OLIGO	13	13.0%
Fetal distress	10	10.0%
Iimminent eclampsia	10	10.0%
Failed induction	18	18.0%
FD+ NR CTG + abnormal doppler	18	18.0%
Long period of infertility+oligo	3	3.00%



## MODE OF DELIVERY

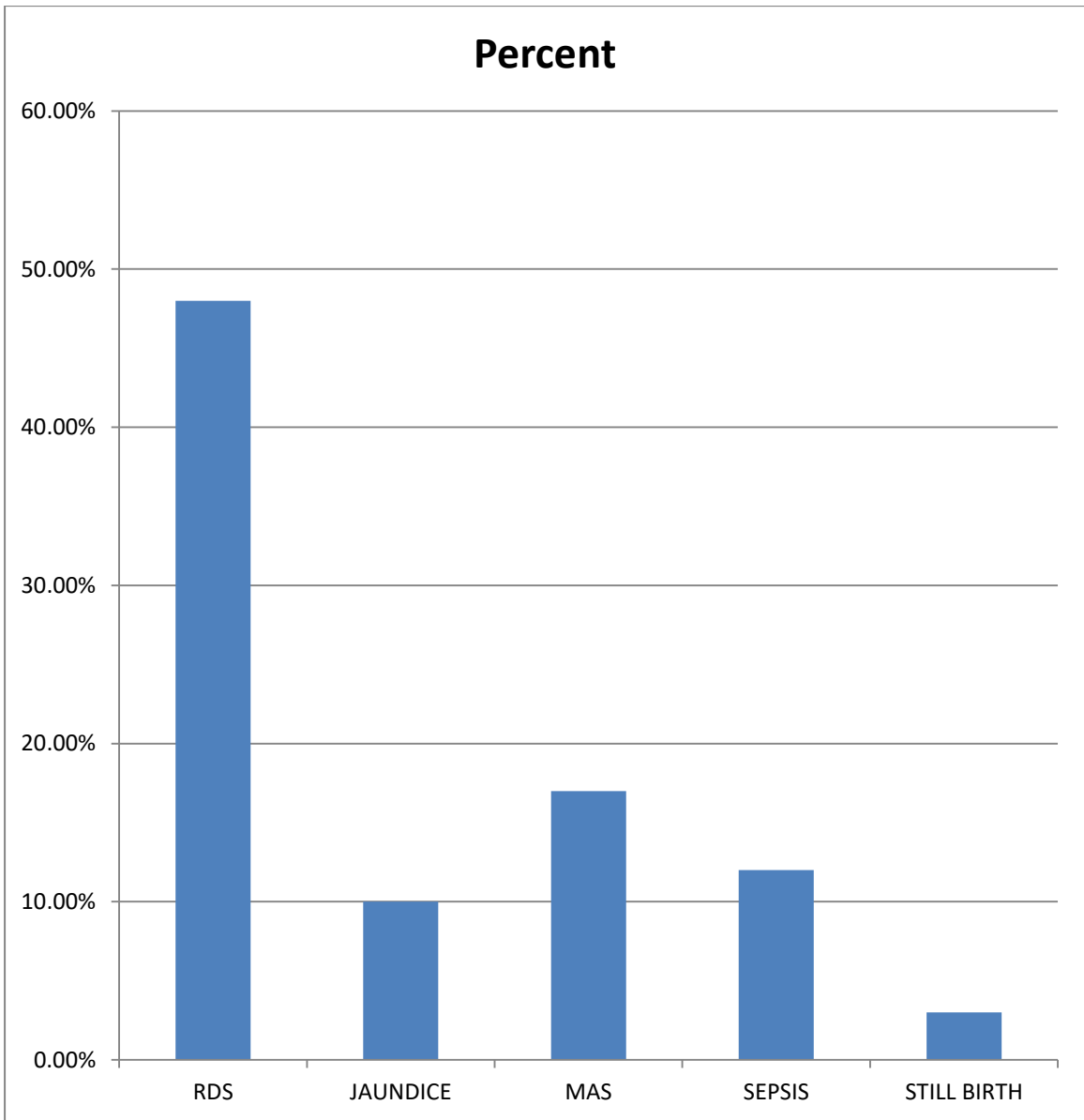
Mode of delivery	Frequency	Percent
Elective LSCS	35	35.0%
Emergency LSCS	27	27.0%
LN	38	38.0%
Total	100	100.0%



## NEONATAL MORBIDITY AND FGR:

NICU admissions were needed in 71 neonates(86%).need of resuscitation in 13% with need of ventilator and CPAP.neonatal morbidity in terms of NICU stay.TERM FGR had shorted NICU stay and EARLY PRETERM (59%) had longer NICU stay due to neonatal morbidities necessitating prolonged intensive care. sepsis, hyperbilirubinemia, respiratory syndrome, prematurity were neonatal morbidities in growth restricted fetuses.

