A STUDY ON FETOMATERNAL OUTCOME IN GESTATIONAL DIABETES MELLITUS DIAGNOSED BY IADPSG (INTERNATIONAL ASSOCIATION OF DIABETES IN PREGNANCY STUDY GROUP) CRITERIA AT TERTIARY CARE HOSPITAL.

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BONAFIDE CERTIFICATE

This is to certify that this dissertation entitled **A STUDY ONFETOMATERNAL OUTCOME IN GESTATIONAL DIABETES MELLITUS DIAGNOSED BY IADPSG (INTERNATIONAL ASSOCIATION OF DIABETES IN PREGNANCY STUDY GROUP) CRITERIA AT TERTIARY CARE HOSPITAL** is the bonafide work done by **Dr.SOLAIRAJALAKSHMI** at the department of Obstetrics and Gynaecology, **Institute of Obstetrics and Gynaecology, Madras Medical College, Chennai** during her post graduate study for MS Branch II Obstetrics and Gynaecology (2021-22) under the guidance of Prof. Dr.S.VIJAYA M.D., D.G.O. This dissertation submitted to **THE TAMILNADU Dr.M.G.R MEDICAL UNIVERSITY** in partial fulfilment of the University rules and regulations for the award of MS Degree in Obstetrics and Gynaecology.

Prof. Dr. S.Vijaya, M.D.,D.G.O
Professor & Director
Institute of Obstetrics & Gynaecology,
Madras Medical College, Chennai

Prof. **DR. E.THERANIRAJAN**, MD.,DCH.,MRCPCH(UK).,FRCPCH(UK)
Dean
Madras Medical College, Chennai

Date :

Place : Chennai

DECLARATION

I hereby declare that this dissertation entitled **“ A STUDY ONFETOMATERNAL OUTCOME IN GESTATIONAL DIABETES MELLITUS DIAGNOSED BY IADPSG (INTERNATIONAL ASSOCIATION OF DIABETES IN PREGNANCY STUDY GROUP) CRITERIA AT TERTIARY CARE HOSPITAL,** is a bonafide and genuine research work carried out by me after studying the cases in outpatient department at Institute of Obstetrics and Gynaecology, Madras Medical College, Chennai during the period November 2020 to November 2021, under the direct guidance and supervision of **Dr. Vijaya, M.D.,DGO,** Director of Institute of Obstetrics and Gynaecology, Madras Medical College, Chennai. It is submitted to The Tamil Nadu Dr.M.G.R Medical University, Chennai, in partial fulfilment of its regulation for the award of **M.S. (Obstetrics And Gynaecology)** Degree to be held in April 2022.

Place: Chennai.

Date:

(SOLAIRAJALAKSHMI.S)

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Dr. S.SOLAIRAJALAKSHMI

CONTENTS

INTRODUCTION

Diabetes Mellitus – constitutes a group of metabolic disorder resulting from either dysfunction of insulin secretion, insulin action, the sensitivity of the tissue to insulin, all resulting in hyperglycemia. Under the influence variety of factors including genetics, environmental & life style factors. There is exponential rise in incidence & prevalence of diabetes mellitus all over the world. Incidence is expected to rise by 20%

The trend of incidence of DM has changed recently from the older people towards the younger population. This change of trend in turn leads to increase in incidence of gestational diabetes mellitus.

GDM is defined as the carbohydrate intolerance of varying severity with maternal & fetal risk during present pregnancy irrespective of the method of treatment .Of all the women diagnosed with Gestational diabetes ,some may have undiagosed Type2 Diabetes mellitus recognised first in the current pregnancy.

Prevalence of Gestational diabetes mellitus varies depends on the population under study, their ethnicity, environmental factors, genetic factors & so on. Nevertheless the outcome depends on the plasma glucose level I.e the glycemic control ascertained.

Our Indian population is said to be a high risk population for Type 2 diabetes mellitus and thus in-turn are at increased risk for the development of GDM .

