

**“MEASUREMENT OF SERUM CHOLESTEROL
LEVELS AS A PREDICTOR OF PRETERM DELIVERY”**

Dissertation submitted to

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In partial fulfilment for the award of the Degree of

M.S. OBSTETRICS AND GYNAECOLOGY

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**THE TAMILNADU Dr.M.G.R MEDICAL UNIVERSITY
DEPARTMENT OF OBSTETRICS AND GYNAECOLOGY,
MADRAS MEDICAL COLLEGE**

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BONAFIDE CERTIFICATE

This is to certify that this dissertation entitled “**MEASUREMENT OF SERUM CHOLESTEROL LEVELS AS A PREDICTOR OF PRETERM DELIVERY**” is the bonafide work done by **Dr.C. SRILAKSHMI** Post Graduate in the Department of Obstetrics and Gynaecology, Madras Medical College, Chennai, towards partial fulfilment of the requirements of The Tamil Nadu Dr.M.G.R University for the award of M.S Degree in Obstetrics and Gynaecology.

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DECLARATION

I solemnly declare that this dissertation entitled “**MEASUREMENT OF SERUM CHOLESTEROL LEVELS AS A PREDICTOR OF PRETERM DELIVERY**” was prepared by me under the guidance and supervision of **Dr. Shoba Kumar, MD, DGO.**, Professor, Department of Obstetrics and Gynaecology, Institute of Obstetrics and Gynaecology, Egmore, Chennai.

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

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INTRODUCTION

INTRODUCTION

Pregnancy is considered as an unique and physiologically normal episode in a women's life. All pregnancies are at risk, while most pregnancies are uneventful. About 15% of pregnant women may develop a potentially life threatening complication in turn may require a major obstetrical intervention to survive.

Labour is a naturally occurring phenomenon which usually starts on its own. It is defined as the spontaneous onset of regular and painful uterine contraction associated with a progressive effacement with dilatation of cervix and descent of presenting part, with or without a show or ruptured membrane.

In the era of modern obstetrics, where there has been a rapid advancement in all specialities, preterm labour remains an enigma for the obstetricians today.

Preterm labour is defined by WHO as the onset of labour after the period of viability, that is after 28wks of gestation and before 37 completed weeks or 259 days of pregnancy.

It is estimated that 15million preterm births occur worldwide, of which more than 1million died as result of their prematurity. Preterm birth is associated with significant perinatal morbidity and mortality rates. Moreover, it is associated with an increased risk of adverse metabolic outcomes in later life such as type 2 Diabetes mellitus, Hypertension, CAD & Stroke. The

mothers who delivered preterm infants also appeared to have increased risk of metabolic diseases later in life.

Preterm birth affect over 10% of all pregnancies and leads to significant neonatal morbidity and mortality¹. Prevention of viable spontaneous preterm birth and low birth weight through screening is one of the key aims of antenatal care as these have implications for the child, mother and society. If women can be identified to be at high risk of these adverse birth outcomes in early pregnancy, they can be targeted for more intensive antenatal surveillance and prophylactic interventions (primary prevention).

CHOLESTEROL IN NORMAL PREGNANCY

In normal pregnancy, the concentration of lipids, lipoproteins, apolipoproteins, are elevated. During early trimesters increased maternal fat accumulation is due to increased lipid synthesis, which subsequently leads to hypertriglyceridemia, occurring in late gestation.

Increased insulin resistance and oestrogen stimulation lead to maternal hyperlipidemia. This is primarily due to enhanced entry of triglyceride rich lipoprotein (VLDL), in to the circulation rather than removal. Also the placental lipoprotein lipase normally increases as term approaches.

The plasma TGL & Cholesterol levels increase during pregnancy & enhanced lipolytic activity play a key role in making free fatty acid available to the fetus. Hyperlipidemia is associated with endothelial dysfunction and also regarded as an instigator of inflammation and stress which is a significant

factor in preterm birth. Hence, elevated levels of cholesterol & triglycerides are a marker for increased risk of preterm labour, we have evaluated the elevated cholesterol as a predictor and its association on risk of preterm delivery.

**REVIEW OF
LITERATURE**

REVIEW OF LITERATURE

DEFINITION: Preterm labour is defined by WHO, as onset of labour after the period of viability, that is after 28 weeks and before 37 completed weeks of gestation or 259 days of pregnancy.

Preterm birth is divided into several categories, based on weeks of gestational age:

- 1) Extremely preterm (<28 weeks)
- 2) Very preterm (28 to <32 weeks)
- 3) Moderate to late preterm (32 to <37 weeks).

Moderate preterm birth may be further split to focus on late preterm birth (34 - <37 completed weeks).

Preterm birth is a syndrome with a variety of causes which can be classified into two broad subtypes:

1. Spontaneous preterm birth (spontaneous onset of labour or following pre-labour premature rupture of membranes (PPROM)) and Provider-initiated preterm birth (defined as induction of labour or elective caesarean birth before 37 completed weeks of gestation for maternal or fetal indications (both “urgent” and “discretionary”), or other non-medical reasons. Around 60% of preterm births in the world occur in Africa and South Asia, and it is truly a global problem. India had 3 519 100 preterm birth in 2010.⁴ Spontaneous rupture of membranes usually coincides with labour

INCIDENCE:

The incidence of preterm births ranges from 5% -8% in most developed & developing countries. The incidence in India being 10% - 14%. But it is increasing worldwide which attributed to the raise in multiple gestations from assisted reproductive techniques, better dating scans and iatrogenic deliveries.

SERUM LIPIDS:

The major serum lipids include free (unesterified) fatty acids lysophospholipids , triglycerides , cholesterol (70% esterified with fatty acids) and phospholipids. Lipids are insoluble in aqueous solution, and are transported in plasma complexed with specific proteins or apoproteins. Free fatty acids and lysophospholipids bound by albumin; cholesterol, cholesterol esters and phospholipids exist as large lipid protein complexes called lipoproteins.

Four major classes of lipoproteins are:

1. CHLYMICRONS
2. β LDL
3. PRE β VLDL
4. α HDL

CHYLOMICRONS- These are assembled in intestinal mucosal cells from dietary lipids, primarily through triacylglycerol, which are degraded by lipoprotein lipase.

VLDL- Produced in the liver, composed predominantly of endogenous triacylglycerol (60%). Their function is to carry this lipid from the liver to peripheral tissues.

LDL- These have a high concentration of cholesterol and cholesterol esters. Its primary function is to provide cholesterol to peripheral tissues or return it to the liver.

HDL- These lipoproteins are present in the liver, small intestine. They take up cholesterol from non-hepatic (peripheral) tissues and return it to the liver by **reverse transport**, which is the primary mechanism by which HDL protects from atherosclerosis.

Apolipoproteins function as structural components of lipoprotein particles, co factors for enzymes, ligands for cell surface receptors and determine the metabolic fate of the particle on which they reside.

Six major classes of apolipoproteins:

APO A1 – Major protein of HDL

APO A2 – Unknown

APO A5 – Biochemical and genetic marker of increased triglyceride concentration

APO B48- Main lipoprotein of chylomicrons and LDL

APO B100- Structural protein of VLDL, IDL, LDL for LDL receptor

APO C2-Activates lipoprotein lipase, liberating fatty acid and monoglyceride from chylomicron

APO D- Component of HDL

APO E- Involved in receptor recognition of IDL, chylomicron remnant by the liver.

Serum lipids in pregnancy:

Pregnancy is associated with significant variation in blood rheology consequent mainly to changes in lipoprotein profiles. Although these changes were first described by Bacquerel and Rodier the exact elucidation of these changes is yet to be defined. An extensive review of literature revealed conflicting observation and implication of lipoprotein fractions metabolism in normal and abnormal pregnancies. All plasma lipoprotein fractions undergo striking increase during pregnancies. All lipids levels significantly increase in second and third trimester.

- Total triglycerides increase 2-3 fold
- Total cholesterol rises 50-60% above non – pregnant levels.
- APO-B increases by 56%
- Total cholesterol increases by 43%
- LDL increases by 36%
- APO-A increases by 32%
- HDL increases by 25% in second trimester

These changes are gradual and increase progressively throughout pregnancy after 25 weeks and peaks at term and return to pre pregnancy level 6-8 weeks post-partum.

Quantitatively some lipoproteins such as HDL, LDL becomes TGL enriched. VLDL however increases cholesterol and TGL 2-5 fold compared to non-pregnant levels. LDL, after an initial drop at 8 weeks, rises steadily up to term by 45-50%. It is the only lipoprotein to remain elevated even after 8 weeks of delivery irrespective to lactation. Post-partum measurements of lipids should be delayed for 6 months in women who did not have hypercholesterolemia prior to pregnancy.

HDL is of a particular interest. Unlike other lipoproteins, which rises through 36 weeks, this lipoprotein reaches to its maximum level at mid gestation. It increases by 45% until 24 weeks and subsequently falls to 15% above non-pregnant level. These changes are confined to HDL sub fraction. HDL changes little.

During early pregnancy, triglycerides, and dense LDL were higher than in the nonpregnant state. With advancing gestation, triglycerides increased and the distribution of apolipoprotein B-100-containing lipoproteins became increasingly dominated by the accumulation of very low density and intermediate density lipoproteins and buoyant, triglyceride-rich LDL. This is the first study that investigates LDL sub fractions in pregnancy using a method that strictly separates LDL sub fractions by virtue of density. The accumulation of buoyant, triglyceride-rich lipoproteins may be related to the down-regulation of maternal lipase activities by placental hormones. As a consequence, the metabolic changes of late pregnancy may result in an

increased flux of lipoprotein-derived lipids to the placenta, which, with advancing gestation, increasingly expresses receptors with a high affinity for triglyceride-rich lipoproteins. During gestation, the effect of oestrogen in enhancing very low density lipoprotein (VLDL) production and decreasing hepatic lipase activity plays a key role in the accumulation of triglycerides in lipoproteins of density higher than VLDL.

The physiology hyperlipidemia is of a potential significance from several point of view increase in plasma TGL may enhance available essential and non-essential TGL and free fatty acids for placental transfer to foetus

- a) LDL rise appears to be necessary for placental steroidogenesis and also for transplacental cholesterol transfer to foetus.
- b) TGL-increases may be a parameter of a general metabolic adaptation by mother to augment nutrient flow to foetus.

The hyperlipidemia may stress maternal lipid homeostasis to an extent that sub clinical hyperlipidemia may be detectable analogous to the prediabetic recognition in women she develops gestational diabetes.

In a review article herrara stressed that during early pregnancy there is increased body fat accumulation associated with both hyperplasia and lipogenesis during late pregnancy that is an accelerated breakdown to fat depots, which play an important role in fetal development.

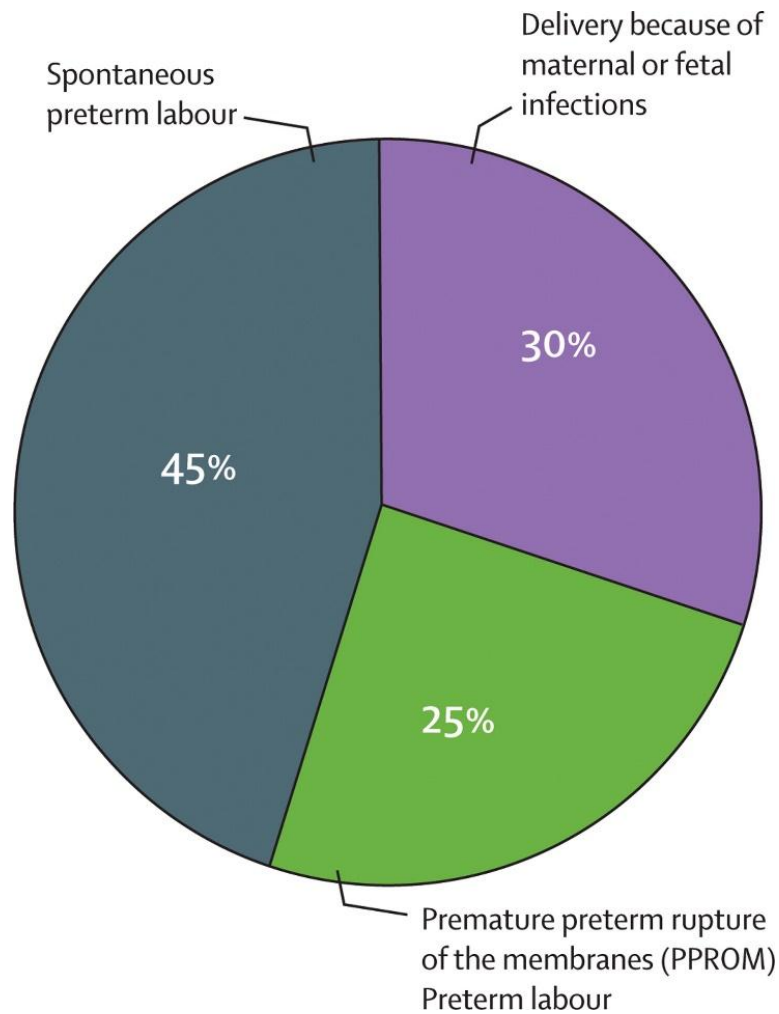
LIPIDS	NON-PREGNANT ADULT	FIRST TRIMESTER	SECOND TRIMESTER	THIRD TRIMESTER
TOTAL CHOLESTEROL (Mg/Dl)	< 200	141 – 210	176 – 299	219 – 349
TRIGLCERIDES (Mg/dl)	< 150	40 – 159	75 – 382	131 – 453

Reference: WILLIAMS OBSTETRICS 24TH EDITION

ETIOLOGY OF PRETERM LABOUR:

The reasons for preterm birth have multiple interacting antecedents and contributing factors. Only when this complex factors causing prematurity are clearly understood, any attempt at prevention and management can be made.

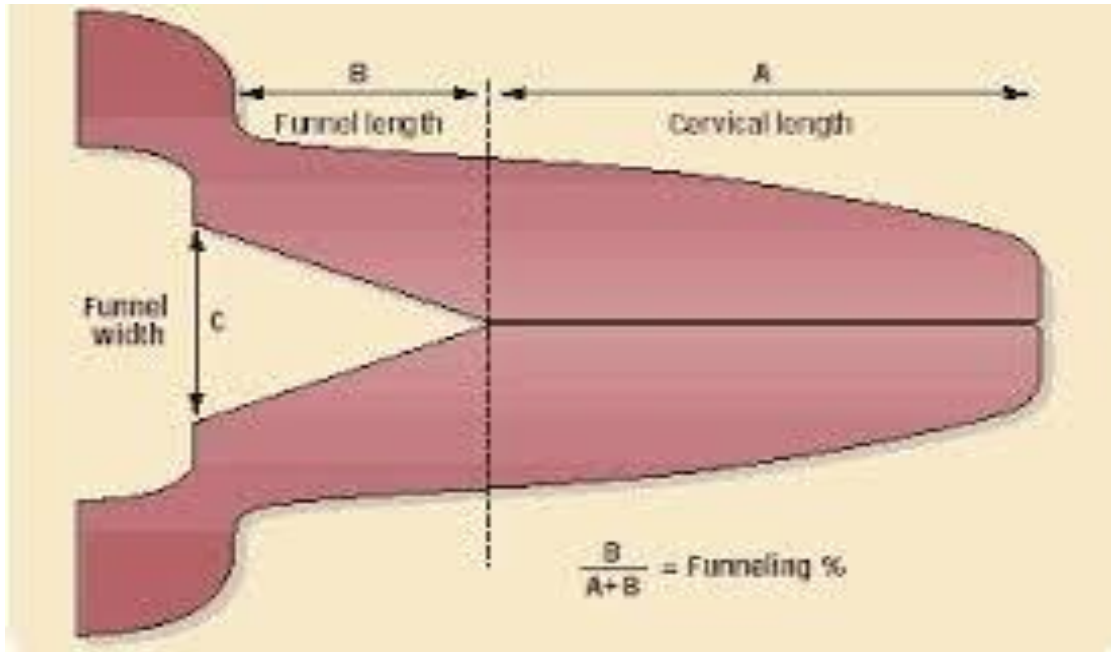
- 50-60% Preterm births occur following spontaneous unexplained labour.
- 30-35% follow idiopathic preterm premature rupture of membranes (PPROM).
- 30% are iatrogenic, delivery for maternal or fetal indication.



CERVICAL INSUFFICIENCY

This is defined as painless cervical effacement and dilation resulting in second trimester loss or preterm delivery.

- Prior preterm labour and short cervical length
- H/O cervical surgery – cone biopsy, large loop excision of transformation zone(LLETZ) laser ablation of the cervix
- H/O obstetric trauma – Cervical laceration or injury during labour or delivery (spontaneous, vacuum, forceps or caesarean)
- Multiple dilatation and evacuation



UTERINE CAUSES:

- Congenital abnormalities 1-3% (septate and bicornuate)
- Uterine over distension – multiple pregnancies, polyhydramnios

INFECTIONS:

- Subclinical – increased dominance of lactobacillus species
- Intrauterine infection
 - Group B streptococcus
 - Chlamydia trachomatis
 - Mycoplasma hominis and ureaplasma urealyticum
 - Asymptomatic bacterial vaginosis and trichomonas vaginalis confers a modest risk of spontaneous preterm labour
 - Gardnerella vaginalis, fusobacterium (Gerber, 2003; Hillier, 1988; Yoon, 1998)

- Extra uterine infection
 - High prematurity rate is associated with asymptomatic bacteruria and UTI (Robertson et al)
 - Other are systemic illness like pneumonia, pyelonephritis periodontal disease is associated with preterm labour. (Xiong X 2006)

Connective Tissue Disorder:

- Ehlers – Danlos Syndrome
- Marfans Syndrome

Fetal:

- Congenital abnormalities

Placental:

- Abnormal placentation (causing decreased uteroplacental blood flow)
- Anatomical abnormalities, Placenta Praevia
- Abruptio placenta

Hyperlipidemia:

Catov et al reported that an elevation in maternal triglycerides or cholesterol level in early gestation was associated with a greater than 2 fold increased risk of preterm delivery.

Genetic:

- CAP Genes (contraction associated proteins) in the myometrium – connexion 43, for oxytocin receptors (Korita, 2002; Lyall, 2002; Sooranna, 2004)
- GRP (Gastrin Releasing Peptide)
- TREK – 1 Potassium channel regulator (Buxton, 2010)
- IL – 1 β , IL – 6, IL – 8, TNF α
- MCP – 1 (Monocyte – chemotactic protein)
- CCL – 2 (chemokine C-C motif ligande-2)
- MMP – 1, MP – 2, MMP – 9 (matrix metallo proteinase)



Pathophysiology: The control of parturition is achieved by complex integration of endocrine, paracrine and autocrine mechanism.