NEONATAL OUTCOME	Frequency	Percent
RDS	48	48.0%
JAUNDICE	10	10.0%
MAS	17	17.0%
SEPSIS	12	12.0%
STILL BIRTH	3	3.0%





## DISCUSSION

Incidence of FGR was 9.8% in present study. The incidence of FGR according to national perinatal database was 9.65% among neonates.

Majority of mothers were in 26-35 years age group (59%) similar to Satyavrathan and Singh A et al.

The rate of FGR in developing countries is six times higher comparing to developed countries. Hypertensive disorders of pregnancy (64%), idiopathic causes (24%), hypothyroid, diabetes, APLA were noted in FGR mothers similar to Sharma et al and Satyavarthan et al

Cesarean mode of delivery remains the primary one. 24% of cases delivered vaginally comparing to 32% as in Seal et al. Imminent eclampsia, HELLP syndrome and eclampsia accounted for IMMEDIATE TERMINATION. The most common indication of termination was fetal

distress(29%),severe oligohydramnios(40%),doppler changes which is observed in Rajarajeswari et al study.

FGR diagnosed between 32-34 weeks majority as with Seal et al.in Lekshmi et al ,60% of the FGR born to the mothers <37 weeks and 29% <32 weeks,while in ours 44% in 35-37 weeks and 32% were born in 30-35 weeks

Birth weight constituted 2-2.5 kg.so FGR with no doppler abnormalities had significantly better birth weights than comparing to abnormal doppler. Gestational age and NICU stay were compared and it was found that neonates born at term had shorter NICU stays comparing to preterm also. FGR babies with normal doppler also has shorter nicu stay. NICU admission in our hospital(77%) similar to Rekha BR et al 77.8%.and Ebrashy et al 66%.thomas et al study shows that FGR with abnormal doppler had early delivery, increased NICU admission, neonatal hyperbilirubinemia(10%),respiratory distress(48%),sepsis (6%).three still births occurred .the APGAR scores >7 for majority of the babies. the inverse relationship between BPP and presence of fetal distress, FGR, NICU admissions were described by manning et al. FGR fetus with non reactive CTG and there is 3.5 times the chance of abnormalities in doppler patternin contrast to the study of Grivell RM reported that NST had no significance

## CONCLUSION

High risk factors for FGR should be evaluated in all the pregnancies. Accurate diagnosis can be obtained through monitoring using serial growth charts ,DFMC,FHR monitoring by using CTG and the doppler studies of uterine,umbilical,MCA ,CPR. Cesarean section remains the primary mode of delivery .

Correction of maternal risk factors and timely delivery optimise the fetal outcome. The diagnosis of uteroplacental insufficiency causing FGR identifies the group of fetus prone for perinatal complications. Abnormal doppler associated significantly with earlier FGR detection, shorter decision delivery interval, longer NICU stay.

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## ABBREVIATIONS

IUGR	:	Intrauterine growth restriction
SGA	:	Small for gestational age
CTG	:	Cardio Tocography
NST	:	Non stress test
Umb. Ar	:	Umbilical Artery
MCA	:	Middle cerebral artery
DV	:	Ductus venosus
USG	:	Ultrasonogram
PI	:	Pulsatility index
RI	:	Resistance index
SFH	:	Symphysio - fundal height
NICU	:	Neonatal Intensive Care Unit
MAS	:	Meconium Aspiration Syndrome
NEC	:	Necrotising Enterocolitis
AEDF	:	Absent end Diastolic flow
REDF	:	Reversed end Diastolic flow
LSCS	:	Lower segment caesarean section

## CONSENT FORM

PATIENT NAME:

IP/OP NO.

STUDY TITLE :

**“A PROSPECTIVE OBSERVATIONAL STUDY OF MATERNAL RISK FACTORS AND PERINATAL OUTCOME IN FETAL GROWTH RESTRICTION AT TERTIARY CARE HOSPITAL”**

1. I have been explained and have understood the procedures involved in the study
2. I confirm that I have read and understand the information sheet for the above study.
3. I have had the opportunity to consider the information, ask questions and have had these answered satisfactorily.
4. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected.
5. I understand that relevant sections of any of my medical notes and data collected during the study may be looked at by responsible individuals from [Madras Medical College], where it is relevant to my taking part in this study. I give permission for these individuals to have access to my records.
6. I agree to take part in the above study.