The prevalence of GDM varies further in urban/semi urban/rural areas. Of which the urban people shares the higher risk owing to their sedentary lifestyle & increasing obesity.

The adverse maternal outcome of women with Gestational diabetes can be improved by better antenatal care and positive lifestyle changes.Despite proper antenatal care,the risk of adverse maternal and fetal outcome has increased drastically with increasing maternal plasma glucose level in the second and third trimester,even with the blood glucose level that is considered normal for pregnancy before.Hence arises the need for revision of already available diagnostic criteria cutoffs and introduction and implementation of newer techniques of screening and diagnosis.

AIMS AND OBJECTIVES OF THE STUDY

1. To evaluate perinatal and maternal outcomes in women diagnosed with Gestational diabetes mellitus (GDM) by IADPSG (International Association of Diabetes in Pregnancy Study Group).

2. To highlight for proper antenatal care regarding screening and diagnosis of GDM which will be helpful in identifying the magnitude of the problem and improving the patient care.

REVIEW OF LITERATURE

Diabetes Mellitus-A Timeline

The ancient Egyptians first described the clinical features of diabetes as early as 1550BC. At the tomb of Thebes,Egypt, a papyrus(paper) was found, which had mentions of polyuria a well known symptom of diabetes. It was told that the same papyrus was bought by George Ebers and was named after him. But then further experimentations suggested that it could be a copy from series of books written later in 3400BC (1,2)

The word ‘Diabetes ‘ was derived from the Greek word for ‘siphon’ which was coined by aretes of cappodocia from Ancient Greece (81-133AD) (3)

Indian hindu physicians charaka ,found that urine of the patients with DM attracted ants and flies due to its sugar content. They called it as honey urine. They described the clinical features of Diabetes Mellitus as polyuria, glycosuria.(4)

This sweetness of urine was first discovered by British physician ThomasWilliam in 1676, who added the word MELLITUS to ‘Diabetes’. (5)

1776 –Dr.Mathew Dobron ,Manchester, demonstrated the presence of sugar in urine & blood.

Early19th century –Claudie Bernard, France –discovered the role of liver in glycogenesis.

1869-Paul Langerhans , a general medical student , identified the pancreatic islets cells ,but he coudnt find their function .(6,7,8)

1893-Gustave Laguesse, named the islet cells after Paul Langerhans .(9,10)

1889- Oseas Minkowski, a diabetologist and Joseph Von Mering, a pharmacist found that a dog after removal of pancreas developed diabetes mellitus.

1921,Dr.Fredrick Banting & JJR Macleod and his assistant Best at the university of Toronto did experiments on dogs. They tried to exract the secretion of pancreas after ligating the duct which induced the hypertrophy of the pancreatic acini cells. These extract where then injected to dogs which were clinically made diabetic by removal of the pancreas .

The extract when injected to the diabetic dogs were successful in prolonging their life.

1922-Banting & Best tried treatment for the first time in human patients,Leonard Thomson were successful(11)

1923-Insulin was commercially produced in large quantities to meet the needs of the people.

1923- Noble prize was awarded for Banting & Macleod for their role in discovering of insulin which was shared by Best (12)

1940- it was the first time ever to notice that maternal blood glucose level affected the pregnancy outcomes

1942- Jorgen Pedersen suggested the pathophysiology of the macrosomia in infants of diabetic mother. It was later named Pedersen hypothesis, which states that maternal hyperglysemia leads to fetal hyperglycemia, which inturn leads to fetal hyperinsulinemia thus leading to accelerated fetal growth & macrosomia.

1961-the therm 'Gestational Diabetes Mellitus ' was first introduced by John B O'Sullivan

1964- O'Sullivan & Mohan – Diagnostic criteria introduced which was modified later by Carpenter & couston.

1970's – the Basic physiology of GDM I.e., facilitated anabolism & accelerated starvation were introduced by Norbert Frenkel.

2005- ACHOIS study described the improved outcomes in pregnancy complicated by GDM which when treated accordingly.

2006- DIPSI guideline for screening of GDM introduced.