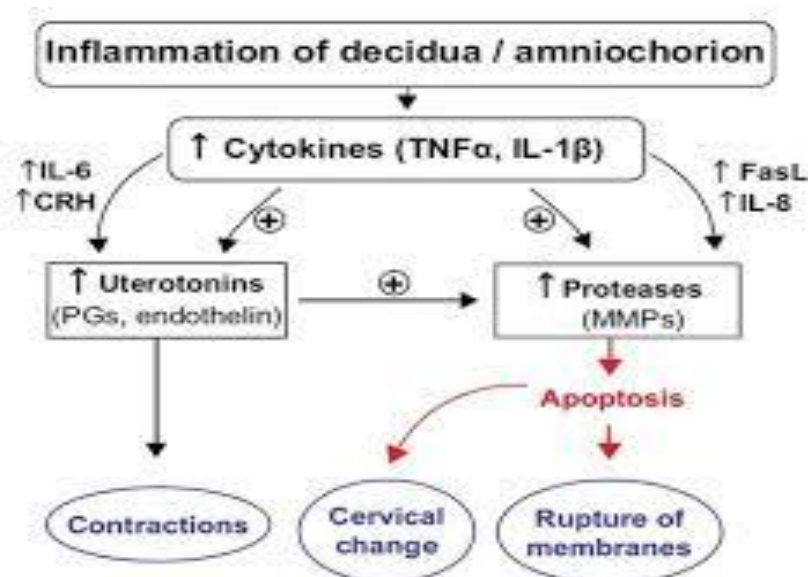
Early uterine distention acts to initiate expression of Contraction Associated Proteins (CAP) and early activation placental – fetal – endocrine cascade.

Rising maternal serum levels placental derived corticotropin – releasing hormone (CRH), which works with adreno corticotropin hormone (ACTH).

This increases adult & fetal adrenal steroid hormone production, causes initiation of fetal cortisol biosynthesis.

Rising levels of CRH stimulate the fetal adrenal dehydroepiandrosterone sulphate (DHEA- S) bio-synthesis. Which, acts as a substrate to increase maternal oestrogen (estriol).

The production of PGE2 and PGF 2 α by the enzymes phospholipases, 15 hydroxy prostaglandin dehydrogenase may be the key in the balance between uterine quiescence and activity. The decidual activation and production of uterotropins is the penultimate event initiation of labor.



Lipid changes in preterm delivery:

Maternal hyperlipidemia is one of the most consistent and striking changes to take place in lipid metabolism during pregnancy. The mechanics responsible for these changes include, increased lipolytic and decreased LPL activities in adipose tissue. (Herrera and Collegues, 2006). The hepatic effects of estradiol and progesterone also play an important role (Desoye and associates 1987).

We have several hypothesis for why high FFA may increase risk of spontaneous preterm delivery.

1. Maternal circulating FFAS can be transferred to the fetus across the placenta. In the support of this hypothesis was the observation that maternal plasma total FFAS were positively correlated with cord plasma FFAS. These include the essential FFAS and their metabolically important derivatives such as arachidonic acid a precursor of the eicosanoids including prostacyclins and prostaglandins.
2. It is known that prostaglandin play an important role by stimulating the uterine contractions that drive preterm delivery, Reece et al reported a higher proportion of maternal RBC and plasma arachidonic acid in preterm cases compared with controls. Thus this could be indirect evidence for excessive maternal arachidonic availability or mobilisation in preterm labor.

3. High FFAS may link to pro-inflammatory pathways. Preterm labor is recognised as an inflammatory phenomenon, even in the absence of infection, specific unsaturated fatty acids, particularly the essential fatty acid linoleic acid, which comes from the maternal diet.
4. Selectively stimulate the development of a proinflammatory response in human endothelial cells studied in Vitro, increases in inflammatory cytokine formation in human decidua stimulate prostaglandins synthesis. Thus high maternal circulating FFA could be inked to inflammation a known risk factor for preterm delivery.

PREDISPOSING FACTORS:

1. Previous Preterm Birth

The recurrent risk for women whose first delivery was preterm was increased 3 fold (Spong, 2007)

Birth Outcome	Recurrence Risk
1 st Birth > 35 weeks	5%
1 st Birth < 35 weeks	16%
1 st & 2 nd Birth < 34 weeks	41%

(Williams Obstetrics 24th edition)

2. Previous Threatened Abortion

3. Cervical incompetence

4. Uterine anomalies

5. Pregnancy complications

- Hydramnios
- Multiple pregnancies
- Preclampsia
- Antepartum haemorrhage

6. Birth Defects was found to be associated with preterm birth and low birth weight (FASTER trial)

7. Stress

Physical work and psychological stress are associated with increased preterm birth (Lockwood, 1999; Petrglia, 2010; Wadhwa,

2010). Also preterm birth is increased in women living alone and those subjected physical abuse.

8. Life Style Factors

- Cigarette smoking, illicit drug abuse (cocaine) (Berns, 2002)
- Poor nutrition, Inadequate maternal weight gain
- Over weight and obese mothers (Cnattingius, 2013)
- Young age or advance maternal age (under 17 and over 35)
(Lumley JM et al 1993)
- Short stature
- Low social economic status tend to be less educated regarding general perinatal and antenatal care (Goffinet F 2005)
- Occupational factors - Long working hours and hard physical labor

9. Inter-pregnancy Interval

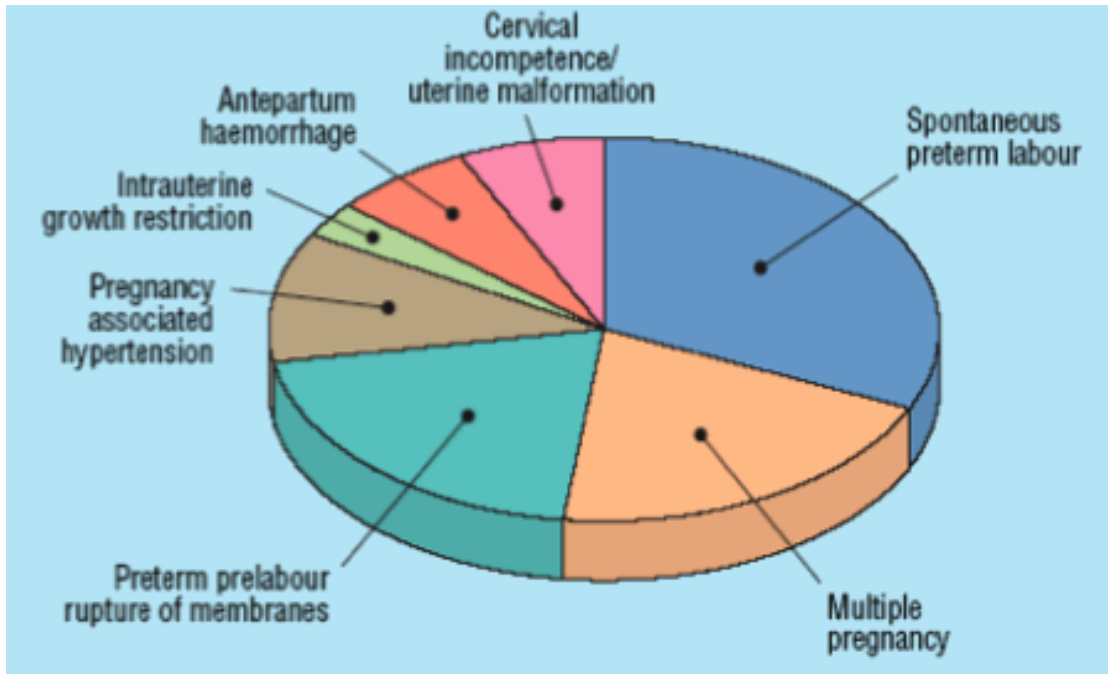
Intervals < 18 months and > 59 months were associated with increased risk for preterm birth and small for gestational age (Conde – Agudelo, 2006)

10. Periodontal Disease

Gum inflammation was significantly associated with preterm birth (ODDS ratio 2.83)

11.Race

The incidence is greater in non-Hispanic black and American Indian / Alaska native (Collins, 2007)



NEONATAL COMPLICATIONS:

Approximately 70% neonatal deaths, 36% infant deaths, 25 to 50% cases of long term neurological impairment in children can be attributed to preterm birth (ACOG, 2016)

- Birth asphyxia
- Respiratory distress syndrome
- Hyaline membrane disease

It is the most important threat when the baby is delivered before 37 weeks of gestation. The incidence RDS is estimated to decrease from 15% at 34 weeks to below 1% at 37 weeks. The incidence RDS was 22.5% in 33 weeks and 5.8% in 34 weeks. It was relatively low

after 34 weeks, it still affects neonates upto 36 weeks with incidence of 10.4% in 35 weeks and 1.5% in 36 weeks. The incidence of RDS was nearly 4 – 5 fold higher in the preterm patients than in term patients.

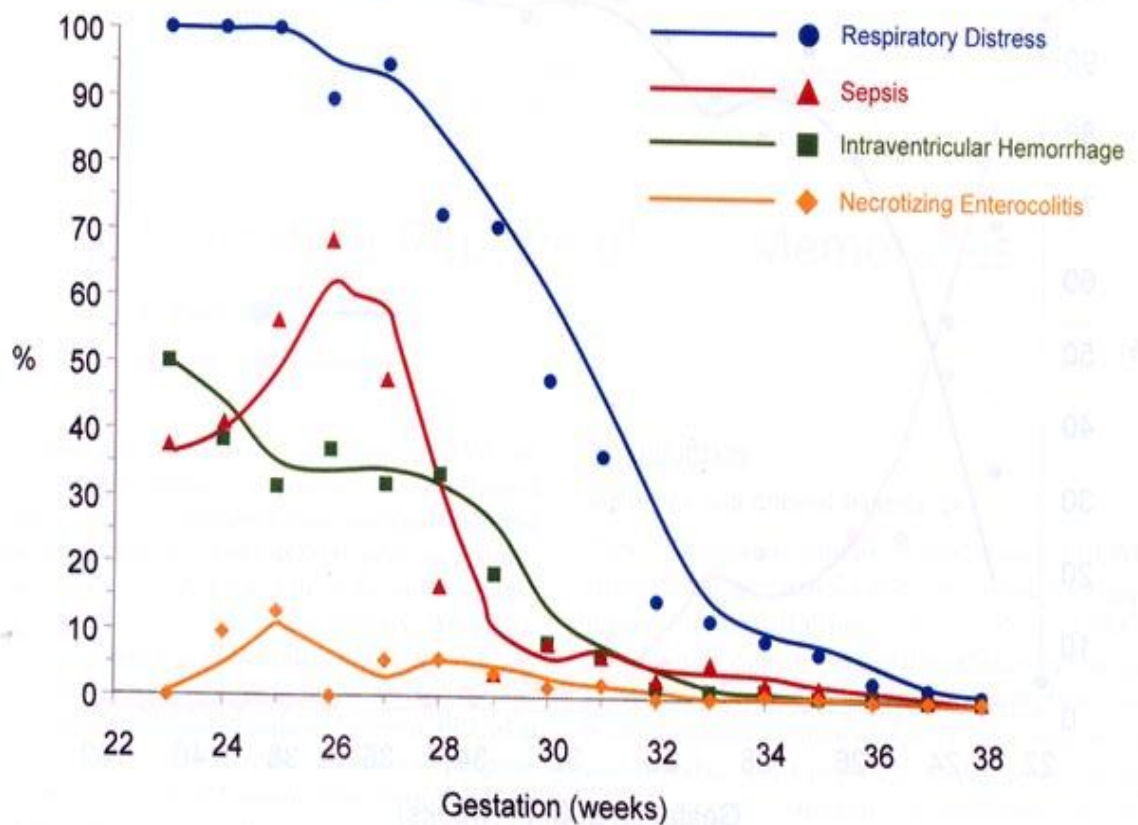
- Apnea of prematurity
- Broncho pulmonary dysplasia
- Neurological damage
- Cerebral palsy

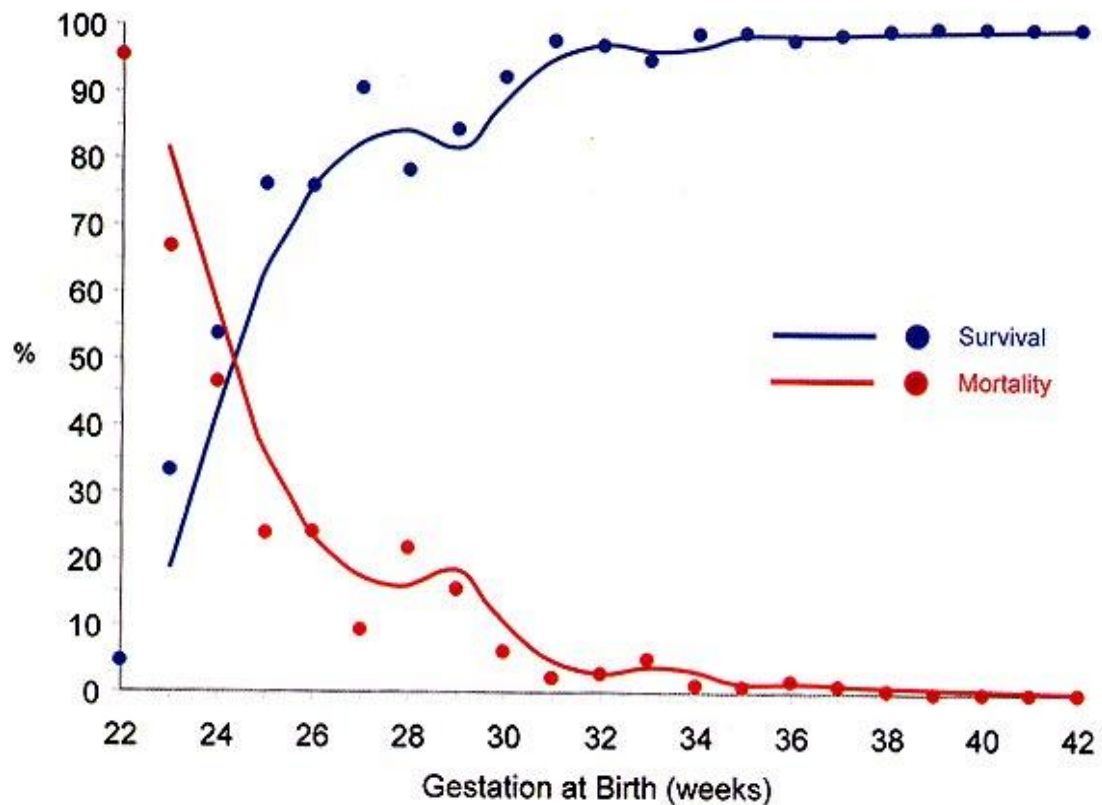
It is long term sequelae especially when complicated with intra ventricular haemorrhage, intra partum fetal acidosis and hypoxia.

- Neuro developmental delay, learning difficulties
- Reduced growth potential
- Sensorineural deafness
- Hyperbilirubinemia
- Necrotizing enterocolitis
- Failure to thrive
- Hospital acquired infection
- Retinopathy of prematurity
- Retrolental fibroplasia
- Patent ductus arteriosus
- Anemia of prematurity
- Recurrent respiratory infection

- Metabolic problem
- Sudden infant death syndrome

The cost of preterm birth can be measured in terms of mortality and morbidity and in short term and long term financial costs which increase with lower gestational age. The components at the costs are nursery, medical staff, stay in neonatal intensive care unit and treatment. Such as ventilation, artificial surfactant, recombinant erythropoietin and surgical procedures. The long term financial implications are unknown if the child is handicapped either physical or mental.





PREDICTION OF PRETERM LABOUR

Cervical assessment: Asymptomatic cervical dilatation after mid pregnancy has gained as a risk factor for preterm delivery. (Copper,1995; Pereira,2007)

Owen and Colleagues (2003) concluded that the value of cervical length to predict preterm birth before 35 weeks is apparent only in women at high risk for preterm birth.

Cervical Length analysis using transvaginal sonography is safe, highly reproducible and more predictive than transabdominal sonography. TVS is

not affected by maternal obesity cervix position or shadowing from fetal presenting part.

Goldenberg and Co-workers found the main cervical length of less than 25mm at 24 weeks of gestation to be a strong predictor of preterm birth.

A positive predictive value of 70% and sensitivity of 60 to 80% is observed with a transcervical ultrasound cervical length (TVS CL) of less than 25mm between 14 to 18 weeks of gestation.

Ultrasound is a better modality than digital evaluation of cervical length because the upper half of cervix which cannot be reached digitally can be measured by ultra-sonogram.

Fetal breathing movements: Absence of fetal breathing movement on ultrasound done at the time of admission on women who presented with threatened preterm labor was also found to be accurate test in predicting spontaneous preterm birth.

Uterine Activity Monitoring

ACOG (1995) has concluded that for most patients home uterine monitoring is not better than frequent nursing contact and support. IAMS and Associates (2002) found that no contraction pattern efficiently predicted preterm birth.

Only patient, who cannot recognize adequately the presence of contractions like multifetal gestation and other over distended uterus may benefit from home uterine monitoring.

Fibronectins

Fetal fibronectin is an extracellular glycoprotein secreted by chorionic tissue at maternal and fetal interface and is present in large quantities in amniotic fluid. The presence of fetal fibronectin in cervicovaginal secretions in late second and early third trimester has been proposed as a specific predictor of preterm labor (Lock wood and coworkers, 1991). It represents disruption of choriodecidal interface which can be caused by preterm labor. It can be detected in the ectocervix and vagina, is measured by ELISA with FDC-6 Monoclonal antibody and values exceeding 50ng/ml are considered positive result. However, of concern is the high false positive rate if there is contamination with amniotic fluid, semen, maternal blood and in patients with cerclage. A positive test is associated with an increased likelihood of birth before 34 weeks of gestation within 14 days of test with a positive predictive value of 16%.

The high negative predictive value of fetal fibronectin can be used to influence management (Honest h et al 2002).

In symptomatic women the group found that cervicovaginal fetal fibronectin and absence of fetal breathing movements of ultrasonogram are likely to be accurate in predicting preterm birth.

New Predictors of Preterm Labor

- Cervical electrography
- Aspiration and CCI

- Quantitative USG and Beam Steering
- Shear wave speed estimation

Biochemical Markers

1. Salivary oestriol: progesterone ratio
2. Salivary oestriol >1.8/ml before 34 weeks has a sensitivity of 68% and specificity of 76% for preterm labor before 35 weeks of gestation (Darne et al)
3. Serum collagenase
4. Tissue inhibitor of metalloproteinase (TIMP) / Matrix
5. Relaxin
6. Corticotrophin Releasing Hormone (CRH)
7. Human chorionic gonadotropin
8. Mediators of inflammation and infection
 - a) C-Reactive protein
 - b) Granulocyte elastase
 - c) Cytokines (IL-6, TNF)
 - d) Amniotic fluid glucose concentration
 - e) Zinc and Lipocortin – 1 (Romeo R et al)
 - f) Positive cultures
 - g) Granulocyte colony stimulating factor

These are not practically helpful in prediction of preterm labor

DIAGNOSIS OF PRETERM LABOR

Preterm labour is defined as regular contractions (6/60m) with documented cervical changes before completion of 37 weeks of gestation (or) cervical dilatation of >2cm and 75% effacement with history of contractions.