Name and signature of interviewer

Signature of Participant

Date:

Date:

# PROFORMA

## A PROSPECTIVE OBSERVATIONAL STUDY OF MATERNAL RISK FACTORS AND PERINATAL OUTCOME IN FETAL GROWTH RESTRICTION AT TERTIARY CARE HOSPITAL”

Serial No:

Name:

Age:

IP No:

Date of Admission:

Time:

Date of Surgery:

Time:

Date of Discharge:

Time:

Socio Economic Status:

BMI

H/O Amenorrhea:

Presenting complaints:

History of presenting complaints:

Obstetric history:

Obstetric score: Gravida    Para    Live/Dead    Abortion

Booked

Previous pregnancy details:

Present pregnancy details:

No of AN USG with / without doppler :

I trimester :

II trimester :

III trimester :

Spontaneous / assisted conception :

Mode of delivery:

Lab nat /LSCS

Comorbids

1.GHT

2.GDM

3.ANAEMIA

Menstrual history:

Age at Menarche:

Menstrual cycles:

LMP:

EDD:

Gestational Age:

Marital history:

Consanguinous / Non consanguinous

Past history:

Family history:

Personal history:

General Physical Examination:

Built:

Nourishment:

General condition:

Height:

Weight:

BMI:

Vitals:

Temperature:

Pulse Rate:

Blood pressure:

Respiratory Rate:

Edema:

Breast

Thyroid:

Spine:

Systemic Examination:

Cardiovascular System:

Respiratory System:

Central Nervous System:

Abdominal Examination:

Per Speculum:

Per Vaginal:

Investigations:

Haemoglobin:

PCV:

Blood Sugar:

1. FBS

2. PPBS

Urine Analysis:

Blood Grouping & Rh typing:

HbsAg:

HIV I & II:

VDRL:

PT:

APTT:

Clotting time:

Obstetric ultrasound:

Details of Delivery:

:

Blood Transfusion:

Time to deliver the baby:

Operating Time:

Duration of Hospital stay:

Maternal outcome:

Fetal outcome:

Birth weight:

Apgar score at 5 minutes:

Preterm birth –Below 37 weeks

No of baby admitted to NICU:

No of still birth:

## CONSENT FORM

PATIENT NAME:

IP/OP NO.

**STUDY TITLE : " A PROSPECTIVE OBSERVATIONAL STUDY  
OF MATERNAL RISK FACTORS AND PERINATAL OUTCOME  
IN FETAL GROWTH RESTRICTION AT TERTIARY CARE  
HOSPITAL "**

1. I have been explained and have understood the procedures involved in the study
2. I confirm that I have read and understand the information sheet for the above study.
3. I have had the opportunity to consider the information, ask questions and have had these answered satisfactorily.
4. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected.
5. I understand that relevant sections of any of my medical notes and data collected during the study may be looked at by responsible individuals from [Madras Medical College], where it is relevant to my taking part in this study. I give permission for these individuals to have access to my records.
6. I agree to take part in the above study.

Name and signature of interviewer

Signature of Participant

Date:

Date:

## INFORMATION SHEET

**TITLE: " A PROSPECTIVE OBSERVATIONAL STUDY OF MATERNAL RISK FACTORS AND PERINATAL OUTCOME IN FETAL GROWTH RESTRICTION AT TERTIARY CARE HOSPITAL "**

Name of the investigator: Dr.R. PUSHPALAKSHMI

Name of the Participant:

Purpose of Research: To Study Maternal Risk Factors and Perinatal Outcome in Fetal Growth Restriction

Study Design: Prospective Observational study

Study Population: This study includes all PREGNANT women (28 — 40 weeks)

Possible Risks:

No risks to the patient

Confidentiality of the Information obtained from you: The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

Can you decide to stop participating in the study? Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at anytime.

How will your decision to not participate in the study affect you? Your decision will not result in any loss of benefits to which you are otherwise entitled.

Signature of Investigator

Signature of Participant

Date:

Place:



## நோயாளியின் ஒப்புதல் படிவம்

கர்ப்பிணி பெண்களின் கர்ப்பகால குழந்தை வளர்ச்சி குறைபாடு ஏற்பட தாய் சேய் காரணிகள் பற்றி அரசு மருத்துவகல்லூரி மருத்துவமனையில் ஆய்வு செய்தல்

முக்கிய ஆய்வாளரின் பெயர் : டாக்டர். ரா. புஷ்பலட்சுமி  
நிறுவன முகவரி : அரசு மகளிர் மகப்பேறு மருத்துவமனை,  
எழும்பூர், சென்னை - 600 008

நீங்கள் இந்த ஆய்வில் பங்கு பெற வரவேற்கப்படுகிறீர்கள், இந்த தாளில் அளிக்கப்பட்டுள்ள விவரங்கள் நீங்கள் ஆய்வில் பங்கு பெறுவது குறித்து தீர்மானிக்க உதவும். சந்தேகங்கள் மற்றும் கேள்விகள் தயக்கமின்றி வரவேற்கப்படுகின்றன.