2008- HAPO study findings published .

2010- IADPSG revises the diagnostic cot off for the diagnosis of GDM.

2012- IADPSG criteria was endorsed by ADA.

2013- NIH asked for more evidence before IADPSG criteria could be universally adopted.

First case of GDM was reported by Benniwitz in Germany in 1853. He considered the hyperglycaemia as a symptom of pregnancy rather than a complication. He also noted that glycosuria which was obvious during pregnancy ,resolved spontaneously after delivery.

CLASSIFICATION OF DM

In general population diabetes mellitus is classified mainly on the basis of etiopathogenesis & clinical manifestations. Type 1 diabetes is characterized by absolute deficiency of insulin where as Type 2 diabetes is attributed to decrease in insulin secretion, increased insulin resistance or increased blood glucose levels exceeding the production of insulin. However both the types of DM before manifesting are preceeded by a period of impaired glucose tolerance. (13)

TYPE 1:

Beta cell destruction – absolute insulin deficiency

Immune mediated

Idiopathic

TYPE 2:

Ranges from predominantly insulin resistance to predominantly an insulin secretory defect with insulin resistance

OTHER TYPE:

Genetic mutations of beta cell function MODY 1-6, others

Genetic defects in insulin action

Genetic syndromes – Down,Klinefelter, Turner

Diseases of exocrine pancreas –pancreatitis , cystic fibrosis

Endocrinopathies –Cushing syndrome, pheochromocytoma others

Drug or Chemical induced –Glucocorticosteroids, Thiazides, Beta –adrenergic agonists,others

Infections –congenital rubella, cytomegalo virus, coxsackievirus

GESTATIONAL DIABETES

MODY- maturity onset diabetes of the young

Pancreatic beta cell destruction, the main cause of DM occurs very easily in patients with type 1 DM and manifest clinically even before the age of 30. Type 2 diabetes usually develop with advanced age, but due to our changing life style incidence now increasing in young individuals especially there with obesity.

Whites classification

Based on,

1. The duration of the disease.
2. Age at onset of disease.
3. The complications.

Type A- Gestational diabetes

Type B-Pregestational diabetes(existed prior to pregnancy)

These groups are further subdivided according to their associated risks and management.

The two subtypes of gestational diabetes are,

Type A 1: Abnormal oral glucose tolerance test, but normal fasting and postprandial (2 hours after meals) blood glucose levels. Dietary modifications are sufficient to control blood glucose levels. l

Type A 2: Abnormal OGTT compounded by abnormal glucose levels during fasting and/or after meals. Additional therapy with insulin or other medications is required.

Type B: Onset at age 20 or older and duration of less than 10 years.

Type C: Onset at age 10–19 or duration of 10–19 years.

Type D: Onset before age 10 or duration greater than 20 years.

Type E: Overt diabetes mellitus with calcified pelvic vessels.

Type F: Diabetic nephropathy .

Type R: Proliferative retinopathy

Type RF: Retinopathy and nephropathy.