SYMPTOMS OF PRETERM LABOUR

- Persistent uterine contractions
- Low, dull back ache
- Pressure (Feels like baby is pushing down)
- Intermittent Abdominal cramping
- Increase or change in vaginal discharge
- Vaginal spotting/bleeding

Assessment of Patients in Preterm Labor

- Labs-CBC, UA +/- culture, electrolytes
- Sterile speculum exam obtaining cultures for group B strep, BV, GC, Chlamydia, obtain fetal fibronectin
- Cervical length measurement
- The last thing is the cervical digital exam

1. Pelvic examination

2. Ultrasonogram

Ultrasonographic assessment in preterm labor

- Fetal viability
- Gestational age

- Estimated fetal weight
- Transvaginal cervical assessment

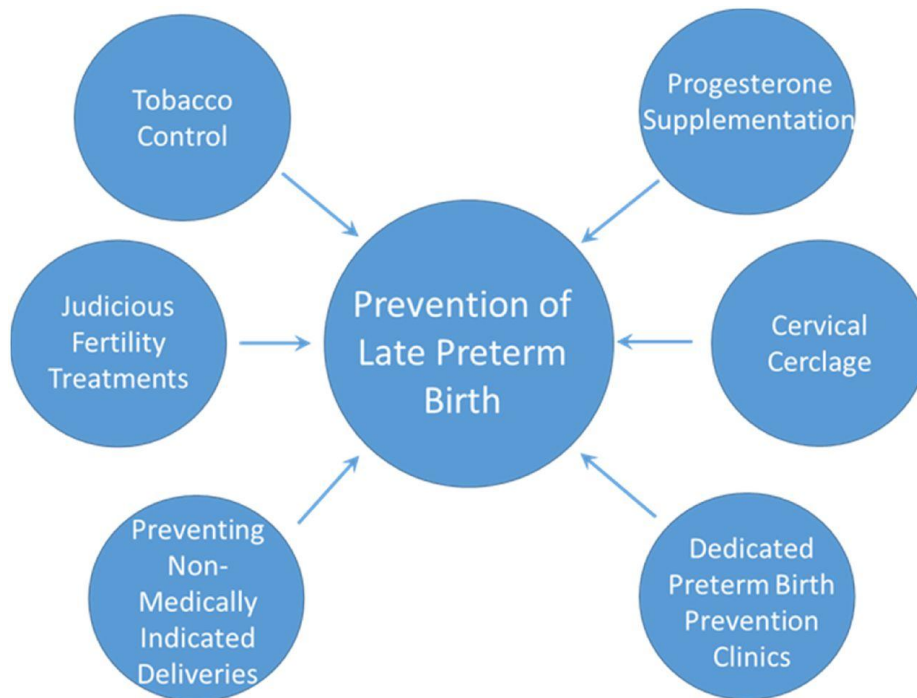
CERVICAL ULTRASOUND

- Cervical length <20mm/contraction criteria-preterm
- Cervical length 20-30mm/contraction criteria- probable
- Cervical length >30mm- preterm labor unlikely regardless of contraction frequency
- Fetal breathing movements
- Amniotic fluid volume
- Number of fetus
- Fetal presentation and lie
- Fetal movements and tone
- Fetal anomaly
- Placental localisation and morphology
- Uterine Fibroid and adnexal mass

3. Tococardiography

The amplitude, duration, shapes of contraction frequency and basal tone and monitored. The uterine activity is monitored. Changes in FHR (Fetal Heart Rate) pattern occurring in preterm labor is normally due to immaturity of cardiovascular system. Repetitive late decelerations, absent variability decelerations are sign of placental insufficiency.

PREVENTION OF PRETERM LABOR



I Basic care:

- a) Support system of family should be developed
- b) Suggestions on coping with physical and mental stresses of maintaining a pregnancy should be described
- c) Education, supportive services from health care provided and financial issues are of common concern
- d) Behavioural and Life style modification
 - i. Smoking cessation (Burguet et al)
 - ii. Adequate nutrition

II Bed rest and Hydration

Although bedrest and hydration are widely used as the first step of prevention and treatment, there is no evidence that this practice is beneficial (Freda MC et al, Goldenberg RL et al.,)

Bed rest should be advised with caution after evaluating its benefits & risks in an individual, and not routinely keeping in mind its adverse like venous thrombosis and pulmonary edema.(Kovacevich,2000)

Women with activity restriction were nearly 2.5 times more likely to have a preterm birth before 34 weeks.(Grobman&associates 2013)

Aggressive Treatment of Cervicovaginal Infection

- According to ORACLE TRIAL II (2008) Kenyon & associates routine administration of antibiotics for treatment of preterm labour with intact membranes is not recommended.

25- 40% PTL results from subclinical intrauterine infections

- GBS prophylaxis
- Inj. AMPICILLIN 2g IV initial dose then 1g every 6 hrs till delivery (or) 250mg ORALLY every 8hrs FOR 7 DAYS
- Inj. Cefazolin,2g IV initial dose then 1g every 8 hrs
- oral erythromycin 250 mg 4 times a day for a maximum of 10 days or until the woman is in established labour (whichever is sooner).

- Who cannot tolerate erythromycin or in whom erythromycin is contraindicated, consider oral penicillin for a maximum of 10 days or until the woman is in established labour (whichever is sooner).
- Do not offer women with P-PROM co-amoxiclav as prophylaxis for intrauterine infection due to increased risk of cerebral palsy.
- Antibiotics reduce risk of persistent infection but did not have reduction in risk of Preterm birth.
- Bacterial vaginosis has been consistently associated with a 1.5 to 3 times increased risk of spontaneous preterm birth. But the efficacy of treatment in reduction of preterm births is conflicting (Goldenberg R, et al, 1998). But recent systematic review by Varma R, Gupta JK 2006 concluded that screening and treatment of asymptomatic bacteruria and bacterial vaginosis in low risk population groups may reduce the rate of preterm deliveries.

Cervical Encerclage

A short cervix diagnosed by ultrasound in asymptomatic women may be an indication for cerclage. The role of cervical for the prevention of preterm delivery is now disputed. A number of systematic reviews which demonstrate a trend towards reduction in preterm delivers before 34 weeks in high risk women who had cerclage compared to those managed expectantly (Honest H, et, al)

PROPHYLACTIC CERCLAGE:

- H/O 2nd trimester loss associated with painless cervical dilation.
 - H/O cerclage in previous pregnancy
 - Cervix length less than 2.5cm before 24 w(TVS)
 - H/O spontaneous preterm birth(less than 34 w)
 - Currently has painless cervical dilation
- Consider 'rescue' cervical cerclage (Terkildsen&workers,2003) for women between 16⁺⁰ and 27⁺⁶ weeks of pregnancy with a dilated cervix and exposed, unruptured fetal membranes:
 - FAILURE>4CM dilatation
 - Explain about the risks VS benefits

Progesterone

Weekly intramuscular administration to women at high risk for preterm labor resulted in lower rates of preterm birth and perinatal mortality when compared with that placebo, Meis and collaborators (2003). The dose used by Meis et al was 250mg of 17 hydroxy progesterone caproate, intramuscularly every week from 20 to 36 weeks.

A Cochrane Systematic review in 2006 by Dodd JM et al found that the use of progesterone on women with history of spontaneous preterm birth resulted in a reduction in risk of preterm birth before 34 weeks of gestation and infant birth weight than 2500 grams. But the dose, route of administration and time commencement of therapy has not been arrived conclusively by this

study for need of further information. For women with threatened preterm labor the role progesterone is uncertain as per this review.

Randomized Trials of Progestin Compounds Given Prophylactically to Prevent Preterm Labor

Investigator	Cervical Length	Progestin Compound	Progestin vs Placebo
Fonseca (2007)	< 15 mm	Progesterone, 200 mg vaginal capsules daily	Delivery < 34 weeks: 19% vs 34% P = .02
Hassan (2011)	10 – 20 mm	Progesterone, 90 mg vaginal gel daily	Delivery < 33 weeks: 9% vs 16% P = .02
Grobman (2012)	< 30mm	17 – OHPC, 250 mg IM weekly	Delivery < 37 weeks: 25% vs 24% P = NS

Williams 24th Edition

- Offer a choice of prophylactic vaginal progesterone or /AND prophylactic cervical cerclage to women:
- with a history of spontaneous preterm birth or mid-trimester loss between 16⁺⁰ and 34⁺⁰ weeks of pregnancy and

- in whom a transvaginal ultrasound scan has been carried out between 16⁺⁰ and 24⁺⁰ weeks of pregnancy that reveals a cervical length of less than <25 mm

MANAGEMENT OF PRETERM LABOR

Hydration and Bedrest

Steroids

In 1995, a national Institute of Health Consensus development, panel recommended corticosteroids for fetal lung maturation in preterm labor. Since then there has been nearly universal acceptance and implementation of these recommendations.

- Consider maternal corticosteroids for women between 23 TO 35⁺⁶ weeks of pregnancy who are in suspected, diagnosed or established PTL, are having a planned PTB, have P-PROM.
- **REDUCES RISK OF**
 - RDS-34%
 - IVH-46%,
 - NEONATAL DEATH-31%
- **SINGLE COURSE- at 24-34 WEEKS**
- **GREATEST BENEFIT 48 Hrs -7 DAYS**
- **RESCUE COURSE**

- Administer a course of ANCS in patients who have received prior course, but do not deliver in 7-14 days, upto 34 wks may receive a repeat course
- **RCOG**
 - SINGLE COURSE 24 – 35WKS
 - All Antenatal , for whom elective LSCS planned prior to 38 wks.

Recommended regimens includes a single course of two doses of 12mg of betamethasone given intramuscularly 24 hours apart, or four doses of 6mg of dexamethasone given intramuscularly 12 hours apart.

Although benefit on neonatal outcome is maximum between 24 hours and 7 days after initiation of therapy, steroids confer survival advantages even when delivery occurs within 24 hours. Therefore, treatment should not be withheld when delivery is probable within 24 hours

Tocolysis:

Tocolysis is pharmacological suppression of uterine activity

Indication:

Preterm delivery is a major cause of perinatal morbidity and mortality. Tocolytic agents are effective in reducing the likelihood of delivery within 48 hours but do not reduce the overall risk of preterm delivery.

Consideration should be given for administration of tocolytics to all women experiencing preterm labor when there is a delay in delivery

- To permit in – utero transfers to a tertiary perinatal centre for multidisciplinary management (obstetrician, neonatologist, anaesthetists)
- To gain upto 48 hours to allow for the administration of corticosteroid to enhance pulmonary maturity.

BETA SYMPATHOMIMETICS

Caritis et al 1976, noted that small doses of epinephrine inhibited uterine hyperactivity. Efforts to produce an epinephrine like compound which lacked the cardiovascular stimulant effect culminated in the synthesis of β agonists.

I generation: - Isoxsuprine, Orciprenaline, isoprenaline

II generation: - Ritodrine, terbutaline, fenoterol.

The most commonly used β_2 agonist for tocolysis is ritodrine, then are terbutaline and salbutamol.

Ritodrine:

Ritodrine infusion is started at a dose of 50 μ b/min and increased every 20 minutes until uterus is quiescent or side limit of dose. Side effects are palpitations, tremor, nausea, headache, chest pain, dyspnoea, pulmonary edema, hypokalemia, myocardial ischemia, arrhythmias.

Terbutaline:

Not used as much as ritodrine, but is effective in temporarily suppressing contraction when given parenterally.

Intravenous dose is 5-10µg/min, increased every 10-15 min to a maximum of 80µg. 2.5 – 5mg is given orally every 4-6 hours & 250µg subcutaneously every 20 – 30 min given as 4-6 doses. Terbutaline has higher a risk of hyperglycemia than ritodrine. Other side effects are similar. But β₂ agonists are no longer the first choice of drugs for tocolysis because of their side effects (RCOG Clinical Guide Lines, 2002 and Anatayanonth et al, 2004)

Contraindications of β₂ agonist:

- Symptomatic cardiac disease especially ventricular outflow obstruction
- Conduction disturbance
- Hyperthyroidism
- Sickle cell disease
- Uncontrolled maternal diabetes mellitus
- Chorioamnionitis
- Eclampsia or severe preeclampsia
- Multifetal gestation
- Severe obstetrical bleeding

MAGNESIUM SULPHATE

MgSO₄ uncouples the depolarisation contraction coupling. During the depolarisation of myometrial cells, Mg⁺⁺ competes with Ca⁺ to for entry into the cell causing less intracellular Ca⁺ to participate in actin –myosin interaction during smooth muscle contraction. It affects neural transmission by modifying acetyl choline release and sensitivity at motor end plate.

Contractility is inhibited at serum level of 5-8 mEq/dl. Deep tendon reflexes are lost at 9-13 mEq/dl. Respiratory depression occurs at >14 mEq/dl.

Dosage: Intravenous loading dose of 4g administered over 20 mins followed by maintenance dose of 1-2 g/hr.

Side effects include flushing, dizziness, nausea, lethargy, chest tightness, Hypocalcaemia, Pulmonary edema, respiratory depression and depressed motor, respiratory activity in fetus. It is contraindicated in myasthenia gravis, heart block, renal disease, recent myocardial infarction.

Magnesium sulphate is an ineffective tocolytic agent as shown by a Cochrane systematic review (Crowther et al, 2002., Cox etal 1990)

PROSTAGLANDIN SYNTHETASE INHIBITORS

Drugs like aspirin, indomethacin, naproxen, fenamate were found to inhibit the prostaglandin synthesis, decrease the myometrial gap junction and decrease the influx of calcium.

Maternal side effects include nausea, vomiting, drug rash headache, gastritis, diarrhoea. In fetus it produces constriction of ducts arteriosus,

pulmonary hypertension and oligohydramnios. Intraventricular haemorrhage, necrotising enterocolitis have also been reported.

They are effective as single dose in inhibiting the myometrial activity in many women at term (Reiss et al, 1997). Two randomised trials which compared the effect of indomethacin and placebo in delaying delivery showed significant delay at 48 hours and at 7 -10 days.

Comparison with agonists show similar efficacy, but a better side effect profile (RCOG GUIDELINE 2002). However, their use is limited because of their effects in the fetus.

CALCIUM CHANNEL BLOCKERS

They are heterogeneous group of organic compounds that inhibit the influx of extracellular calcium across the cell membrane during inward calcium current of action potential. They block the voltage sensitive L type of calcium channels. They also inhibit the release of intracellular calcium from the sarcoplasmic reticulum. Thus they reduce the tone of smooth muscles, the commonly used drug Nifedipine is a potent inhibitor of myometrial contractions in non-pregnant, pregnant and post-partum uterus (Anderson et al, 1979)

- Consider nifedipine for tocolysis for women between 24⁺⁰ and 36 weeks of pregnancy who have intact membranes and are in suspected PTL

- If nifedipine is contraindicated, offer oxytocin receptor antagonists for tocolysis.

Treatment Regime:

The optimal dosing regimen of Nifedipine has not yet defined. Read and Wellby 1986, George et al 1997, showed that an initial dose of 30mg followed by 20mg 8th hourly for 3 days, reported a 75% successful tocolysis in 71% and 76% respectively. The tocolytic regimen given in Obstetrics and Gynaecology Clinics of North America (Andrienne Z et al) is loading dose 30 mg orally and maintenance dose of 10 -20 mg orally every 4-6 hrs.

In Clinical Obstetrics and Gynaecology 2000, Amy E et al found the following dosing regimens in various study protocols. Most administered a initial loading dose of 30mg of oral nifedipine followed by 10mg to 20mg dose every 4 to 6 hrs, upto a maximum dose of 160mg. Sublingual Nifedipine loading doses are no longer advised.

OXYTOCIN ANTAGONISTS (ATOSIBAN)

Oxytocin is fundamentally involved in the mechanism of labour. Oxytocin receptors are crucial for the onset of labor. There will be increase in myometrial oxytocin receptors in labor. The analogue competitively blocks the oxytocin receptors and inhibits preterm labor.

Atosiban acts only on myometrium/myoepithelial tissue (MOST UTEROSPECIFIC) Atosiban is given intravenously 6.75mg bolus over one minute followed by infusion at 18mg/hr for 3 hrs and then 6mg/hr for upto 45

hrs. Duration of treatment should not exceed 48 hrs and the dose should not exceed 330mg of atosiban. Side effects are nausea, chest pain, vomiting and dyspnoea. Compared to β agonist atosiban has similar efficacy but a better side effect profile.

Royal College of Obstetricians and Gynaecologists 2002, suggest that if tocolytics are administered, the first choice should be oxytocin antagonists or Nifedipine. But compared with other tocolytics atosiban therapy is costly.

NITRIC OXIDE DONORS (GLYCERYL TRINITRATE)

Nitric oxide is a potent endogenous hormone causing smooth muscle relaxation. The NO donors inhibit corticotrophin releasing hormone secretion which acts as a promoter of parturition. 10mg of Glyceryl Trinitrate patch is applied over the fundal region of maternal abdomen. If tocolysis is not achieved in one hour, another 10mg patch can be applied to a maximum dose of 20mg in 24 hours. Cochrane review (2000) by Duckitt K et al showed that nitroglycerine did not delay delivery or improve neonatal outcome when compare with placebo, no treatment or alternative tocolytics.

K⁺ CHANNEL OPENERS:

Diazoxide is a medication structurally related to thiazide diuretics that is used in treatment of hypertensive crisis. It inhibits contractility of smooth muscles there by rendering myometrial quiescence.

Dosage is 5mg/kg, given intravenously slowly in 15-30 minutes. The drug is diluted in half normal saline. It can also be given in boluses of 50 – 100 mg every 5 minutes. Side effects are hypotension, tachycardia, hyperglycemia and decreased uteroplacental blood flow secondary to maternal hypotension. The fetal side effects are hypoglycaemia and fetal distress secondary to maternal hypotension. Further evaluation of this newer group of tocolytic drugs is needed.

AIM OF THE STUDY

AIM OF THE STUDY

To evaluate the association of elevated cholesterol levels at 14 to 24 weeks gestation in uncomplicated pregnancy and preterm delivery. To associate the elevated levels of cholesterol as predictor of preterm delivery

Primary Objective:

To determine the association between elevated Serum Cholesterol in Pregnancy & Pre-term Births

Secondary Objective:

To determine the association between elevated Serum Cholesterol in Pregnancy & Pre-term Births

MATERIALS AND METHODS

MATERIALS AND METHODS

The study group includes 300 healthy pregnant women, fasting cholesterol levels during 14 to 24 weeks gestation who have come to antenatal check-up at the **Department of Obstetrics and Gynaecology, Madras Medical College**, during the period August 2016 to July 2017.

Only those patients we could follow up to term and planning delivery at IOG were included in the study. The biochemical investigations were done at the Institute of Biochemistry, MMC. The hospital ethical committee approved the study.

Out of 322 antenatal mothers selected on the basis of inclusion and exclusion criteria 22 were excluded during the study for various reasons. 4 developed PIH, 7 developed Gestational diabetes Mellitus, one case was a multiple pregnancy, 2 were abruption placenta, and 8 were lost to follow up.

INCLUSION CRITERIA:

Age = 17 – 35 years

Gestational age = from participants last menstrual period or modified by USG.

EXCLUSION CRITERIA:

- GDM
- Pregnancy included Hypertension
- Previous H/O preterm delivery

- Multiple pregnancy
- Hydramnios
- Cervical Incompetence
- Pre-existing medical disease
- Cardiovascular disorder
- Renal disorder
- Congenital anomalies of fetus
- Smoking
- Unknown last menstrual period

STUDY METHOD:

The design of the study prospective study. In all these antenatal mothers detailed history with special reference to diet and habits, followed by complete general and obstetric examination were done. The purpose of interrogation and investigation was explained to every patient and her informed consent obtained.