நாங்கள் இந்த ஆய்விற்காக தலைமை நெறிமுறை குழுவின் (Institutional Ethics Committee) அனுமதி பெற்றுள்ளோம்.

### பகுதி - I

#### நோயாளியின் தகவல் படிவம்:

கர்ப்பிணி பெண்களில் கர்ப்பகால குழந்தை வளர்ச்சி குறைபாடு ஏற்பட தாய் சேய் காரணிகள் பற்றிய ஆய்வு.

இந்த ஆய்வில் மேற்கண்ட பாதிப்புகள் எந்த அளவில் ஏற்படுகிறது. மேலும் அதன் விளைவால் குழந்தைக்குக் ஏற்படும் பாதிப்புகள் பற்றியும் ஆய்வு நடத்தப்படுகிறது.

இந்த பரிசோதனையின் மூலம் எந்தவிதமான பாதிப்புகள் அதிக அளவில் ஏற்படுகிறது எனவும், அதற்கான காரணிகளை கண்டறிந்து அவற்றை சர்செய்து, கர்ப்ப காலத்தில் நிகழும் பாதிப்புகளை முன்னரே அறிந்து கொள்ளவும். அதன் வீரியத்தை குறைக்கவும், அதனால் ஆரோக்கியமான குழந்தைக்கு வழி வகுக்கும் என்பதையும் அறியலாம்.

## உங்கள் தகவல் குறித்த நம்பிக்கை

உங்களை பற்றிய தகவல் (பரிசோதனை விவரங்கள்) எவருக்கும் தெரிவிக்கப்படமாட்டாது. இந்த ஆய்விலிருந்து அறியப்படும் விவரங்கள் கூட்டங்களில், பத்திரிக்கைகளில் இடப்படும் போது உங்களைப் பற்றிய தனிப்பட்ட தகவல்கள் இரகசியம் காக்கப்படும்.

நீங்கள் இந்த ஆய்வில் பங்கு கொள்ளாவிட்டாலும் உங்களுடைய மருத்துவ சிகிச்சையோ அல்லது ஆய்வாளருடன், மருத்துவமனையுடன் உங்களது உறவு பாதிக்கப்படாது. இதனால் உங்களுக்கு கிடைக்கப்பெற இருக்கும் எந்த ஒரு சிகிச்சை முறையிலும் மாறுதல் ஏற்படாது. நீங்கள் இந்த ஆய்வில் பங்கு பெறுவது உங்களுடைய விருப்பம். எந்த நேரத்திலும், எந்த விளக்கமும் அளிக்காமல் நீங்கள் விலகிக் கொள்ள உரிமை உண்டு.

ஆய்வாளரின் கையொப்பம்

பங்கேற்பவரின் பெயர் :

பங்கேற்பவரின் கையொப்பம்

நாள்

இடம்:

சுய ஆய்வு ஒப்புதல் படிவம்

ஆய்வு செய்யப்படும் தலைப்பு :

“கர்ப்பிணி பெண்களின் கர்ப்பகால குழந்தை வளர்ச்சி குறைபாடு ஏற்பட தாய் சேய் காரணிகள் பற்றி அரசு மருத்துவகல்லூரி மருத்துவமனையில் ஆய்வு செய்தல்

ஆய்விடம் :

மகப்பேறு மகளிர் நோயியல் மற்றும் அரசு தாய்சேய் நல மருத்துவமனை, எழும்பூர், சென்னை.

பங்கு பெறுபவரின் பெயர் :

பங்கு பெறுபவரின் எண்:

பங்கு பெறுபவரின் வயது:

மருத்துவமனை எண்:

1. எனக்கு தரப்பட்ட ஆராய்ச்சியில் பங்கு பெறுவோர்க்கான தகவல் படிவத்தை முழுமையாக படித்து புரிந்து கொண்டேன்.
2. ஆராய்ச்சியின் தன்மை முழுமையாகவும் விரிவாகவும் எடுத்து உரைக்கப்பட்டது.
3. எனது எல்லா கேள்விகளுக்கும் விடையளிக்கப்பட்டது.
4. ஆய்வாளர் என் உரிமைகளையும், பொறுப்புகளையும் நன்கு விளக்கினார்.
5. நான் ஆய்வாளருக்கு முழு ஒத்துழைப்பு கொடுக்கவும், பரிசோதனை செய்து கொள்ளவும் அனுமதிக்கிறேன்.
6. எனக்கு இரத்த பரிசோதனை, ஸ்கேன் மற்றும் ஆய்விற்கு தேவையான அனைத்து பரிசோதனைகளும் செய்து கொள்ள சம்மதம்.
7. எனக்கு இந்த ஆய்வின் போது அறுவை சிகிச்சை மேற்கொள்ளும் போது தேவைப்பட்டால் என் வயிற்று பகுதியின் பாதிப்புகளை புகைப்படம் எடுக்கவும், அதனை மருத்துவரின் தேவைக்கேற்பு உபயோகிக்கவும் அனுமதிக்கிறேன்.
8. நான் இந்த ஆராய்ச்சியில் பங்கேற்பதால் ஏற்படும் சாதகபாதகங்களை ஆய்வாளர் விளக்கிக் கூற அறிந்து கொண்டேன்.

9. எப்பொழுது வேண்டுமானாலும் நான் இந்த ஆய்வில் இருந்து விலகி கொள்ளலாம் என்பதை அறிவேன். அவ்வாறு விலகிக் கொள்வதால் எனக்கு கொடுக்கப்படும் சிகிச்சையில் எந்த மாற்றமும் இருக்காது என அறிந்து கொண்டேன்.
10. இந்த ஆய்வுக்காக பெறப்படும் தகவல்களை ஆய்விதழ்களிலோ, கருத்தரங்கிலோ வெளியிட எனக்கு எந்தவித மறுப்போ, ஆட்சேபணையோ இல்லை.
11. எனது அடையாளங்கள் மற்றும் தனிப்பட்ட விவரங்கள் ஆய்விதழ்களிலோ, கருத்தரங்கிலோ வெளியிடப்படமாட்டாது என்று எனக்கு உறுதியளிக்கப்பட்டது.
12. எனக்கு இந்த ஆராய்ச்சி குறித்த சந்தேகம் இருந்தால் உடனே ஆய்வாளரை கேட்டு தெளிவுபடுத்தி கொள்ளலாம் என உறுதியளிக்கப்பட்டது.
13. இந்த ஒப்புதல் படிவத்தில் கையொப்பமிடுவதின் மூலம் இந்த படிவத்தில் உள்ளவையாவும் எனக்கு தெளிவாக எடுத்துரைக்கப்பட்டது. அதை நான் நன்கு புரிந்து கொண்டேன் என தெரிவித்துக் கொள்கிறேன்.

நோயாளியின் பெயர்

கையொப்பம் / பெருவிரல்சுவடு

தேதி

ஆராய்ச்சியாளர் பெயர்

கையொப்பம் / பெருவிரல்சுவடு

தேதி

சாட்சி 1

பெயர் கையொப்பம் / பெருவிரல்சுவடு

தேதி

சாட்சி 2

பெயர் கையொப்பம் / பெருவிரல்சுவடு

தேதி

## **PLAGIARISM CERTIFICATE**

This is to certify that this dissertation work titled “**A PROSPECTIVE OBSERVATIONAL STUDY OF MATERNAL RISK FACTORS AND PERINATAL OUTCOME IN FETAL GROWTH RESTRICTION AT TERTIARY CARE HOSPITAL**”, of the candidate **Dr. PUSHPALAKSHMI. R, REGISTRATION No. 221916883** for the award of **M.S in the branch of OBSTETRICS AND GYNAECOLOGY**. I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion pages and the result shows **TWELVE** percentage of plagiarism in the dissertation (D126295876)






Signature and Seal of the Guide

**PROF DR.N. HEMALATHA, MS., D.G.O.,**  
PROFESSOR OF OBSTETRICS AND  
GYNAECOLOGY,  
INSTITUTE OF OBSTETRICS AND  
GYNECOLOGY,  
MADRAS MEDICAL COLLEGE,  
CHENNAI- 600008

## Document Information

Analyzed document	dissertation.docx (D126295876)
Submitted	2022-01-27T15:28:00.0000000
Submitted by	Pushpalakshmi
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## Sources included in the report