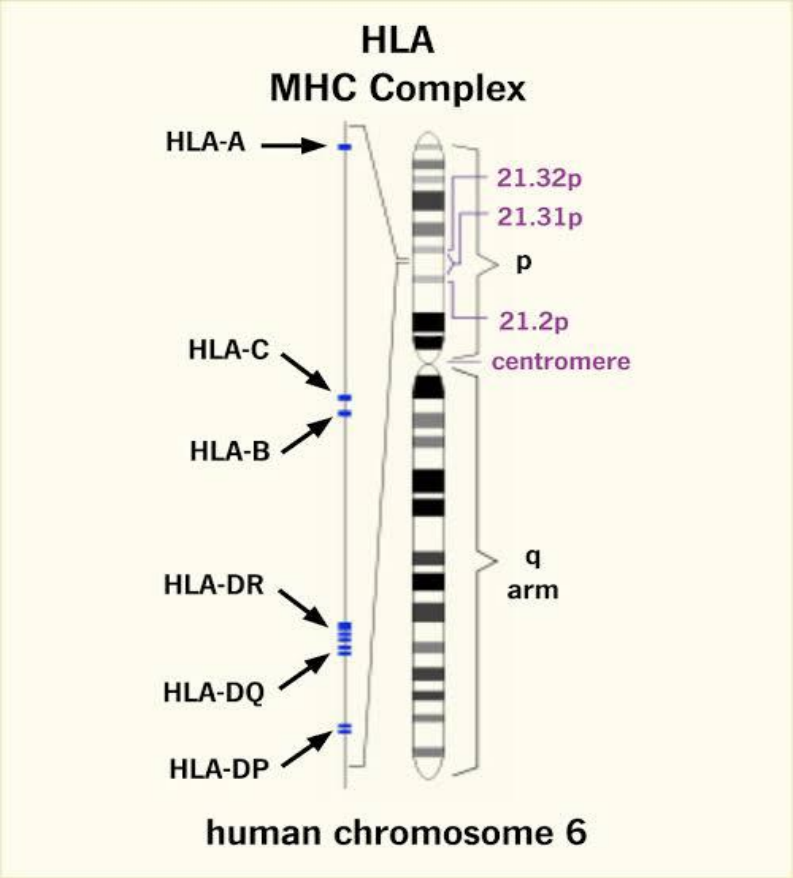
Type H: Ischemic heart disease.

Type T: Prior kidney transplant.

TYPE 1 DM

It is also called insulin dependent ,immune mediated or -------- diabetes. It is characterized by cellular mediated autoimmune destruction of the beta cells of Langherhans pancreas, the exact cause of this process is not known & its attributed to multiple genetic & environmental factors

The autoimmune cause of type1 DiabtesMellitus is confirmed by presence of markers i.e., ICA,EAA,GADA,IA2A which were demonstrated in 85-90% of individuals of type 1 DM. A strong association between type 1 DM & HLA antigen on chromosome 6p2, DQA & DQB genes were demonstrated.



Though type 1 diabetes can affect people of any age,it most commonly occurs in children,young adults.The progression of disease varies among individuals.Young adults show more rapid progression and often require hospital admissions especially due to ketoacidosis.

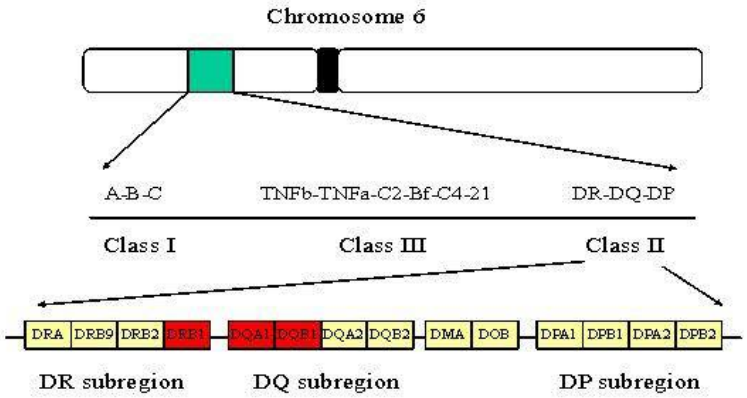


Figure 1: The HLA region on chromosome 6 (from Mehers and Gillespie 2008). The T1D associated haplotypes are DRB1*03-DQB1*02 and DRB1*04-DQB1*0302

The majority of beta cells of pancreas are destroyed by autoimmune process in type 1 diabetes hence,insulin remains their main drug of treatment.

Incidence of type1 diabetes can't b prevented and those women with type1 diabetes who gets pregnant lands up in a variety of maternal and fetal complications.