GENERAL EXAMINATION:

Height, weight, pulse, blood pressure, edema, anemia, cardiovascular, respiratory and central nervous system disorder were examined.

OBSTETRIC EXAMINATION: Per Abdomen Examination

PROCEDURE:

From all the antenatal mothers who were included in the study, blood sample was taken after overnight fasting.

Under strict aseptic precaution blood was obtained for other investigations by venepuncture.

Cholesterol between 14 to 24 weeks gestation were obtained after overnight fast. The patients were followed till delivery.

LABORATORY TESTS:

The blood samples for cholesterol were collected and analysed.

TOTAL CHOLESTEROL:

Serum total cholesterol were determined by automated enzymatic method using Burstein, Lopes – Vivella CHOD – PAP method. Chylomicrons are precipitated by adding phosphotungstic acid solution and magnesium ion to the sample. Centrifugation leaves HDL in supernatant fluid. The cholesterol content is determined enzymatically.

PROCEDURE:

200ml of sample with 500ml of precipitant fluid were mixed together and a precipitate is obtained by allowing it to stand for 10 minutes at room temperature. This is then centrifuged and 100 ml of the supernatant fluid is mixed with 1000ml of reagent solution. Phosphotungstic acid. The results are obtained by analysing the absorbance of sample and standard using semi auto analyser.

PRINCIPLE:

The formation of coloured phenazone compound with the reagent forms the basis of the test.

Sample of 10 ml and 1000ml of reagent are mixed and incubated for 5 minutes at 37C and absorbance of sample measured within 60 minutes.

Results were analysed in semi auto analyser (ERBAKEMP).

**OBSERVATION AND
ANALYSIS**

OBSERVATION AND ANALYSIS

Statistical Tools:

The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using Epidemiological Information Package (EPI 2010) developed by Centre for Disease Control, Atlanta.

Using this software range, frequencies, percentages, means, standard deviation, chi square and 'p' values were calculated. Kruskal Wallis chi-square test was used to test the significance of difference between quantitative variables and Yate's chi square test for qualitative variables and Yate's chi square test for qualitative variables. A 'p' value less than 0.05 is taken to denote significant relationship.

RESULTS

A. Profile of Cases Studied

Table 1. Age distribution

AGE_GROUP	Frequency	Percent
19-21 YEARS	120	40.0
22-24 YEARS	142	47.3
25-28 YEARS	38	12.7
Total	300	100.0

Descriptive Statistics

	N	Minimum	Maximum	Mean	Std. Deviation
AGE	300	19.00	28.00	22.13	2.00451

Majority of the women included in the study were 22 to 24 years old. The study group had mean age of 22.13 years, standard deviation of 2.004 years.

AGE DISTRIBUTION

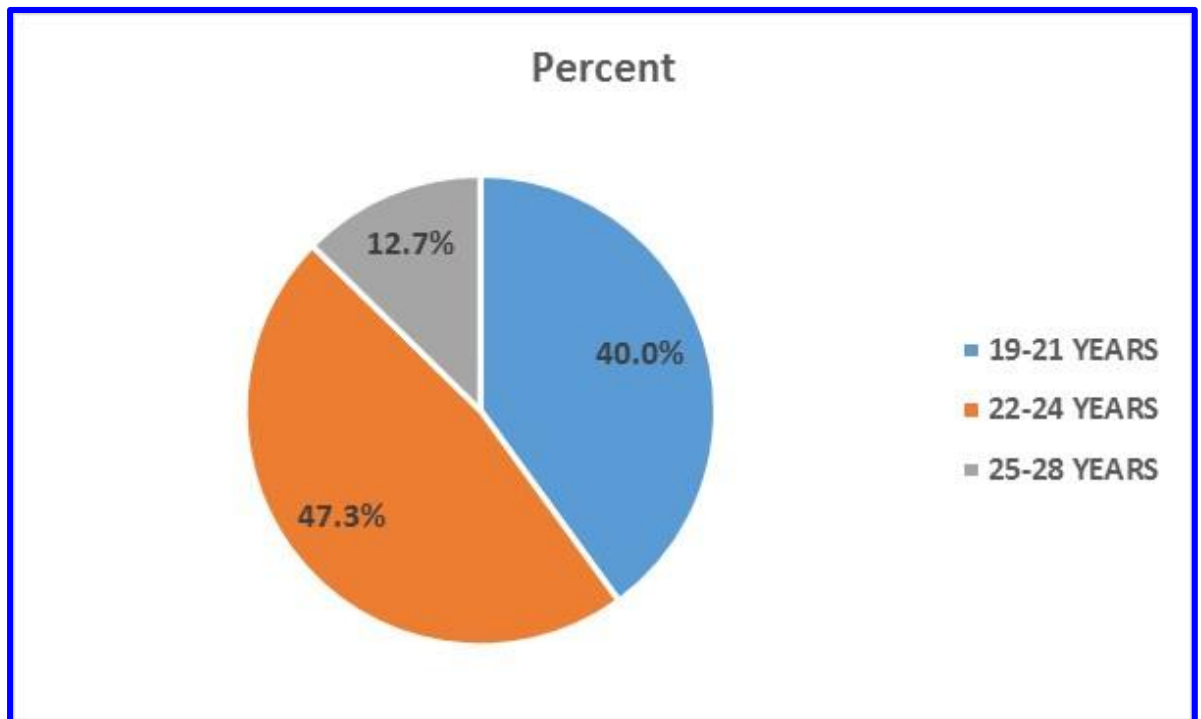


Table 2. Obstetric Code

OBSTETRIC_CODE	Frequency	Percent
PRIMI	140	46.7
G2PIL1	160	53.3
Total	300	100.0

46.7% of mothers included in the study were primis. Remaining mothers (53.3%) were second gravida mothers.

OBSTETRIC CODE

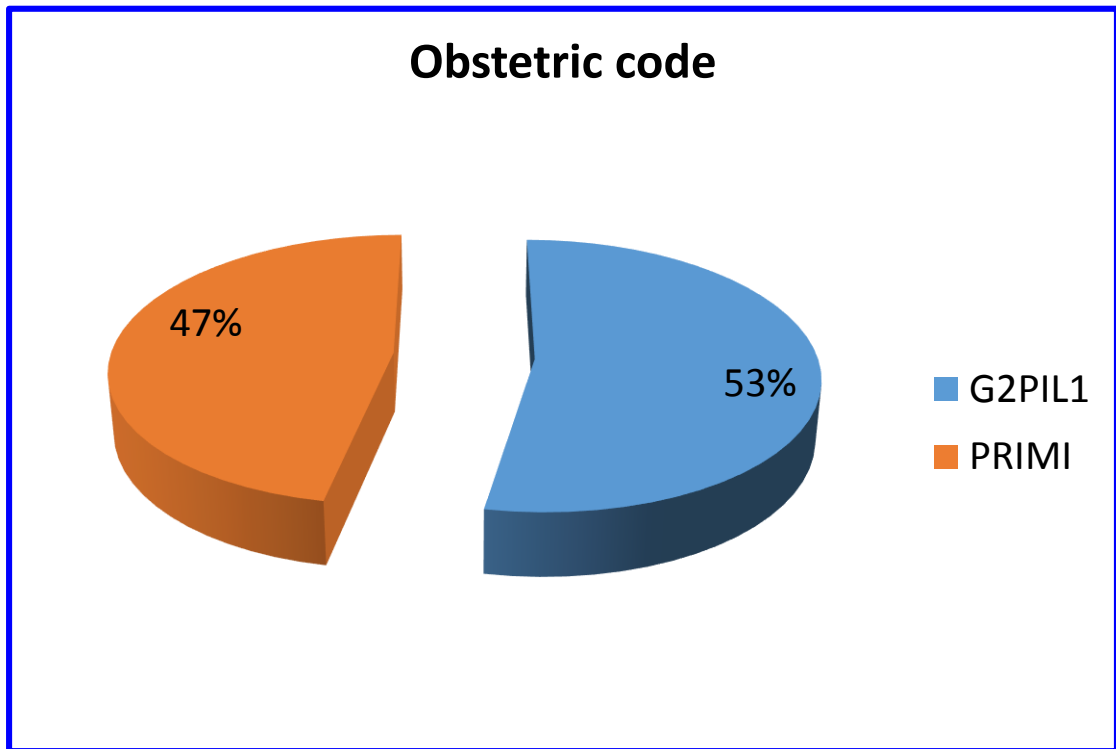


Table 3

Gestational age at which blood collected for analysis

BLOOD_COLLECTION_WEEKS	Frequency	Percent
16.00	2	0.7
18.00	72	24.0
20.00	75	25.0
22.00	78	26.0
24.00	73	24.3
Total	300	100.0

Serum cholesterol levels were assessed for 300 mothers during 16 to 24 weeks of gestation.

GESTATIONAL AGE

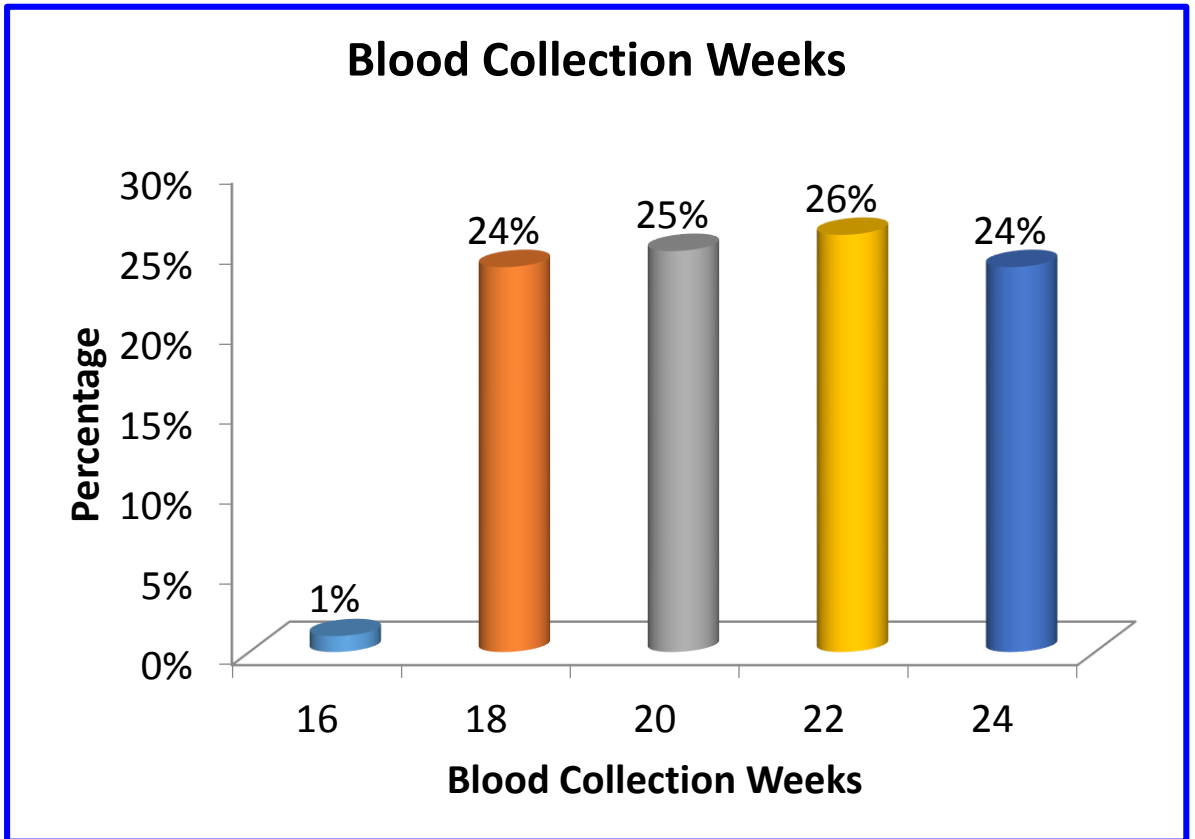


Table 4
Serum Cholesterol Level

Serum Cholesterol Level	Frequency	Percentage
Normal	281	93.7%
Abnormal	19	6.3%

About 19 mothers (6.3%) had abnormal cholesterol levels, and the study group had a mean serum cholesterol level of 266.2 mg/dl

SERUM CHOLESTEROL LEVEL

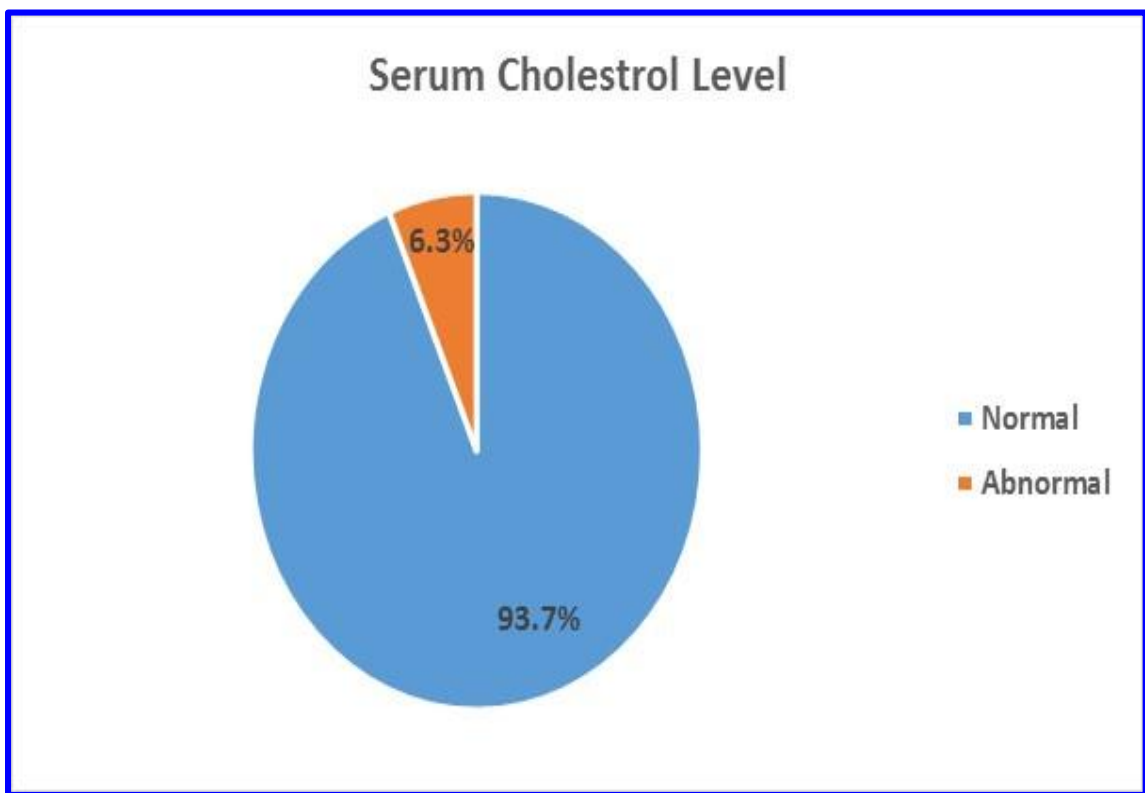


Table 5

Outcome of delivery - Preterm / Term

Outcome of delivery	Frequency	Percent
TERM	277	92.3
PRE TERM	23	7.7
Total	300	100.0

Out of the 300 mothers included in the study, 23 mothers (around 7.7%) had preterm delivery.

OUTCOME OF DELIVERY

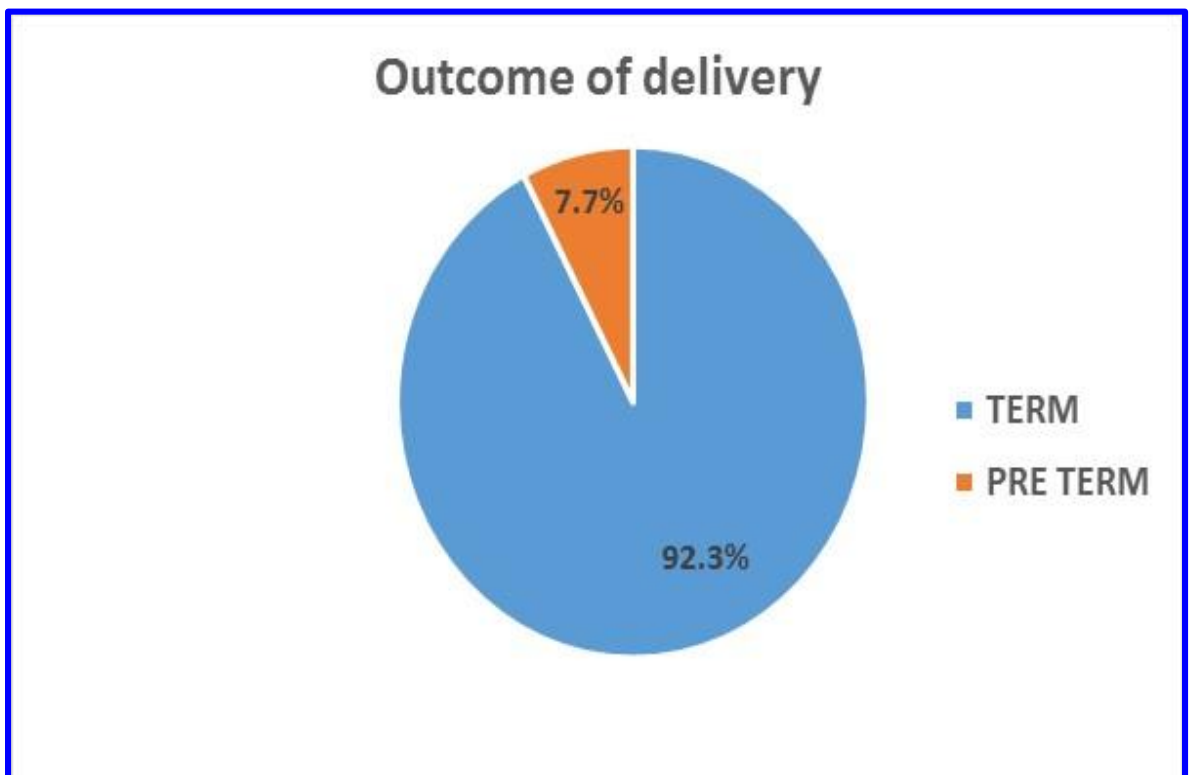


Table 6

Relation between cholesterol & other variables with outcome of delivery

Age of Mother and Outcome of delivery

			OUTCOME		Total
			PRE TERM	TERM	
AGE_GROUP	19-21 YEARS	Count	5	97	102
		% within OUTCOME	21.74%	35.02%	34.00%
	22-24 YEARS	Count	6	126	132
		% within OUTCOME	26.09%	45.49%	44.00%
	25-28 YEARS	Count	12	54	66
		% within OUTCOME	52.17%	19.49%	22.00%
Total		Count	23	277	300
		% within OUTCOME	100.0%	100.00%	100.00%