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<b>SA</b>	<b>ASSOCIATION OF MATERNAL FACTORS WITH PERINATAL OUTCOME OF GROWTH RESTRICTED FETUSES WITH REFERENCE TO DOPPLER STUDIES.docx</b> Document ASSOCIATION OF MATERNAL FACTORS WITH PERINATAL OUTCOME OF GROWTH RESTRICTED FETUSES WITH REFERENCE TO DOPPLER STUDIES.docx (D121177072)	 1
<b>SA</b>	<b>ASSOCIATION OF MATERNAL FACTORS WITH PERINATAL OUTCOME OF GROWTH RESTRICTED FETUSES WITH REFERENCE TO DOPPLER STUDIES.docx</b> Document ASSOCIATION OF MATERNAL FACTORS WITH PERINATAL OUTCOME OF GROWTH RESTRICTED FETUSES WITH REFERENCE TO DOPPLER STUDIES.docx (D121271207)	 1

**INSTITUTIONAL ETHICS COMMITTEE  
MADRAS MEDICAL COLLEGE, CHENNAI 600 003**

EC Reg.No.ECR/270/Inst./TN/2013/RR-16  
Telephone No.044 25305301  
Fax: 011 25363970

**CERTIFICATE OF APPROVAL**

To  
**Dr.PUSHPALAKSHMI R,**  
MS (OG), Post Graduate,  
Madras Medical College,  
Chennai - 600003.


Dear Dr. PUSHPALAKSHMI R,

The Institutional Ethics Committee has considered your request and approved your study titled "**MATERNAL RISK FACTORS AND PERINATAL OUTCOMES IN FETAL GROWTH RESTRICTION**"- **NO.18022021**. The following members of Ethics Committee were present in the meeting held on **02.02.2021** conducted at Madras Medical College, Chennai 3.

1. Prof.P.V.Jayashankar :Chairperson
2. Prof.N.Gopalakrishnan,MD.,DM., FRCP, Director, Inst.of Nephrology,MMC,Ch. : Member Secretary
3. Prof. K.M.Sudha, Prof. Inst. of Pharmacology,MMC,Ch-3 : Member
4. Prof. Alagarsamy Jamila ,MD, Vice Principal, Stanley Medical College, Chennai : Member
5. Prof.Rema Chandramohan,Prof.of Paediatrics,ICH,Chennai : Member
6. Prof.S.Lakshmi, Prof. of Paediatrics ICH Chennai :Member
7. Tmt.Arnold Saulina, MA.,MSW., :Social Scientist
8. Thiru S.Govindasamy, BA.,BL,High Court,Chennai : Lawyer
9. Thiru K.Ranjith, Ch- 91 : Lay Person

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

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

  
Member Secretary - Ethics Committee

**MEMBER SECRETARY  
INSTITUTIONAL ETHICS COMMITTEE  
MADRAS MEDICAL COLLEGE  
CHENNAI-600 003.**

S. NO.	NAME	AGE	SE class	BMI	parity	GA IN WKS	GHTN	GDM	ANEMIA	Prev h/o	hypothyroid	APLA	Idiopathic	others
1	kamatchi	25	3	26.1	G3P2L1	40	Severe PE							
2	parimala	38	3	31	primi	35	Y	INSULIN	-	-	-	-	-	-
3	vijayalaksmi	28	3	32.8	g2p1l1	35+5								ITP
4	mageswari	19	3	23.6	primi	39			two prbc					
5	Vishnupriya	31	3	27.6	G5P1L1A3	32+2	severe PE							ITP
6	selvi	37	3	42.1	G3A2	31	Chronic	type 2 dm						
7	priyadarsini	21	4	21.8	primi	37		OHA						
8	pavithra	22	4	32.9	primi	31+1	ghtn							
9	sivaranjini	24	4	33	primi	33+4	ghtn	180u insulin						
10	priya tamil	29	3	22.2	G4A3	37+2		meal plan				1		
11	subasree	25	3	22	primi	36	ghtn							
12	revathi	25	3	32	primi	34	severe PE							
13	devika	20	3	26.5	primi	34	severe PE							
14	rekhavinot	42	3	40.4	primi	29+4	severe PE	12unit insu						
15	devika	35	4	26.5	primi	36	GHTN	GDM						
16	manimegalai	31	3	22.7	primi	37	GHTN							
17	padmapriya	38	4	30.1	G5P1L1A3	36+5	GHTN	INSULIN		PREV h/o				TORCH +
18	nandhini	23	4	32.1	G2A1	37+6	GHTN							
19	Sivabackyam	23	4	31.6	G2A1	36								SICKLE CELL
20	parimala	34	3	32.1	G3A2	34						ACL IGM+		
21	DIVYA	29	3	33.6	G4P2LO	32							1	
22	BABY	26	4	32.1	G4P2L1A1	37			ANEMIA					
23	hemalatha	27	3	27.1	PRIMI	37		GDM						
24	ridhika	19	3	24	primi	32	GHTN							
25	sevagapriya	26	4	23	G3P2L2	36	GHTN							
26	puvitha	22	3	24.1	primi	34	severe PE							
27	sridevi	28	4	28	G2P1L1	38		GDM						
28	kamatchi	25	3	26.1	G3P2L1	40	Severe PE							
29	bharathi	17	3	29	G2P1L1	30	SEvere PE							