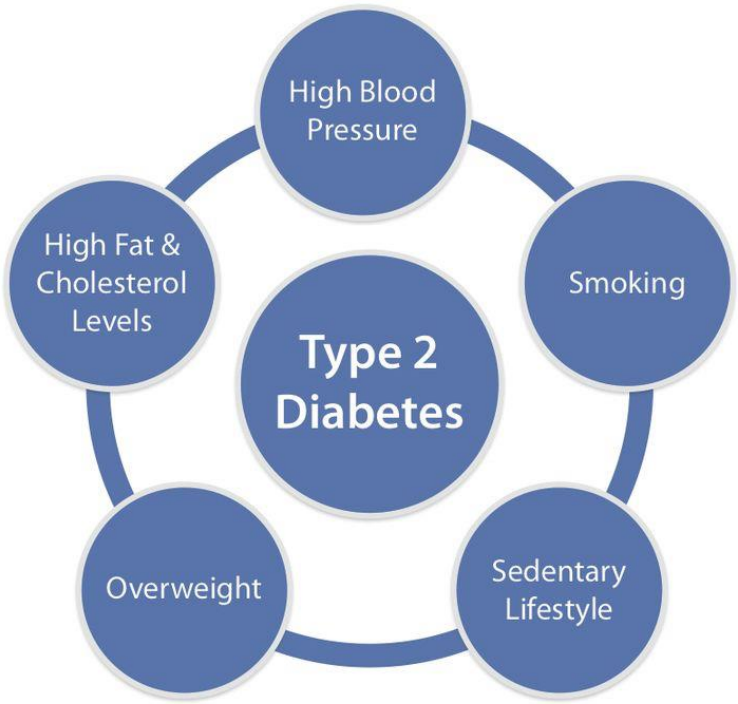
Evers et al, in their study found that the perinatal mortality in babies born of type 1 diabetic mother was 3.2% and prevalence of congenital anomalies was 4.8% (14).

Griffin et al, from their study at netherlands,showed a perinatal mortality of 2.8% in babies born of women with type1 diabetes and the prevalence of congenital malformations as 8.8%.

TYPE 2 DIABETES MELLITUS

It is also the non insulin dependent/ adult onset diabetes. It is characterised by relative insulin deficiency or insulin resistance or both. It is more common in older people and are more pronounced in people with obesity(15).Though it is called non insulin dependent diabetes,patients may over time need insulin as treatment.Type 2 diabetes often has an indolent course and are mostly diagnosed incidentally at regular check up or during evaluation for associated illnesses (16,17) .Though the

incidence of type 2 diabetes cannot be completely prevented,the clinical manifestations and its complications can be delayed by proper lifestyle modifications with a healthy diet and regular exercises. Pregnancy complicated by type2 diabetes has its own adverse outcomes.



In a UK study conducted during 1990-2002,the perinatal mortality rate was found to be 2.5% & that of congenital malformations was found to be 9.9% in women with type2 diabetes mellitus.

Griffin et al, in their study showed a perinatal mortality of 3.2% & the prevalence major congenital anomalies as 4.3%.

On comparing the pregnancy outcomes in women with type2 diabetes to those with type 1diabetes ,the incidence of congenital

malformations and perinatal mortality were found to be almost same(18,19,20).

GESTATIONAL DIABETES MELLITUS

Gestational diabetes mellitus is defined as" carbohydrate intolerance of any degree with its onset or first recognition during present pregnancy ".Gestational diabetes may resolve after delivery and the women may return to euglycemic status in postpartum. Despite that the definition doesn't exclude the glucose intolerance which was present before conception. Due to our changing lifestyle,number of women with obesity,overweight and diabetes has increased drastically leading to increase in number of women with undiagnosed type2 diabetes mellitus.

RISK FACTORS FOR GDM- nice

1) Body mass index more than 30kg/m2
2) Previous macrosomic baby weighing 4.5kg or more
3) Previous gestational diabetes
4) Family h/o diabetes (first degree relative with diabetes)
5) Family origin with a high prevalence of diabetes.