Comparison of Age group with outcome

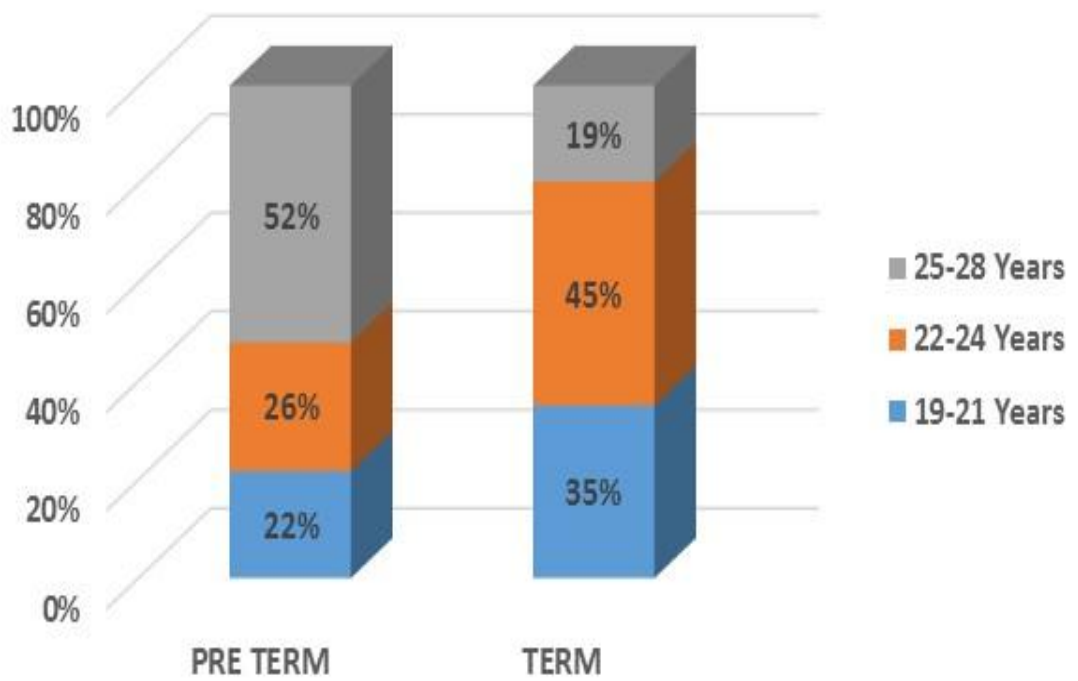


Table 7

Obstetric code and outcome of delivery						
			OUTCOME		Total	
			PRE TERM	TERM		
OBSTETRIC CODE	G2PIL1	Count	15	145	160	
		% within OUTCOME	65.2%	52.3%	53.3%	
	PRIMI	Count	8	132	140	
		% within OUTCOME	34.8%	47.7%	46.7%	
	Total		Count	23	277	300
			% within OUTCOME	100.0%	100.0%	100.0%

Pearson Chi-Square =1.413, P=0.234

The percentage of pre-term deliveries among PRIMIs and second Gravida did not have statistical significant difference (p = 0.234)

OBSTETRIC CODE & OUTCOME

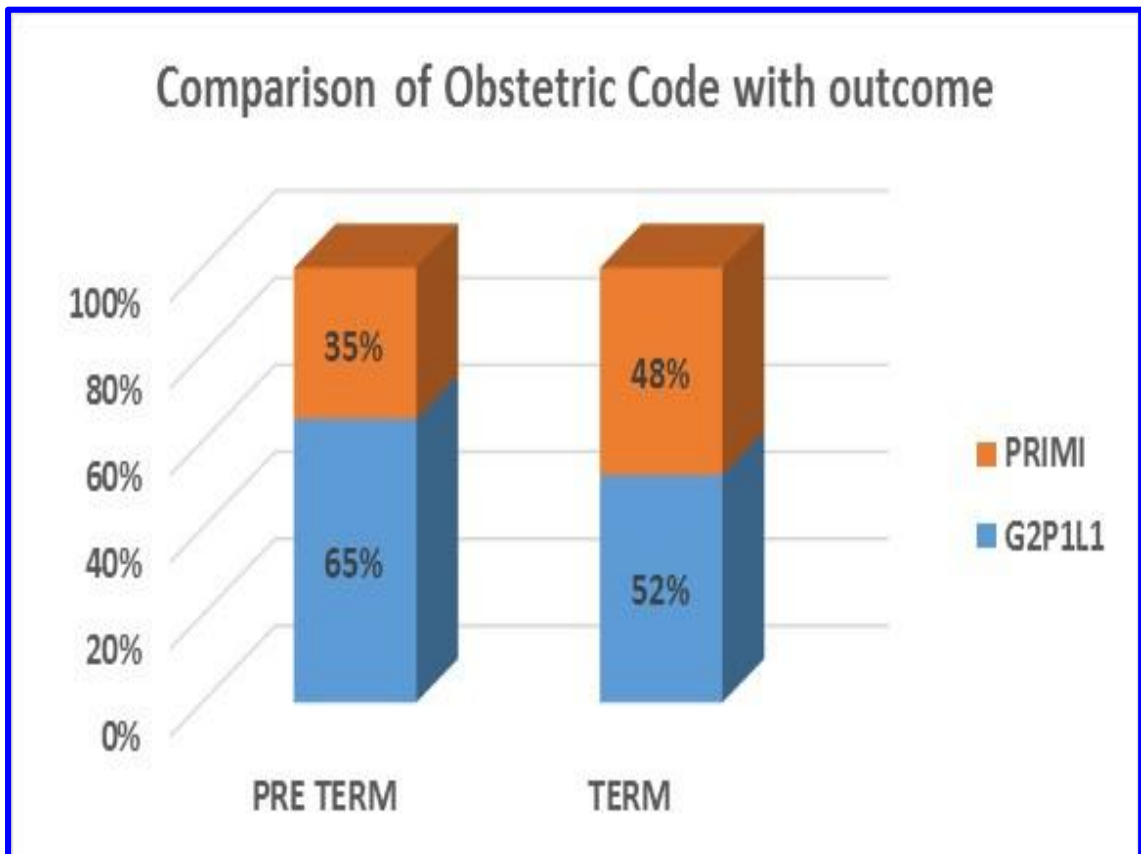


Table 8**Serum cholesterol and Outcome of delivery**

			OUTCOME		Total	
			TERM	PRETERM		
SC NORMAL	<200	Count	104	6	110	
		% within OUTCOME	94.5%	5.5%	100%	
	200-300	Count	166	5	171	
		% within OUTCOME	97.1%	2.9%	100%	
	ABOVE 300	Count	7	12	19	
		% within OUTCOME	36.8%	63.2%	100%	
	Total		Count	277	23	300
			% within OUTCOME	92.3%	7.7%	100.0%

Pearson Chi-Square =88.84** P<0.0001

Mothers with normal cholesterol values had 96.1% term delivery and around 3.9% had pre-term delivery, whereas mothers with abnormal cholesterol had 63.1% pre-term delivery. The difference is statistically significant (p < 0.0001)

SERUM CHOLESTEROL AND OUTCOME OF DELIVERY

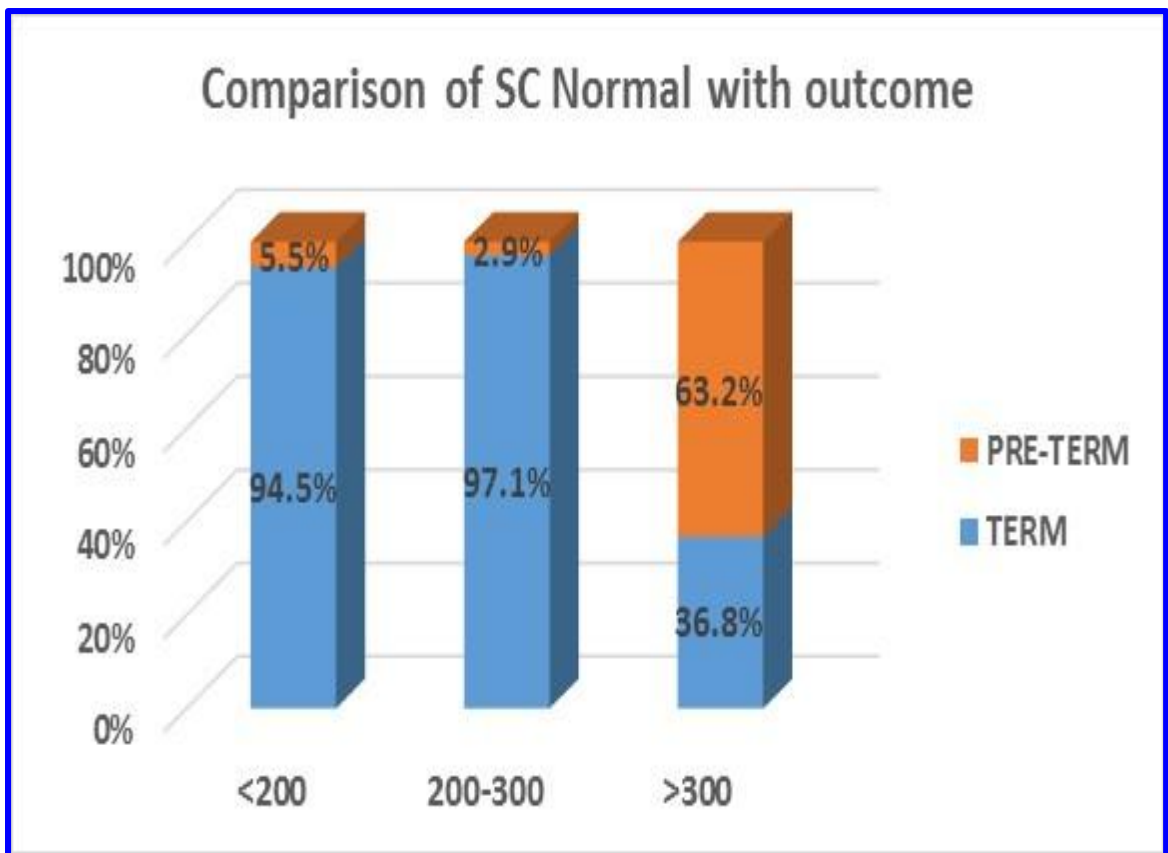


Table 9

Outcome of delivery vs Mode of delivery

Outcome of delivery		Mode of Delivery	
		Normal	LSCS
Term	Count	168	109
	%age	60.6%	39.4%
Pre-Term	Count	19	4
	%age	82.6%	17.4%

Pearson Chi-Square =4.36, P<0.05

Outcome of delivery vs Mode of delivery

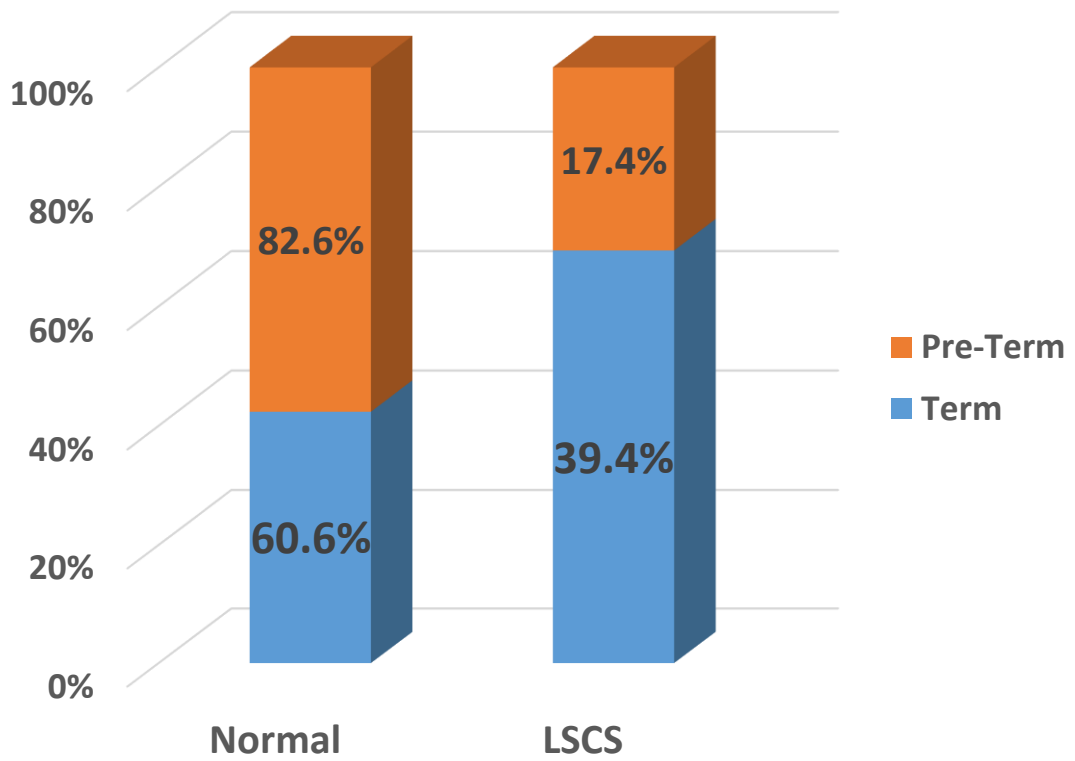


Table 10

Outcome of Delivery	Fetal Weight (in Kgs)		
	#	Mean	SD
TERM	277	2.7812	0.2105
PRETERM	23	1.9541	0.3869

The mean fetal weight of children delivered at term are significantly higher than that of pre-term children.

Outcome of delivery in terms of fetal weight

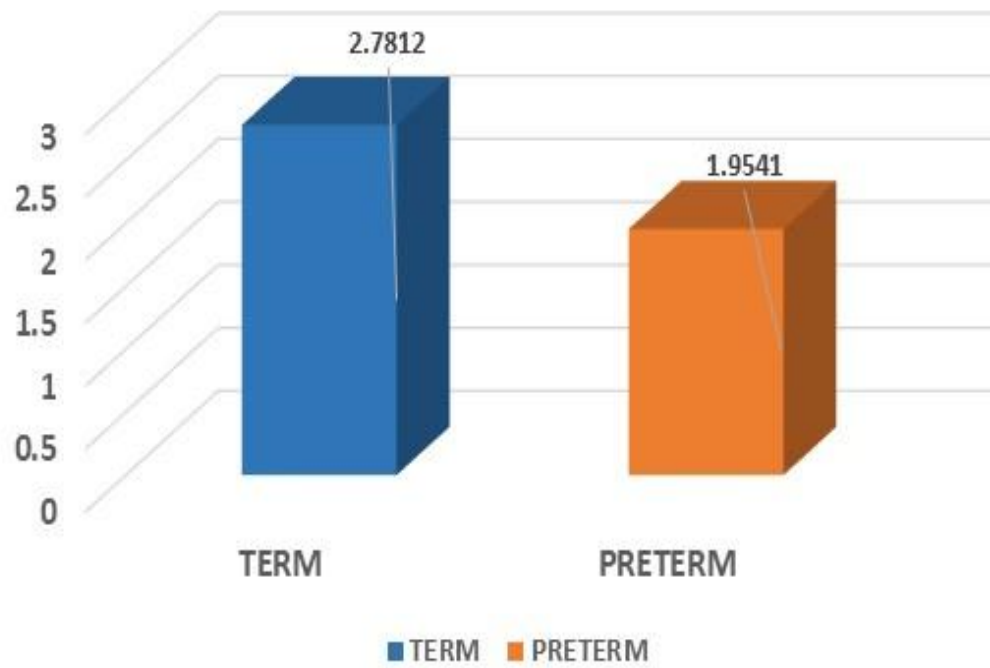


Table 11

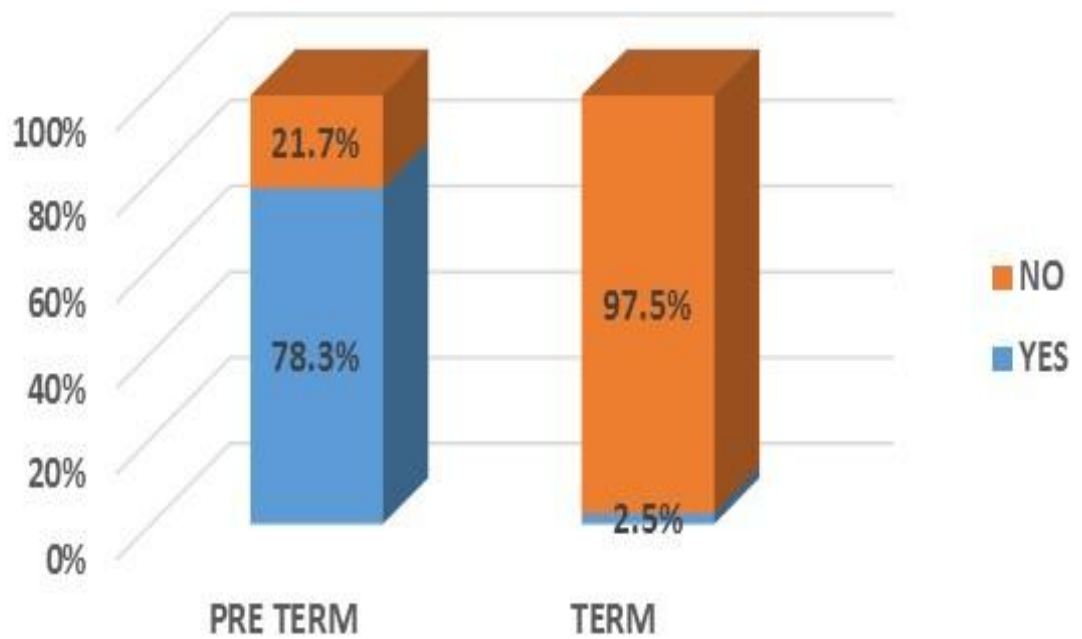
Outcome of delivery and NICU Admissions

			OUTCOME1		Total
			PRE TERM	TERM	
NICU_ ADMISSION	NO	Count	5	270	275
		% within OUTCOME1	21.7%	97.5%	91.7%
	YES	Count	18	7	25
		% within OUTCOME1	78.3%	2.5%	8.3%
Total		Count	23	277	300
		% within OUTCOME1	100.0%	100.0%	100.0%

Person Chi-Square =159.45** P<0.0001

The outcome of deliveries had significant association with NICU admissions (p< 0.0001)

Comparison of NICU Admission with outcome



DISCUSSION

DISCUSSION

Preterm delivery is defined as by WHO, birth occurring prior to 37 completed weeks gestation. The incidence in India is 10 – 12% and developed countries is 5% to 10%.

Serum cholesterol levels increase during pregnancy & is necessary uteroplacental vascularisation, placental transport functions. Hyperlipidaemia is also regarded as an instigator of inflammation and stress, which is a significant factor in preterm birth.

In this study, 300 antenatal mothers with singleton pregnancy between the gestational age of 14-24 weeks were enrolled. After detailed history taking and physical examination, fasting maternal serum cholesterol levels were estimated and the patients were followed till delivery (preterm / term).

The incidence of preterm delivery, mode of delivery, gestational age at delivery was noted; to study the linear trend and to estimate if there is a dose response relationship between increasing cholesterol values and gestational age at delivery.

American medical journal OBSTETRICS AND GYNAECOLOGY 2007, December 197 (6) 610. Cator jim, Bodnar studied in early pregnancy lipid concentration and spontaneous preterm birth.