96	nirmala	22	3	29.6	G2P1L1	34							
97	darani	22	3	26.5	primi	36	ghtn				1		
98	vijayalaksmi	20	3	31.8	G2P1L1	36						1	
99	anbu	27	3	30.8	primi	36+4							
100	syedalifathima	29	4	26.1	primi	32							

SFH	ga of termin	AFI	DOPPLER	ANsteroids	mgso4	mode of deli	indication	BW	APGAR at	birth	RDS	Jaundice	MAS	SEPSIS	Still birth	discharged
6	40	5	CPR<1			lscs	Fetal distre	2.4	5				1			alive
4	37	10CM	N	4 DOSE	YES	LN	INDUC	2.6	7		1					YES
2	36	4CM	N	4 DOSE	YES	repeat lscs	oligo	2.5	8				1			yes
4	39	7 cm	normal	-	-	lscs	failed ind	2.5	8					1		yes
2	32+2	5 CM	NORMAL	2 DOSE	YES	LSCS	Fetal distre	1.8	5		1	1				yes
3	32	7	normal	4 dose	YES	LN	INDUC	1.5	7	1	1			1	1	
2	38	10cm	cpr<!	-	-	LN	INDUC	2.5	8	observation						yes
2	34	6 cm	cpr<1	4 dose	yes	lscs	failed ind	2.17	8		1					yes
4	34	5cm	cpr<!	4 dose	yes	elective lscs		2.33	8		1					yes
4	38	5cm	normal			lscs	failed ind	1.705	7		1					yes
2	37	5cm	normal			lscs	failed ind	1.85	7		1					yes
4	34+4	adequate	normal	2 dose		lscs	fetal distre	720gms	6	1					1	
6	34	6cm	cpr<1	4 dose	yes	LN	INDUC	1kg	4	1	1					yes
4	34+4	10	ADEF	4 DOSE	YES	LN		1.3	5		1	1				YES
2	37	2cm	cpr<1			elective lsc	svere oligo	2.1	6		1					yes
3	37	4cm	cpr=1			lscs	Fetal distre	2.6	7				1			yes
4	37	8CM	high resist	4 dose		LN	INDUC	1.6	7		1					yES
4	38	8 CM				LN	INDUC	2.9KG	8	OBservation						YES
4	37	10	Normal			LN	SPONTANE	1.5	8		1					YES
2	38WKS	8CM	Normal			LN	INDUC	2.75	7	OBservation						yES
4	37	10	NOrmal	4 DOSE	YES	LN	INDUC	2.2	8	OBservation						yES
4	38	6	normal			lscs	Fetal distre	2.8	7				1			yES
4	38	7CM	normal			lscs	failed ind	2.4	7				1			yES
4	34	6 CM	Normal	4 DOSE	YES	LN	INDUC	1.8	5				1			YES
4	38	7CM	cpr<1			LSCs	failed ind	2.4	7		1		1			yES
2	34	7	cpr<1			lscs	fetal distre	2.3	7			1				yES
4	38	4	AEDF			elective lscs		2.2	6			1				yES
6	40	5	CPR<1			lscs	Fetal distre	2.4	5				1			yES
4	30	6	cpr<!	4 DOSE	YES	LSCS	Fetal distre	1.2	7		1					yES