Those who are diagnosed with GDM may further be categorised as

1) Those with normal glucose tolerance before pregnancy, and develop Gestational diabetes which later becomes normal after delivery.
2) Women with mild impaired glucose tolerance before pregnancy that worsened during pregnancy.
3) Undiagnosed Type 2 Diabetes
4) Undiagnosed Type 1 Diabetes

The definition of Gestational diabetes mellitus which states the disease as onset or first recognition of impaired glucose tolerance diagnosed durning which may also include those with undiagnosed type2 or type 1 diabetes mellitus which could have been present even before pregnancy. The American College of Obstetricians and Gynecologists (ACOG),2019 and National Institute of Health(NIH) still endorses this definition.

CARBOHYDRATE METABOLISM

Carbohydrates are defined as the aldehyde or ketonic derivatives of higher polyhydroxy alcohols or anhydrides of such derivatives.(21)The energy requirements of the body are met preferentially by the oxidation of carbohydrates.The final end product of complex carbohydrate digestion are monosaccharides- glucose,fructose and galactose.

The metabolism of carbohydrate is subdivided as follows.(22)

1. Glycolysis-oxidation of glucose/ glycogen to pyruvate and lactate.(Embden- Meyerhof pathway)
2. Glycogenesis-synthesis of glycogen from glucose
3. Glycogenolysis-conversion of glycogen to glucose
4. Kreb's cycle (citric acid cycle) - final common pathway of oxidation of glucose, fatty acids and amino acids. Carbon dioxide and water are the end products.
5. Hexose monophosphate pathway- alternate pathway of oxidation.
6. Gluconeogenesis-synthesis of glucose from non carbohydrate source such as glycerol, lactate, amino acids, pyruvate, etc.

REGULATION OF BLOOD SUGAR

Alpha-beta D –glucose is the blood sugar(22). The normal blood sugar level is 70- 140mg/dl. The factors that increase blood glucose level are

1. Absorption from intestinal tract

2. Hepatic glycogenolysis

3. Gluconeogenesis

The factors that decrease the blood sugar level

1. Lipogenesis(synthesis of fats)

2. Glycogenesis in the liver

3. Glycogenesis in the muscle

4. Synthesis of glycoproteins and lactose

Kidneys play regulatory role in carbohydrate metabolism by reabsorption of glucose in the renal tubules. But once the blood sugar level crosses 180mg/dl (threshold level), sugar appears in the urine,called glycosuria.

INSULIN:

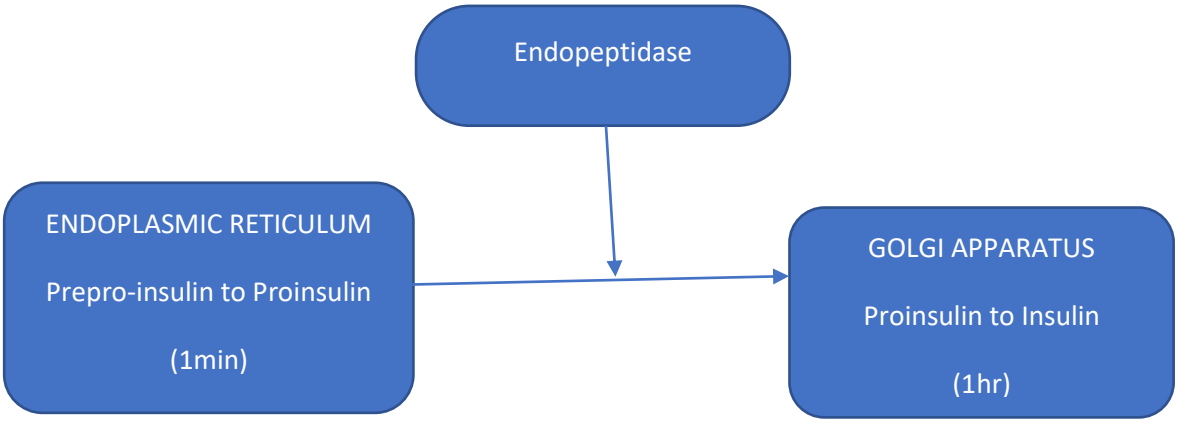
Insulin is a polypeptide hormone secreted by beta cells of pancreas.

Daily dose of secretion: 30-40units of insulin.