In that case control study of women with spontaneous preterm birth, cholesterol, high density lipoprotein, low density lipoprotein, triglycerides were evaluated. Lipid concentration and gestational changes as well as risk

for preterm birth were evaluated in women who delivered < 34 weeks (n=23) > or = 34 (n=67) and > or = 37 weeks (n=199)

High cholesterol, triglycerides < or = 15 weeks were associated with a 2.8 fold and 2.0 fold (1.0 – 3.9) increased risk for preterm birth < 34 weeks and > or 34 < 37, respectively. Overweight female who delivered < 34 weeks had particularly elevated early pregnancy concentrations of cholesterol and low density lipoprotein. . There was a reduced triglycerides response in the first half of pregnancy among female who delivered < 34 weeks. Results indicate that the presence of dyslipidemia in female results spontaneous preterm birth our study us similar to it and our result are statistically significant.

In current study out of 300 Antenatal mothers 190 are primi and 210 are second gravida. Blood collected at 14 – 24 weeks, respectively in 300 mothers.

Catov et al., however, suggest that early sPTD (<34 weeks) is associated with both high and low pre-pregnancy TC [18]. This same finding is supported by Edison et al. during second trimester pregnancy and by Mudd et al. only for high TC level [13, 25].

Another study, Adegbesan-Omilabu Maymunah,¹ Okunade Kehinde (2012) evaluated Hypercholesterolaemia in pregnancy as a predictor of adverse pregnancy outcome).

The incidences of the two adverse pregnancy outcomes examined in the study (preterm births and low birth weight (LBW) in term neonates) were 8.0% and 14.4% respectively. Preterm birth was 6.89-times more common in mothers with high cholesterol than in control mothers with normal total cholesterol level (38.5% versus 5.4%, $P=0.029$) while LBW was 7.99-times more common in mothers with high total maternal cholesterol than in mothers with normal cholesterol (87.5% versus 10.5%, $P=0.019$).

Inflammation and dyslipidemia related to risk of spontaneous preterm birth.

Serum Cholesterol and Outcome of Delivery

Sr, Cholesterol	No. of cases	Outcome of delivery			
		Good		Preterm	
		No	%	No	%
Normal	281	270	92.6	11	8.4
Abnormal	19	7	36.8	12	63.2
<u>Sr.Cholesterol</u>					
Mean		246.4			
SD		42.86			
‘p’		< 0.0001			

All the mothers with normal cholesterol values had 92.6 % good outcome whereas of 63.2 % mothers with abnormal cholesterol values had preterm deliveries. This difference is statistically significant ($p=0.0001$).

A study by Catov JM¹, Bodnar LM, Ness RB, Barron SJ, Roberts JM. In the Department of Epidemiology, University of Pittsburgh, Pittsburgh, (PA 15261, USA. jmcst43@pitt.edu)

The authors considered that inflammation in women with spontaneous preterm birth (sPTB) might be related to their metabolic profile, such as lipids, and tested this in a nested case-control study from the Pregnancy Exposures and Preeclampsia Prevention Study (1997-2001). Cases were women with sPTB at 34-<37 weeks ($n = 76$) or < 34 weeks ($n = 33$). Controls were randomly selected women with term births ($n = 228$). Early pregnancy inflammation (C-reactive protein: $> \text{ or } = 8$ microg/ml) and dyslipidemia (cholesterol: > 230 mg/dl or triglycerides: > 140 mg/dl) were evaluated in serum collected at < 21 weeks. Polytomous logistic regression was used to estimate the effects of dyslipidemia on the risk of sPTB subtypes. After adjustment for race, body mass index, periconceptional vitamin use, and gestational age at sampling, early pregnancy inflammation (odds ratio = 2.9, 95% confidence interval (CI): 1.1, 7.2) and dyslipidemia (odds ratio = 2.0, 95% CI: 1.0, 4.2) were independently associated with sPTB at 34-<37 weeks. The presence of both conditions increased risk of sPTB at < 34 weeks 6.4-fold (95%)

These data may have important clinical significance because they provide a possible link between preterm delivery and high lipid level.

Finding by Catov and coworkers²⁶ showed that an elevation in maternal cholesterol level early in gestation was associated with an increased risk of preterm delivery. This was corroborated by the finding from this present cohort study where we reported an elevated risk for preterm birth among mothers with high maternal cholesterol.

There are several limitations to this study. Their demographic, socioeconomic or medical characteristics could be small. Our results from the specific ethnic groups may not be generalizable to other population. Socioeconomic factor such as dietary intake may affect triglycerides and cholesterol concentration and risk.

Finally, elevated maternal cholesterol levels may play a role in the mechanism of underlying preterm delivery or may simply be a marker for risk of preterm delivery.

SUMMARY

SUMMARY

- This prospective study group included 300 uncomplicated pregnant women of 14 to 24 weeks of gestation during the period August 2016 to July 2017 at Department of obstetrics and Gynaecology, MMC
- From all the antenatal mothers included in the study the blood sample of serum cholesterol was taken after overnight fasting and were followed till delivery.
- 47% of this study population were in the age group of 22 – 24 years. (mean – 22.3)
- 46.7% were primis, 53.3% were second gravidas.
- In this study, we have not seen much influence of age and parity to the outcome of delivery.
- 93.7% mothers had normal cholesterol values, 6.3% had elevated cholesterol values.
- The study group with normal cholesterol values had good outcome were as 63.2% study group with elevated cholesterol values had preterm delivery is statistically significant ($p = 0.0001$)
- The mean fetal weight delivered at term are significantly higher than those of preterm
- Among the preterm deliveries, 78.3% babies were admitted in NICU, which is statistically significant ($p < 0.0001$)
- In the current study cholesterol levels was found to be simple marker for preterm delivery

CONCLUSION

CONCLUSION

Serum Cholesterol levels was evaluated in 300 antenatal mothers at 14 to 24 weeks of gestation with fasting cholesterol levels.

The findings of the present study showed that serum cholesterol levels were found to be elevated in patients who have gone in for preterm labour than those gone for term pregnancy.

Hence, Serum cholesterol levels has been found to be useful simple marker for preterm delivery.

This observation helps us to describe a generic framework for combining this screening information for designing a prophylactic intervention in future.

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delivery.

ANNEXURES

PROFORMA

NAME		Age
IP.No		Unit
Educational status:		
Income	Occupation	SE Status
Gravida		
Para		Last Menstrual Period
(LMP)		
Live		Expected date of
delivery		
(EDD)		
Abortion		Corrected EDD
(C.EDD)		
Menstrual Cycle		
Height	Weight	BMI
Booked / Unbooked (UB)		
Immunized / Not		
DOA (Date of Admission)		
Duration of Hospital stay		
DOD (Date of Discharge)		
Period of gestation		

Present Complaints

Lower abdominal pain

Dull low backache

Vaginal discharge

Fluid leaking per vaginum

Fever

UTI (Urinary Tract Infection)

URI (Upper Respiratory Tract Infection)

Bleeding

Obstetric History

Trimester

Hyperemesis

Exanthematous fever

Bleeding

Radiation exposure

Medication

Pain abdomen

II. Trimester

Date of Quickening

Bleeding per vaginum

History of (H/O) PIH

H/O GDM (Gestational Diabetes Mellitus)

III. Trimester

Bleeding per vaginum

UTI

Cervico vaginal infection

Coitus

Diabetes

Hypertension

Fever

Trauma

Past Obstetric History

Previous child birth

H/O abortion

H/O Preterm Labour

H/O babies with congenital anomalies

Past Medical History

Tuberculosis

Bronchial Asthma

STD (Sexually Transmitted Diseases)

Jaundice

Heart Disease

Diabetes mellitus

Epilepsy

Renal Disease

General Examination

Temperature (T)

Pallor

Pedal Edema

PR BP RR

RS

CVS

Obstetric Examination - Per abdomen

Investigations

Urine analysis

Urine culture sensitivity

Complete Blood Count

Blood urea

Sugar

S.Creatinine

S.Electrolytes

HIV, HBsag

VDRL

ECG

CTG

USG Abdomen

Fetal Outcome

ABBREVIATIONS

FHR	: Fetal Heart Rate
FLM	: Fetal Lung Maturity
GA	: Gestational Age
GBS	: Group B Streptococcus
HMD	: Hyaline Membrane Disease
IVH	: Intra Ventricular haemorrhage
LN	: Labour Natural
LMP	: Last Menstrual Period
LSCS	: Lower Segment Caesarean Section
MMP	: Matrix MetalloProteinaeses
NEC	: Nectrotising Enterocolitis
NICU	: Neonatal Intensive Care Unit
PROM	: Premature Rupture Of Membranes
PPROM	: Preterm Premature Rupture Of Membranes
RDS	: Respiratory Distress Syndrome
ROM	: Rupture of Membranes
TIMP	:Tissue Inhibitor MetalloProteinaeses
USG	: Ultra Sono Gram

INFORMATION SHEET

- We are conducting a study on “MEASUREMENT OF SERUM CHOLESTEROL LEVELS AS A PREDICTOR OF PRETERM DELIVERY” over a period of 1 year which is very valuable to us.
- The purpose of this study was to evaluate whether serum cholesterol associated with preterm labor.
- The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.
- Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.
- The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of investigator

Signature of participant

Date:

சுய ஒப்புதல் படிவம்

ஆய்வு தலைப்பு :

அதிக கொழுப்புச்சத்தின் அளவைக்கொண்டு குறைப்பிரசவத்தை
கண்டறிய ஆய்வு மேற்கொள்ளுதல்

பெயர் :

வயது :

தேதி :

வெளிநோயாளி எண்:

..... என்பவராகிய நான் இந்த ஆய்வின் விவரங்களும் அதன்
நோக்கங்களும் முழுமையாக அறிந்து கொண்டேன். எனது சந்தேங்கள் அனைத்திற்கும் தகுந்த
விளக்கம் அளிக்கப்பட்டது. இந்த ஆய்வில் முழு சுதந்திரத்துடன் மற்றும் சுயநினைவுடன் பங்கு
கொள்ள சம்மதிக்கிறேன்.

எனக்கு விளக்கப்பட்ட விஷயங்களை நான் புரிந்து கொண்டு நான் எனது சம்மதத்தைத்
தெரிவிக்கிறேன். இச்சுய ஒப்புதல் படிவத்தை பற்றி எனக்கு விளக்கப்பட்டது.

இந்த ஆய்வினை பற்றிய அனைத்து தகவல்களும் எனக்கு தெரிவிக்கப்பட்டது. இந்த
ஆய்வில் எனது உரிமை மற்றும் பங்கினை பற்றி அறிந்து கொண்டேன்.

இந்த ஆய்வில் பிறரின் நிர்பந்தமின்றி என் சொந்த விருப்பத்தின் பேரில் தான் பங்கு
பெறுகிறேன் மற்றும் நான் இந்த ஆராய்ச்சியிலிருந்து எந்நேரமும் பின் வாங்கலாம் என்பதையும்
அதனால் எந்த பாதிப்பும் ஏற்படாது என்பதையும் நான் புரிந்து கொண்டேன்.

இந்த ஆய்வில் கலந்து கொள்வதன் மூலம் என்னிடம் பெறப்படும் தகவலை ஆய்வாளர்
இன்ஸ்டிடியூசனல் எத்திக்ஸ் கமிட்டியினரிடமோ, அரசு நிறுவனத்திடமோ தேவைப்பட்டால்
பகிர்ந்து கொள்ளலாம் என் சம்மதிக்கிறேன்.

இந்த ஆய்வின் முடிவுகளை வெளியிடும்போது எனது பெயரோ, அடையாளமோ
வெளியிடப்படாது என அறிந்து கொண்டேன். இந்த ஆய்வின் விவரங்களைக் கொண்ட தகவல்
தாளைப் பெற்றுக் கொண்டேன். இந்த ஆய்விற்காக இரத்தப் பரிசோதனை செய்துகொள்ள
சம்மதம் தெரிவிக்கின்றேன்.

இந்த ஆய்வில் பங்கேற்கும் பொழுது ஏதேனும் சந்தேகம் ஏற்பட்டால், உடனே ஆய்வாளரை
தொடர்பு கொள்ள வேண்டும் என அறிந்து கொண்டேன்.

இச்சுய ஒப்புதல் படிவத்தில் கையெழுத்திடுவதன் மூலம் இதிலுள்ள அனைத்து
விஷயங்களும் எனக்கு தெளிவாக விளக்கப்பட்டது என்று தெரிவிக்கிறேன் என்று புரிந்து
கொண்டேன். இச்சுய ஒப்புதல் படிவத்தின் ஒரு நகல் எனக்கு கொடுக்கப்படும் என்று தெரிந்து
கொண்டேன்.

பங்கேற்பாளர் /பாதுகாவலர் கையொப்பம்

தேதி :

ஆய்வாளர் கையொப்பம்

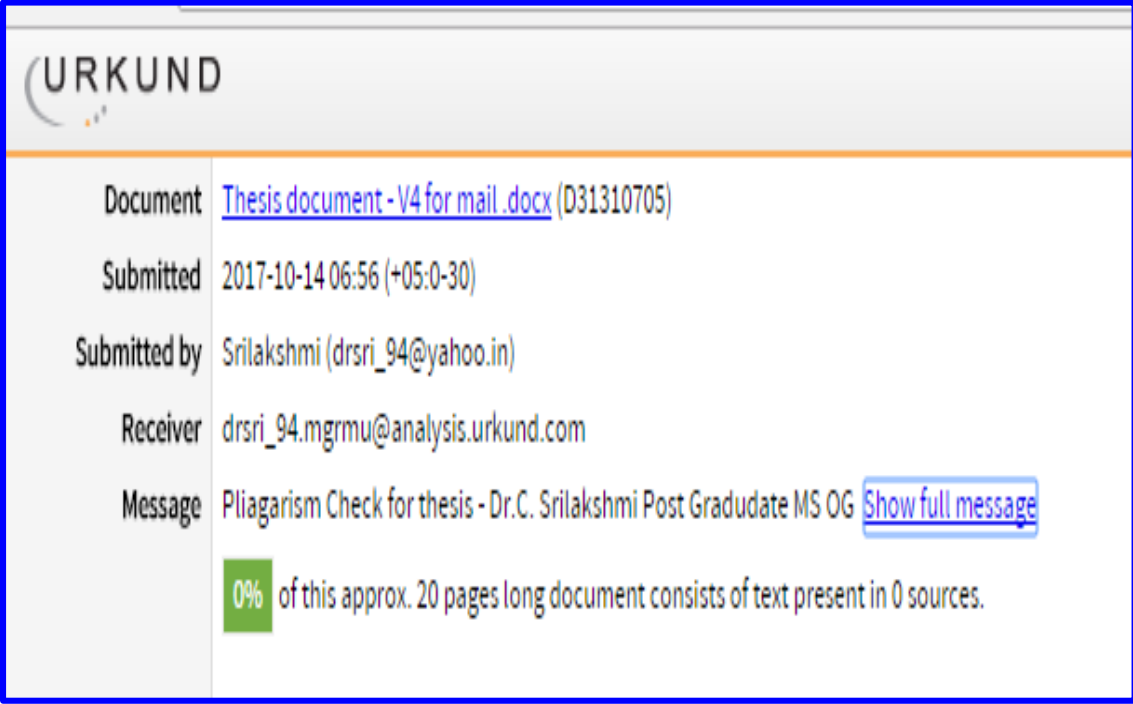
தேதி :

PLAGIARISM CERTIFICATE

This is to certify that this dissertation work titled **“MEASUREMENT OF SERUM CHOLESTEROL LEVELS AS A PREDICTOR OF PRETERM DELIVERY”** of the candidate Dr.C. SRILAKSHMI with registration number 221516003 for the award M.S in the branch of OBSTETRICS & GYNECOLOGY. 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 result shows zero percentage of plagiarism in the dissertation.

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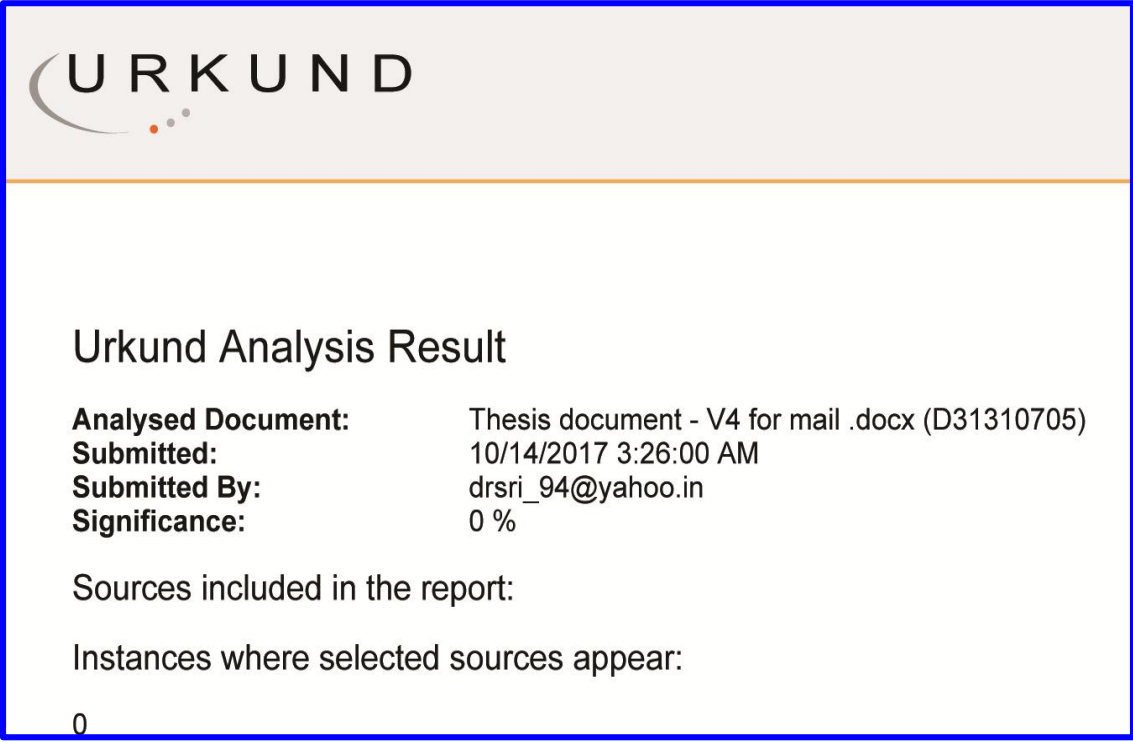
PLAGIARISM SCREEN SHOT



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Submitted	2017-10-14 06:56 (+05:0-30)
Submitted by	Srilakshmi (drsri_94@yahoo.in)
Receiver	drsri_94.mgrmu@analysis.orkund.com
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Urkund Analysis Result

Analysed Document:	Thesis document - V4 for mail .docx (D31310705)
Submitted:	10/14/2017 3:26:00 AM
Submitted By:	drsri_94@yahoo.in
Significance:	0 %

Sources included in the report:
0

Instances where selected sources appear:
0

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

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

CERTIFICATE OF APPROVAL

To
Dr. C.Srilakshmi M.B.B.S
First Year Post Graduate in M.S Obstetrics and Gynecology.
Institute of Obstetrics and Gynecology
Egmore Maternity Hospital
Madras Medical College, Chennai

Dear ,

The Institutional Ethics Committee has considered your request and approved your study titled **"MEASUREMENT OF SERUM CHOLESTEROL LEVELS AS A PREDICTOR OF PRETERM DELIVERY " NO.11092016** .