2	36	8	cpr<1			lscs	fetal distre	2.6	7		1					yes
2	36	6	high resist			lscs	failed ind	2.5	5		1					yes
4	36	8	cpr<1	4 dose	yes	lscs	oligo	2.4	6		1					yES
4	32	7	AEDF	4 dose	yes	lscs elective		1.2	5		1					yes
4		6	CPR<1			LSCS elective		2.6	6		1					YES
2	36	7	cpr<1	4 dose	yes	LN		2.2	8	OBservation						YES
2	36	4	REVERSAL			LSCS elective		2.6	5		1					YES
2	36	2	REVERSAL	2 DOSE	YES	LSCS elective		2.1	4		1			1		YES
2	34	6	AEDF	2 dose	yes	lscs elective		1.1	6		1					yes
4	36	6	cpr<!			LN		2.8	7		1					YES
2	34WKS	6	CPR>!	4 DOSE	YES	LN		1.9	7		1					YES
2	32	2	AEDF	4 DOSE	YES	elective lscs		1.4	6		1					yes
6	36	4	CPR<1	2 DOSE	YES	LN		1.6	5		1					YES
6	39	5	cpr>1			lscs	failed ind	3.1	7				1			yes
2	34	6	normal	4 dose	yes	lscs	cord pres	2.1	6		1					yes
3	36	4	normal			lscs	fetal distre	2.5	8		1					yes
2	36	2	cpr<1			LN		2.7	8	OBservation						YES
4	36	4	CPR<1			LSCS	Fetal distre	2.5	7		1					YES
2	37	7	cpr=1			LN	FAiled ind	3.1	8							YES
2	36	6	AEDF			LSCS	failed ind	1.465	6		1					YES
4	38+4	7	cpr>1			LSCS	FEtal distre	2.8	7		1					YES
4	38	5	cpr<1			lscs	failed ind	2.9	8							yes
6	40+3	4	CPR>!			LN		2.7	6	OBservation						YES
4	40+1	6	cpr<1			lscs	msl	2.6	8					1		yes
4	39	2	normal			lscs	severe olig	2.9	7					1		yes
3	32	4	cpr<1	2 DOSE	YES	LSCS	immin ecl	1.3	4		1					yes
4	38	5	normal	4 dose		LN	INDUC	2.7	7	OBservation						YES
4	36	5	cpr>1			LN		2.6	7						1	YES
4	38	6	Normal			ELECTive lscs		3.1	6				1			YES
4	40	4	CPR<1			LN	INDUC	2.5	8							YES
2	37	6	cpr<1			LN		2.4	7	OBservation						YES
2	37	5	cpr=1			LN		2.4	8							YES
4	38	6	cpr<1			lscs	failed ind	2.6	6		1					YES

4	36	8	cpr<1			lscs	failed ind	2.7	7		1					YES
4	36	7	cpr<1			LN		2.4	8		1					YES
2	40	4	cpr>1			lscs	failed ind	2.5	6		1					YES
4	37	4	cpr>!			lscs	msl	2.9	8							YES
2	40+3	4	cpr>1			lscs	fetal distre	3	6					1		YES
4	34	6	CPR>!			LN	Fetal distre	2.4	7							YES
4	36	6	normal			LSCS	SEVEre olig	2.3	7		1			1		YES
4	34	7	CPR<!			LSCS	fetal distre	2.2	6		1		1			yes
4	36	4	AEDF			LSCS	doppler ch	2.2	6		1					yes
2	34	5	NORmal			LSCS	SEvere olig	2.4	5		1					yES
2	36	7	CPR REVER			LSCS	ELEctive	2.3	7		1					yES
4	38	6	CPR<1			LN		2.6	4		1		1			yES
4	38	4	cpr<1			lscs	oligo	2.6	7	oBservati						yES
6	37	4	normal			LN		2.2	8							YES
4	37	5	cpr<1			ln		2.3	6		1					YES
4	term	6	cpr<1			LSCS	fetal distre	2.9	7		1					YES
4	38	7	normal			LN		2.8	8		1					YES
2	37	6	reversal of flow			lscs	ELEctiv	doppler ch	2.7	4	1					YES
4	37	4	normal			LSCS	failed ind	2.4	6		1					YES
4	36	5	CPR<1			LN		2.3	8					1		YES
4	38	8	normal			LN		2.8	8		1					YES
4	32	7	CPR<1	4 dose	yes	LN		850GMS	2						STILLBIRTH	YES
2	34	8	cpr>1	4 DOSE	YES	LN		1.1	5		1			1		YES
4	36	5	CPR<1			LSCS	OLigo	2.1	6		1					YES
4	37	5	cpr>1			LSCS	FAiled ind	2.4	6				1			YES
4	37+3	6	CPR<1			LSCS	FAiled ind	2.7	4		1					YES
2	32	2	AEDF			LSCS	ELEctive	1.2	4					1		YES
6	36	5	Normal			LN		2.5	5							YES
4	TERM	6	CPR<1			LSCS	FAiled ind	2.6	7				1			YES
4	34	7	CPR<1			LN		2.7	6					1		YES
4	39	6	Normal			LN	failed ind	2.8	7							YES
4	39	7	CPR<1			LSCS	fetal distre	3	8		1					YES
4	39	6	REVERsal of flow			LSCS	ELEctive	2.2	8					1		YES

4	34	6	Normal			LN	FEtal distre	2.3	6		1				YY
4	36	7	Normal			LSCS	OLigo	2.6	4		1		1		YES
4	36	6	Normal			LSCS	FEtal distre	2.6	5		1				YES
2	37	5	Normal			LSCS	failed ind	2.7	7				1		YES
4	34	6	CPR>!			LN	Fetal distre	2.4	7						YES