The following members of Ethics Committee were present in the meeting hold on **06.09.2016** conducted at Madras Medical College, Chennai 3

- | | |
|--|--------------------|
| 1. Prof. C. Rajendran, MD. | Chairperson |
| 2. Prof. Dr. M.K. Muralidharan, M.S, M.Ch., MMC ,Ch-3 | Deputy Chairperson |
| 3. Prof. Sudha Seshayyan, MD., Vice Principal, MMC.Ch- 3. | Member Secretary |
| 4. Prof. B.Vasanthi,MD.,Prof of Pharmacology, MMC, | Member |
| 5. Prof. P.Raghumani.MS., Professor of Surgery, Inst. of surgery | Member |
| 6. Prof. R.Padmavathy,MD., Professor, Inst.of Pathology, MMC,Ch | Member |
| 7. Tmt.J.Rajalakshmi, Junior Administrative Officer,MMC,Ch | Layperson |
| 8. Thiru.S.Govindasamy., B.A.B.L., High Court, Chennai-1 | Lawyer |
| 9. Tmt.ArnoldSaulina, MA., MSW., | Social Scientist |

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**

MASTER CHART

S.NO	NAME	AGE	OBSTETRIC CODE	LMP	EDD	BLOOD COLLECTION WEEKS	S.CHOLESTEROL	OUTCOME	MODE OF DELIVERY	FETAL WEIGHT (KG)	NICU ADMISSION
1	SUBHA	24	PRIMI	12.04.16	19.01.17	24	228	TERM	LSCS	2.9	NO
2	HEMAVATHI	22	PRIMI	08.03.16	15.12.16	18	248	TERM	NORMAL	2.8	NO
3	ANITHA	26	G2P1L1	21.05.16	28.02.17	22	285	TERM	LSCS	3.2	NO
4	GOMATHI	23	G2P1L1	26.03.16	02.01.17	16	189	TERM	LSCS	2.6	NO
5	MYTHILI	25	PRIMI	15.04.16	22.01.17	20	233	TERM	NORMAL	2.8	NO
6	LAKSHMI	23	G2P1L1	09.05.16	16.02.17	18	215	TERM	NORMAL	2.7	NO
7	FARIDHA	24	G2P1L1	18.03.16	25.12.16	22	249	TERM	LSCS	3	NO
8	KANIMOZHI	26	PRIMI	22.06.16	29.03.17	20	192	TERM	NORMAL	2.8	NO
9	USHA	23	PRIMI	07.04.16	14.01.17	18	266	TERM	NORMAL	2.6	NO
10	NARAYANI	27	PRIMI	12.05.16	19.02.17	22	276	TERM	LSCS	3.1	NO
11	SUJI	28	G2P1L1	03.04.16	10.01.17	16	168	TERM	NORMAL	2.8	NO
12	RADHA	24	G2P1L1	27.05.16	03.03.17	20	234	TERM	LSCS	2.5	NO
13	SUSEELA	23	G2P1L1	19.05.16	26.02.17	20	206	TERM	NORMAL	2.7	NO
14	SATHYA	21	G2P1L1	02.06.16	09.03.17	18	249	TERM	LSCS	2.6	NO
15	SARANYA	25	PRIMI	07.05.16	14.02.17	22	312	TERM	LSCS	2.9	NO
16	AKILA	27	G2P1L1	09.06.16	16.03.17	24	186	TERM	LSCS	2.5	NO
17	UMA	22	G2P1L1	29.04.16	06.02.17	18	273	TERM	NORMAL	2.8	NO
18	VIJAYA	25	PRIMI	14.05.16	21.02.17	24	201	TERM	LSCS	2.4	NO
19	SAINA	21	PRIMI	12.06.16	19.04.17	22	238	TERM	LSCS	3.2	NO
20	DIVYA	24	G2P1L1	22.06.16	29.03.17	20	177	TERM	LSCS	2.9	NO
21	VASANTHI	21	PRIMI	12.05.16	19.02.17	22	193	TERM	NORMAL	2.6	NO
22	ESWARI	20	PRIMI	09.06.16	16.03.17	24	254	TERM	NORMAL	2.8	NO
23	RENU	20	G2P1L1	16.06.16	23.03.17	18	251	TERM	LSCS	2.5	NO
24	SHALIMA	23	PRIMI	29.05.16	05.03.17	18	214	PRETERM33	NORMAL	2.8	YES
25	DEEPA	24	G2P1L1	23.06.16	30.03.17	20	276	TERM	NORMAL	2.6	NO
26	RANGITHA	21	G2P1L1	02.07.16	09.04.17	22	309	TERM	LSCS	2.6	NO
27	SUGANDHI	22	PRIMI	03.06.16	10.03.17	24	289	TERM	LSCS	2.7	NO
28	BHAVANI	23	PRIMI	20.06.16	27.03.17	22	230	TERM	NORMAL	2.7	NO
29	GOWRI	25	PRIMI	12.06.16	12.03.17	18	189	TERM	NORMAL	1.9	YES
30	VANITHA	20	G2P1L1	05.06.16	12.03.17	20	219	TERM	NORMAL	2.8	NO
31	SOWMIYA	21	PRIMI	10.06.16	17.03.17	24	243	TERM	NORMAL	2.8	NO
32	SUNDHARI	23	PRIMI	04.07.16	11.04.17	22	239	TERM	NORMAL	2.8	NO
33	RAMYA	20	G2P1L1	18.07.16	25.04.17	24	217	TERM	LSCS	2.9	NO
34	MOHANA	20	G2P1L1	15.07.16	22.04.17	18	176	PRETERM32	NORMAL	1.8	YES
35	HEMAVATHI	21	G2P1L1	02.08.16	09.05.17	20	219	TERM	NORMAL	2.8	NO
36	THEENA	22	G2P1L1	13.06.16	20.03.17	22	256	TERM	LSCS	3.1	NO
37	JERINA	23	PRIMI	09.07.16	16.04.17	18	230	TERM	NORMAL	2.8	NO
38	MANGALAM	21	G2P1L1	25.06.16	02.04.17	22	267	TERM	NORMAL	2.8	NO
39	INDHIRA	23	G2P1L1	20.05.16	27.02.17	20	276	PRETERM32	NORMAL	1.8	NO
40	FARITHABANU	24	PRIMI	01.07.16	08.04.17	24	248	TERM	NORMAL	2.8	YES
41	RASATHI	25	PRIMI	04.08.16	11.05.17	20	298	TERM	NORMAL	2.8	NO
42	THASEEN	20	G2P1L1	18.06.16	25.03.17	22	213	TERM	NORMAL	2.8	NO
43	SANTHA	20	PRIMI	06.07.16	13.04.17	24	256	TERM	NORMAL	2.7	NO

S.NO	NAME	AGE	OBSTETRIC CODE	LMP	EDD	BLOOD COLLECTION WEEKS	S.CHOLESTEROL	OUTCOME	MODE OF DELIVERY	FETAL WEIGHT (KG)	NICU ADMISSION
44	MARY	21	G2PIL1	13.07.16	20.04.17	20	247	TERM	LSCS	2.7	NO
45	ROHINI	22	G2PIL1	15.08.16	22.05.17	18	209	TERM	NORMAL	2.7	NO
46	REKHA	22	PRIMI	18.08.16	25.05.17	22	354	PRETERM34	NORMAL	2.2	YES
47	REHIMA	23	G2PIL1	21.06.16	28.03.17	24	254	TERM	NORMAL	2.8	NO
48	SHARMILA	23	PRIMI	30.07.16	10.04.17	20	198	TERM	NORMAL	2.8	NO
49	SHABEBA	24	G2PIL1	08.07.16	15.04.17	18	227	TERM	NORMAL	2.7	NO
50	FATHIMA	25	G2PIL1	10.07.16	17.04.17	22	286	PRETERM36	LSCS	2.4	YES
51	JANANI	19	G2PIL1	26.06.16	03.04.17	24	233	TERM	NORMAL	2.8	NO
52	RAJI	19	PRIMI	11.07.16	18.04.17	20	256	TERM	LSCS	2.7	NO
53	MURUGAMMA	20	G2PIL1	29.05.16	05.03.17	18	176	PRETERM34	NORMAL	1.8	YES
54	RASAMMA	20	PRIMI	30.06.16	07.04.17	24	289	TERM	LSCS	3.125	NO
55	PONMANI	21	PRIMI	22.06.16	29.03.17	22	264	TERM	NORMAL	2.8	NO
56	PUSHPA	21	G2PIL1	18.07.16	25.04.17	18	182	PRETERM33	NORMAL	1.3	YES
57	THILAGAVATHI	23	G2PIL1	31.05.16	07.03.17	20	178	TERM	NORMAL	2.8	NO
58	REKHA	23	G2PIL1	14.06.16	21.03.17	22	402	TERM	NORMAL	2.8	NO
59	PONKODI	26	G2PIL1	23.05.16	30.03.17	24	343	TERM	NORMAL	1.8	YES
60	SARANYA	21	G2PIL1	12.05.16	17.02.17	18	249	TERM	NORMAL	2.8	NO
61	JOTHIKA	22	G2PIL1	16.07.16	23.04.17	18	264	TERM	NORMAL	2.8	NO
62	LAVANYA	23	PRIMI	19.06.16	26.03.17	24	230	TERM	NORMAL	2.8	NO
63	MUNIYAMMAL	24	PRIMI	25.06.16	02.03.17	22	276	TERM	NORMAL	1.9	YES
64	RANI	24	G2PIL1	06.07.16	13.04.17	20	321	TERM	NORMAL	2.7	NO
65	REVATHI	20	G2PIL1	15.07.16	22.04.17	24	243	PRETERM34	LSCS	1.9	YES
66	JENITHA	20	PRIMI	03.08.16	10.05.17	22	237	PRETERM33	NORMAL	1.3	YES
67	PONGAVANAM	21	G2PIL1	10.08.16	17.05.17	18	234	TERM	LSCS	2.9	YES
68	MUMTAJ	21	PRIMI	24.07.16	01.05.17	20	276	TERM	LSCS	2.9	NO
69	SHARMILA	23	PRIMI	11.08.16	18.05.17	18	278	TERM	NORMAL	1.3	YES
70	PECHI	19	G2PIL1	05.08.16	12.05.17	24	210	TERM	NORMAL	2.8	NO
71	ELAKIYA	19	G2PIL1	10.07.16	17.04.17	22	240	PRETERM35	NORMAL	2.3	YES
72	BARANI	24	PRIMI	12.08.16	19.05.17	22	267	TERM	NORMAL	2.8	NO
73	TAMILSELVI	24	G2PIL1	16.06.16	23.03.17	18	245	PRETERM35	NORMAL	2.3	YES
74	SAGAYAM	25	G2PIL1	06.06.16	13.03.17	24	256	TERM	LSCS	3	NO
75	JASMINE	24	G2PIL1	19.07.16	26.04.17	20	287	TERM	NORMAL	1.3	YES
76	LALITHA	20	PRIMI	21.08.16	28.05.17	22	245	TERM	LSCS	2.7	NO
77	KOKILA	21	G2PIL1	23.06.16	30.03.17	18	256	TERM	NORMAL	2.8	NO
78	ANANDHI	23	G2PIL1	14.06.16	21.03.17	22	287	TERM	NORMAL	2.7	NO
79	SENBAGAM	23	PRIMI	20.07.16	27.03.17	24	245	TERM	NORMAL	1.9	YES
80	LALITHA	21	PRIMI	21.08.16	28.05.17	20	278	TERM	NORMAL	2.7	NO
81	PRABHA	20	G2PIL1	11.06.16	18.04.17	18	221	TERM	NORMAL	2.8	NO
82	GEETHA	20	PRIMI	25.07.16	01.05.17	22	190	TERM	NORMAL	2.8	NO
83	JOTHI	21	PRIMI	19.08.16	26.05.17	24	287	TERM	NORMAL	2.8	NO
84	MANJULA	21	G2PIL1	03.09.16	10.06.17	20	187	TERM	NORMAL	2.8	NO
85	MANGAI	23	G2PIL1	05.07.16	12.04.17	18	280	TERM	NORMAL	2.7	NO

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86	NAGALAKSHMI	25	G2PIL1	12.07.16	19.04.17	24	236	TERM	LSCS	2.9	NO
87	GANGA	20	PRIMI	23.08.16	30.05.17	20	251	TERM	LSCS	2.9	NO
88	ARTHI	20	PRIMI	26.09.16	03.07.17	22	302	PRETERM33	NORMAL	1.3	YES
89	PRAMA	22	G2PIL1	07.07.16	14.04.17	18	289	TERM	LSCS	2.9	NO
90	SUMITHRA	22	PRIMI	09.08.16	17.05.17	22	213	TERM	LSCS	2.9	NO
91	PARIMALA	21	G2PIL1	02.09.16	09.06.17	24	187	TERM	LSCS	2.9	NO
92	DAICY	23	PRIMI	14.07.16	21.04.17	18	336	PRETERM35	NORMAL	2.3	YES
93	JERCY	23	G2PIL1	17.08.16	24.05.17	20	245	TERM	LSCS	2.7	NO
94	GIRIJA	19	PRIMI	17.08.16	24.05.17	24	265	TERM	NORMAL	2.7	NO
95	BALA	20	PRIMI	03.09.16	10.06.17	22	176	TERM	LSCS	2.7	NO
96	RAGINI	20	PRIMI	09.09.16	16.06.17	18	234	TERM	NORMAL	2.7	NO
97	ANNALAKSHMI	21	G2PIL1	10.08.16	17.05.17	22	211	PRETERM34	LSCS	1.9	YES
98	MUTHULAKSHMI	22	G2PIL1	11.07.16	18.04.17	20	287	TERM	NORMAL	2.8	NO
99	RASATHI	21	PRIMI	08.09.17	15.06.17	24	240	TERM	NORMAL	2.8	NO
100	VEEDAVALLI	22	PRIMI	09.08.16	17.05.17	20	243	TERM	NORMAL	2.8	NO
101	GANDHIMADHI	25	PRIMI	13.06.16	20.03.17	18	298	TERM	NORMAL	2.8	NO
102	KALPANA	23	PRIMI	09.06.16	16.03.17	22	248	TERM	NORMAL	2.8	NO
103	MAHALAKSHMI	24	G2PIL1	22.06.16	29.03.17	20	256	PRETERM34	NORMAL	1.8	YES
104	MANGAI	19	PRIMI	27.07.16	03.04.17	18	287	TERM	LSCS	2.9	NO
105	NAKISBANU	21	G2PIL1	16.08.16	23.05.17	24	280	TERM	LSCS	2.9	NO
106	PACHAYAMMA	22	PRIMI	10.06.16	17.03.17	20	234	TERM	LSCS	2.9	NO
107	ASLIMA	26	PRIMI	19.06.16	26.03.17	22	345	TERM	LSCS	2.9	NO
108	DURGADEVI	20	G2PIL1	23.06.16	30.03.17	18	256	TERM	NORMAL	2.8	NO
109	BAKYAVATHI	20	PRIMI	08.07.16	15.04.17	22	278	PRETERM36	LSCS	2.4	NO
110	SALIMA	19	PRIMI	13.08.16	20.05.17	24	267	TERM	LSCS	3	NO
111	VALLI	21	PRIMI	04.06.16	11.03.17	20	287	TERM	LSCS	2.9	NO
112	AKSHYA	23	G2PIL1	28.07.16	04.05.17	18	213	TERM	LSCS	2.9	NO
113	INDRA	20	G2PIL1	20.09.16	27.06.17	22	327	TERM	LSCS	2.9	NO
114	ROHINI	21	G2PIL1	03.08.16	10.05.17	20	267	TERM	NORMAL	2.8	NO
115	PREMA	19	PRIMI	08.07.16	15.04.17	24	289	PRETERM33	NORMAL	1.3	YES
116	CHITRA	20	PRIMI	10.06.16	17.03.17	18	298	TERM	NORMAL	2.8	NO
117	SHAJITHA	20	G2PIL1	30.06.16	07.03.17	22	234	TERM	NORMAL	2.8	NO
118	BEGAM	21	G2PIL1	14.07.16	21.04.17	20	222	TERM	NORMAL	2.8	YES
119	JANAKI	24	PRIMI	12.06.16	19.03.17	24	221	TERM	NORMAL	2.8	NO
120	SUDHA	24	PRIMI	22.06.16	29.03.17	22	256	TERM	NORMAL	2.8	NO
121	SUSELA	21	G2PIL1	12.07.16	19.04.17	18	246	TERM	NORMAL	2.8	NO
122	AYESHA	20	PRIMI	09.06.16	16.03.17	20	324	PRETERM-35WKS	NORMAL	2.1	YES
123	MUMTAJ	20	G2PIL1	16.06.16	23.03.17	24	240	TERM	NORMAL	2.8	NO
124	CELLIN	21	G2PIL1	29.07.16	05.04.17	18	200	TERM	LSCS	2.9	NO
125	JERON	21	PRIMI	23.06.16	30.03.17	24	204	TERM	LSCS	3.2	NO
126	HEMAVATHI	22	PRIMI	02.06.16	09.03.17	20	278	TERM	LSCS	2.9	NO
127	RAM THAI	24	G2PIL1	03.07.16	10.04.17	22	245	TERM	LSCS	2.9	NO

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128	SANKU	25	G2PIL1	20.08.16	27.05.17	24	231	TERM	LSCS	2.9	NO
129	NANDHINI	22	G2PIL1	12.07.16	19.04.17	18	256	TERM	NORMAL	2.8	NO
130	LORETTA	23	PRIMI	05.08.16	12.04.16	22	251	TERM	NORMAL	2.8	NO
131	VIJAYA	24	PRIMI	10.08.16	17.05.17	20	287	TERM	NORMAL	2.8	NO
132	STELLA	20	G2PIL1	04.07.16	11.04.17	24	265	TERM	NORMAL	2.8	NO
133	VASANTHI	21	G2PIL1	18.09.16	25.06.17	20	249	TERM	NORMAL	2.8	NO
134	JAMUNA	23	PRIMI	15.06.16	22.03.17	18	232	TERM	NORMAL	2.8	NO
135	THENMOZHI	21	G2PIL1	02.07.16	09.04.17	22	274	TERM	NORMAL	2.8	NO
136	SUSILA	20	PRIMI	13.09.16	20.06.17	20	245	TERM	NORMAL	2.8	NO
137	SIVAGAMI	20	PRIMI	09.08.16	16.05.17	24	303	TERM	NORMAL	2.8	NO
138	MANI	22	G2PIL1	25.07.16	01.04.17	18	212	TERM	NORMAL	2.8	NO
139	KUMARI	21	PRIMI	20.06.16	27.03.17	22	235	TERM	LSCS	2.9	NO
140	RAMA	23	G2PIL1	01.08.16	08.05.17	20	267	TERM	NORMAL	2.8	NO
141	RANI	21	PRIMI	03.09.16	10.06.17	20	287	TERM	LSCS	2.9	NO
142	SELVI	23	PRIMI	17.07.16	24.04.17	18	245	TERM	LSCS	2.9	NO
143	KUMUTHA	26	G2PIL1	05.08.16	12.05.17	22	265	TERM	NORMAL	2.8	NO
144	KANNAMA	21	PRIMI	14.06.16	21.03.17	24	287	TERM	NORMAL	2.8	NO
145	RASATHI	23	G2PIL1	18.07.16	25.04.17	22	267	TERM	NORMAL	2.8	NO
146	MEENAKSHI	23	PRIMI	19.09.16	26.06.16	18	231	TERM	NORMAL	2.8	NO
147	KARPAGAM	21	PRIMI	22.07.16	29.04.17	20	271	TERM	NORMAL	2.8	NO
148	SANTHI	25	G2PIL1	29.06.16	06.04.17	18	269	TERM	LSCS	3.1	NO
149	HEME	21	G2PIL1	08.08.16	15.05.17	24	265	TERM	NORMAL	2.8	NO
150	MALLIGA	23	G2PIL1	10.09.16	17.06.17	20	236	TERM	NORMAL	2.8	NO
151	CHANDRA	24	G2PIL1	26.07.16	02.05.17	22	301	TERM	LSCS	2.9	NO
152	AMUDHA	21	PRIMI	11.08.16	18.05.17	24	310	TERM	LSCS	2.9	NO
153	RAMA RANI	25	G2PIL1	29.06.16	06.04.17	18	278	TERM	NORMAL	2.7	NO
154	MYTHILI	24	G2PIL1	30.07.16	06.05.17	22	211	TERM	LSCS	2.7	NO
155	POONGODI	21	PRIMI	22.08.16	29.05.17	20	287	TERM	NORMAL	2.7	NO
156	VENNILA	23	G2PIL1	19.06.16	26.03.17	22	256	TERM	NORMAL	2.7	NO
157	RAMANI	24	PRIMI	30.07.16	06.05.17	24	287	TERM	NORMAL	2.7	NO
158	RENUKA	26	PRIMI	13.08.16	20.05.17	20	287	TERM	LSCS	2.7	NO
159	GAYATHRI	25	G2PIL1	24.06.16	01.04.17	18	218	TERM	NORMAL	2.7	NO
160	SREJA	24	PRIMI	11.06.16	18.03.17	24	254	TERM	NORMAL	2.7	NO
161	RAMADEVI	21	G2PIL1	18.09.16	25.06.17	22	265	TERM	NORMAL	2.7	NO
162	POORAKALA	20	PRIMI	20.06.16	27.03.17	18	239	TERM	NORMAL	2.7	NO
163	RADHIKA	21	G2PIL1	24.06.16	01.04.17	20	245	TERM	LSCS	2.7	NO
164	USHA RANI	20	PRIMI	10.07.16	17.04.17	22	231	TERM	NORMAL	2.7	NO
165	DEVI	21	PRIMI	30.07.16	06.04.17	24	180	TERM	LSCS	3.3	NO
166	USHA RANI	23	G2PIL1	06.08.16	13.05.17	18	188	TERM	NORMAL	2.8	YES
167	KAMAKSHI	23	PRIMI	18.08.16	25.05.17	20	204	TERM	NORMAL	2.8	NO
168	ARULMOZHI	25	G2PIL1	14.07.16	21.04.17	22	321	TERM	NORMAL	2.8	NO
169	MAINA	24	G2PIL1	18.08.16	25.05.17	24	239	TERM	NORMAL	2.8	NO
170	GOMATHI	20	PRIMI	10.08.16	17-05.17	20	310	TERM	NORMAL	2.8	NO

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171	SASIKALA	23	G2PIL1	11.07.16	18.04.17	22	239	TERM	NORMAL	2.8	NO
172	SIVAGAMI	21	G2PIL1	10.08.16	17.05.17	18	235	TERM	NORMAL	2.8	NO
173	PAVALAM	21	G2PIL1	17.06.16	23.03.17	24	293	TERM	NORMAL	2.8	YES
174	SUNDARI	22	G2PIL1	05.06.16	30.03.17	20	234	TERM	NORMAL	2.8	NO
175	VALARMATHI	25	G2PIL1	20.07.16	27.04.17	24	211	TERM	NORMAL	2.8	NO
176	PARAMESWARI	20	PRIMI	21.08.16	28.04.17	22	203	TERM	NORMAL	2.8	NO
177	BAKYA	24	G2PIL1	23.06.16	30.03.17	24	243	TERM	NORMAL	2.8	NO
178	BHARATHI	21	PRIMI	14.06.16	21.03.17	22	278	TERM	NORMAL	2.8	NO
179	GAYATHRI	21	PRIMI	20.07.16	27.04.17	20	231	TERM	NORMAL	2.7	NO
180	PADMA	23	G2PIL1	22.08.16	29.05.17	18	238	TERM	LSCS	2.7	NO
181	RASATHI	22	PRIMI	12.09.16	19.06.17	20	278	TERM	NORMAL	2.7	NO
182	NISHA	27	G2PIL1	28.07.16	04.05.17	18	273	TERM	NORMAL	2.7	NO
183	INDHU	26	PRIMI	22.08.16	29.05.17	24	323	TERM	LSCS	2.7	NO
184	VANI	25	PRIMI	24.09.16	01.07.17	22	269	TERM	NORMAL	2.7	NO
185	DEVIPRIYA	19	PRIMI	15.07.16	22.04.17	20	261	TERM	NORMAL	2.8	NO
186	PRIYA	22	G2PIL1	19.06.16	26.03.17	18	256	TERM	NORMAL	2.7	NO
187	SATHYA	23	PRIMI	30.08.16	06.06.17	24	203	TERM	NORMAL	2.8	NO
188	MANIMEGALAI	24	PRIMI	27.09.16	04.07.17	22	206	TERM	NORMAL	2.8	NO
189	SRIDEVI	19	G2PIL1	17.07.16	24.04.17	20	243	TERM	NORMAL	2.7	NO
190	VIJAYA	19	G2PIL1	20.08.16	27.05.17	18	192	TERM	NORMAL	2.8	NO
191	SHYLAJA	20	G2PIL1	10.09.16	17.06.17	24	347	PRE TERM ~34 WEEKS	NORMAL	2	YES
192	SHAKELA	20	PRIMI	18.07.16	25.04.17	22	288	TERM	LSCS	2.9	NO
193	UMA	21	G2PIL1	27.08.16	03.06.17	20	268	TERM	LSCS	2.9	YES
194	SAFEEDA	23	G2PIL1	26.09.16	03.07.17	24	232	TERM	LSCS	2.9	NO
195	LATHA	24	PRIMI	10.08.16	17.05.17	20	195	TERM	LSCS	2.9	NO
196	SATHYA	24	G2PIL1	11.09.16	18.06.17	20	262	TERM	NORMAL	2.8	NO
197	SARASWATHI	21	G2PIL1	19.08.16	26.05.17	22	280	TERM	LSCS	3.25	NO
198	SARITHA	21	PRIMI	20.07.16	27.04.17	24	194	TERM	NORMAL	2.8	NO
199	AMBIKA	23	PRIMI	28.09.16	05.07.17	20	184	TERM	NORMAL	2.7	NO
200	NARMATHA	26	PRIMI	29.08.16	05.06.17	22	198	TERM	NORMAL	2.7	NO
201	SELVI	25	PRIMI	13.06.16	20.03.17	22	232	TERM	NORMAL	2.7	NO
202	SARANYA	23	PRIMI	19.06.16	26.03.17	24	278	TERM	NORMAL	2.7	NO
203	AGALYA	21	G2PIL1	16.07.16	23.04.17	20	329	PRE TERM ~32 WEEKS	NORMAL	2	YES
204	SANGETA	22	G2PIL1	29.06.16	06.04.17	18	243	TERM	NORMAL	2.7	NO
205	DEEPA	21	PRIMI	28.06.16	05.04.17	22	267	TERM	NORMAL	2.7	NO
206	RAJAKUMARI	19	PRIMI	02.08.16	09.05.17	18	189	TERM	NORMAL	2.7	NO
207	PAVITHRA	19	PRIMI	03.07.16	10.04.17	22	198	TERM	LSCS	2.9	NO
208	BARANI	21	G2PIL1	10.06.16	17.03.17	24	200	TERM	LSCS	2.9	NO
209	SAVITHA	22	PRIMI	14.07.16	21.04.17	20	278	TERM	LSCS	3	YES
210	MALATHI	22	G2PIL1	20.06.16	27.03.17	18	356	TERM	LSCS	2.9	NO
211	RENUKA	21	G2PIL1	10.07.16	17.04.17	22	333	TERM	NORMAL	2.7	NO

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212	PRADEEPA	23	PRIMI	24.07.16	01.05.17	20	234	TERM	NORMAL	2.7	NO
213	SWETHA	20	G2PIL1	18.08.16	25.05.17	24	198	TERM	NORMAL	2.8	NO
214	LAVANYA	20	PRIMI	22.07.16	29.04.17	18	264	TERM	NORMAL	2.8	NO
215	ANITHA	25	G2PIL1	19.08.16	26.04.17	20	244	TERM	NORMAL	2.8	NO
216	ROSE	24	G2PIL1	23.06.16	30.03.17	24	224	TERM	NORMAL	2.8	NO
217	KAMAKSHI	21	PRIMI	21.07.16	28.04.17	24	186	TERM	NORMAL	2.7	NO
218	SUDHA	22	PRIMI	26.06.16	03.03.17	20	254	TERM	NORMAL	2.7	NO
219	RANI	23	PRIMI	20.06.16	27.03.17	18	348	PRE TERM ~32 WEEKS	LSCS	1.95	YES
220	SEETHA	27	G2PIL1	02.07.16	09.04.17	24	244	TERM	NORMAL	2.8	NO
221	POORNA	21	PRIMI	14.09.16	21.06.17	22	186	TERM	LSCS	2.9	NO
222	ANUJA	20	G2PIL1	20.07.16	27.04.17	20	202	TERM	LSCS	2.9	NO
223	AMMU	21	G2PIL1	18.08.16	25.05.17	24	319	TERM	LSCS	2.9	NO
224	ANUSHYA	21	PRIMI	30.06.16	07.03.17	18	265	TERM	NORMAL	2.8	NO
225	PRIYA	20	G2PIL1	30.07.16	06.03.17	22	278	TERM	LSCS	2.9	NO
226	KRISHNAVENI	20	G2PIL1	16.09.16	23.06.17	18	184	TERM	NORMAL	2.8	NO
227	KAVITHA	21	PRIMI	04.08.16	11.05.17	24	196	TERM	LSCS	2.9	NO
228	CHITHRA	23	G2PIL1	11.07.16	18.04.17	20	212	TERM	NORMAL	2.8	NO
229	KALAIVANI	21	G2PIL1	15.09.16	22.06.17	22	310	TERM	LSCS	2.9	NO
230	DIVIYA	20	G2PIL1	20.06.16	27.03.17	24	186	TERM	LSCS	3.1	NO
231	NATHIYA	22	G2PIL1	14.08.16	21.05.17	18	243	TERM	NORMAL	2.7	NO
232	RAJALAKSHMI	21	PRIMI	24.07.16	31.04.17	22	254	TERM	NORMAL	2.7	NO
233	JAYALAKSHMI	23	G2PIL1	24.08.16	31.05.17	20	259	TERM	NORMAL	2.8	NO
234	SUBHA	25	G2PIL1	02.07.16	09.04.17	18	337	TERM	NORMAL	2.8	YES
235	MANOGRI	24	PRIMI	20.07.16	27.04.17	24	183	TERM	NORMAL	2.8	NO
236	MANGALAM	23	PRIMI	02.07.16	09.04.17	22	198	TERM	NORMAL	2.8	NO
237	CHANDRA	21	G2PIL1	22.08.16	29.05.17	24	224	TERM	NORMAL	2.8	NO
238	SUGANYA	24	PRIMI	16.07.16	23.04.17	22	324	TERM	LSCS	2.9	NO
239	SANGEETHA	23	G2PIL1	10.08.16	17.05.17	20	186	TERM	LSCS	3.3	NO
240	RATHIKA	21	G2PIL1	12.06.16	19.03.17	18	192	TERM	LSCS	2.9	NO
241	SUGANYA	26	G2PIL1	11.06.16	18.03.17	22	240	TERM	LSCS	2.9	NO
242	REVATHI	27	PRIMI	18.06.16	25.03.17	24	244	TERM	LSCS	2.9	YES
243	MALAR	20	G2PIL1	04.09.16	11.06.17	20	254	TERM	LSCS	2.9	NO
244	SUGANTHI	22	G2PIL1	25.08.16	01.06.17	18	250	TERM	NORMAL	2.8	NO
245	SOLAIYAMMA	21	PRIMI	18.06.16	25.03.17	22	182	TERM	NORMAL	2.8	NO
246	JAYANTHI	22	G2PIL1	20.07.16	27.04.17	24	224	TERM	NORMAL	2.8	NO
247	KIRUTHIKA	24	G2PIL1	19.08.16	26.05.17	18	194	TERM	LSCS	2.9	NO
248	KAVYA	23	PRIMI	02.09.16	09.06.17	22	224	TERM	LSCS	2.9	NO
249	SOBANA	21	G2PIL1	08.07.16	15.04.17	24	341	PRE TERM ~34 WEEKS	NORMAL	2.2	YES
250	TAMIL	23	G2PIL1	11.07.16	18.04.17	20	187	TERM	NORMAL	2.7	NO
251	PUSHPA	19	G2PIL1	12.06.16	19.05.17	18	198	TERM	NORMAL	2.6	NO

S.NO	NAME	AGE	OBSTETRIC CODE	LMP	EDD	BLOOD COLLECTION WEEKS	S.CHOLESTEROL	OUTCOME	MODE OF DELIVERY	FETAL WEIGHT (KG)	NICU ADMISSION
252	THARA	20	G2PIL1	22.08.16	29.05.17	24	322	PRE TERM ~32 WEEKS	LSCS	1.9	YES
253	SHEELA	21	PRIMI	20.07.16	27.04.17	22	194	TERM	NORMAL	2.8	YES
254	CLARA	26	G2PIL1	03.06.16	10.03.17	20	226	TERM	LSCS	2.6	NO
255	KANIKA	23	G2PIL1	07.07.16	14.04.17	18	208	TERM	NORMAL	2.8	NO
256	KAVITHA	23	PRIMI	10.06.16	17.03.17	20	246	TERM	LSCS	3.5	NO
257	GAYATHRI	23	PRIMI	30.07.16	06.04.17	22	268	TERM	NORMAL	2.7	NO
258	SAROJA	24	G2PIL1	15.06.16	22.03.17	24	278	TERM	NORMAL	2.7	NO
259	KANMANI	20	G2PIL1	24.08.16	01.06.17	20	256	TERM	NORMAL	2.7	NO
260	NANDHINI	19	PRIMI	05.09.16	12.06.17	18	286	TERM	NORMAL	2.7	NO
261	KALAIVANI	19	PRIMI	28.06.16	05.04.17	18	185	TERM	LSCS	2.9	NO
262	JANCY	22	PRIMI	30.07.16	06.04.17	24	189	TERM	LSCS	2.9	NO
263	SIVARANJANI	21	G2PIL1	22.06.16	29.03.17	20	192	TERM	LSCS	2.9	NO
264	RATHI	24	G2PIL1	11.07.16	18.04.17	22	244	TERM	NORMAL	2.7	NO
265	SHALINI	25	PRIMI	12.08.16	19.05.17	18	186	TERM	LSCS	2.9	NO
266	ALAMELU	26	PRIMI	15.07.16	22.04.17	24	230	TERM	LSCS	2.9	NO
267	MARY	19	G2PIL1	13.06.16	20.03.17	22	240	TERM	NORMAL	2.8	NO
268	ELAVARASI	25	PRIMI	15.09.16	22.06.17	20	254	TERM	NORMAL	2.7	NO
269	JAYA	21	G2PIL1	07.09.16	14.06.17	18	198	TERM	LSCS	2.7	NO
270	AJITHA	23	G2PIL1	21.06.16	28.03.17	24	248	TERM	NORMAL	2.7	YES
271	RAJI	22	G2PIL1	30.06.16	07.04.17	18	234	TERM	NORMAL	2.7	NO
272	RAGA	21	G2PIL1	03.10.16	10.07.17	24	188	TERM	NORMAL	2.7	NO
273	SHEELA	22	PRIMI	29.07.16	05.05.17	22	190	TERM	NORMAL	2.7	NO
274	MALLI	23	G2PIL1	30.08.16	06.06.17	20	194	TERM	NORMAL	2.8	NO
275	MARY	19	G2PIL1	12.09.16	19.06.17	22	223	TERM	NORMAL	2.6	NO
276	JAYA	20	PRIMI	07.06.16	13.03.17	18	256	TERM	LSCS	2.6	NO
277	SHANTHI	20	PRIMI	08.06.17	15.03.17	22	326	PRE TERM ~32 WEEKS	NORMAL	1.85	YES
278	MALIKA	22	G2PIL1	22.07.16	29.04.17	20	286	TERM	LSCS	2.9	NO
279	JAMILA	22	PRIMI	23.08.16	30.05.2017	24	285	TERM	NORMAL	2.8	NO
280	BLESSY	21	G2PIL1	24.06.16	01.04.17	18	245	TERM	NORMAL	2.8	NO
281	SHREYA	23	G2PIL1	04.06.16	11.03.17	20	196	TERM	NORMAL	2.8	NO
282	RAMYA	24	PRIMI	08.06.16	15.03.17	18	220	TERM	NORMAL	2.8	NO
283	VALLI	22	G2PIL1	12.07.16	19.04.17	22	314	TERM	NORMAL	2.7	YES
284	SUJI	25	PRIMI	24.07.16	31.04.17	18	188	TERM	LSCS	2.7	NO
285	ANITHA	20	G2PIL1	30.07.16	06.05.17	24	179	TERM	NORMAL	2.7	NO
286	SUKUMARI	20	PRIMI	02.08.16	09.05.17	22	306	TERM	NORMAL	2.8	NO
287	THARANI	24	G2PIL1	22.08.16	29.05.17	20	250	TERM	NORMAL	2.8	NO
288	BHARATHI	21	PRIMI	02.09.16	09.06.17	24	224	TERM	NORMAL	2.8	NO
289	KALAI	19	PRIMI	09.06.16	16.03.17	20	228	TERM	NORMAL	2.8	NO
290	KOUSALYA	19	G2PIL1	22.06.16	29.03.17	18	218	TERM	NORMAL	2.8	NO
291	NATHIYA	20	G2PIL1	21.08.16	28.05.17	22	210	TERM	NORMAL	2.8	NO
292	KUMARI	26	G2PIL1	03.06.16	10.03.17	24	289	TERM	NORMAL	2.8	NO

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293	RANI	21	PRIMI	04.08.16	11.05.17	20	190	TERM	NORMAL	2.8	NO
294	MANJU	21	G2PIL1	22.09.16	29.06.17	18	230	TERM	NORMAL	2.8	NO
295	VANI	22	G2PIL1	02.06.16	09.03.17	22	335	TERM	NORMAL	2.8	NO
296	SATHYA	23	PRIMI	09.06.16	16.03.17	24	178	TERM	NORMAL	2.8	NO
297	AMBIKA	25	PRIMI	17.07.16	24.04.17	20	238	TERM	LSCS	2.9	NO
298	ANITHA	24	G2PIL1	02.08.16	09.05.17	22	223	TERM	LSCS	2.9	NO
299	MARIYAM	20	PRIMI	19.09.16	26.06.17	24	178	TERM	LSCS	2.9	NO
300	USHA	19	PRIMI	20.08.16	27.05.17	20	200	TERM	LSCS	2.9	NO