20units of basal and 20 units in response to nutrients11.

Insulin gene is situated on the short arm of chromosome number 11.

Glucose has no effect on the conversion of proinsulin to insulin.



FUNCTIONS OF INSULIN :

1. Facilitates absorption of glucose across the cell membrane .
2. Promotes glycolysis.
3. Promotes glycogenesis

4. Inhibits glycogenolysis
5. Synthesis of proteins from amino acids
7. Inhibits gluconeogenesis from proteins
8. Facilitates absorption of potassium, amino acids and phosphate into the cells.

Glucose Homeostasis:

Glucose homeostasis is pregnancy iinvolves strict regulation of maternal glucose to maintain the fetal blood glucose at optimal levels.

MATERNAL METABOLISM:

During normal pregnancy pregnancy,in order to maintain uninterrupted nutritional supply to the fetus a few changes occur in the metabolism of carbohydrate,lipids and protein.These changes also help in building the maternal stores to meet the demands of lactation.

At the early trimester of pregnancy,the metabolism is not altered such that the maternal glucose and insulin levels remains the same and the glucose tolerance is within normal limits or may even appear improved.Such that the fasting blood glucose levels appear to fall 10-15mg/FL lower that that of the non pregnant state(23)

As the gestation advances ,insulin secretion-both basal as well as the post prandial increases and almost reaches twice that of normal level by the end of third trimester. This increased insulin secretion is also accompanied by growing insulin resistance. Insulin resistance peaks at around 34-36weeks.(24)

Hormones held responsible for this insulin resistance include,

1) Human placental lactogen(HPL) also known as Human chorionic somatomammotropin
2) Progesterone
3) Estrogen
4) Prolactin
5) Cortisol

All of this hormones work together to lower the maternal fasting blood glucose levels and to increase the postprandial blood glucose levels.

EFFECTS OF DIABETES ON PREGNANCY:

Gestational as well as presentational diabetes both affect pregnancy invariably resulting in a variety of complications.

Maternal effects are due to macro vascular as well as micro vascular complications. These effects are more pronounced in patients with long-standing pre gestational diabetes.

Severity of the complications depends mainly on the duration of the disease and the level of glycemic control achieved.

Complications include,

1) End organ damage- Nephropathy

   Neuropathy

   Retinopathy

DIABETIC NEPHROPATHY

Renal disease develops in around 25–30% women with insulin-dependent diabetes of a long duration,

STAGES:

Stage 1: microalbuminuria (albumin to creatinine ratio ./5 3.5mg/mmol or 24 hr urinary collection showing urine albumin excretion of 20–199 mcg/min or 30–299 mg/24 hr)

Stage 2: macroalbuminuria (albumin to creatinine ratio ./5 30mg/mmol or urinary albumin concentration of 200mg/L or more)

Stage 3: end stage renal disease.

Stage 1 and 2 are mild disease and the patient may be asymptomatic. Proteinuria in these stages is usually unmasked by physical exercise. Further progression of renal damage can be prevented by optimal glycemic control and insulin therapy.

Assessment of renal function is important since, nephropathy can increase potential risks of preeclampsia, fetal growth restriction, preterm birth and chronic hypertension and maternal morbidity. Treatment of nephropathy prior to conception and after delivery with ACE inhibitors and Angiotensin receptor blockers is recommended. However these drugs are contraindicated in pregnancy due to their teratogenic effects like renal tubular dygenesis in the baby,oligohydramnios.

DIABETIC RETINOPATHY:

Non proliferative retinopathy needs only optimal glycemic control.

Proliferative retinopathy can be treated with pan retinal photo coagulation.

Diabetic macular edema needs focal/grid laser.

Untreated diabetic retinopathy may end up in increased intraocular pressure which may lead to rupture of blood vesssels resulting in intravitreal hemorrhage.

Recommended fundus examinations in known diabetics

1) Prior to conception
2) At first antenatal visit.
3) At 28 weeks.
4) Follow up after delivery.

Neuropathy may manifest just in the form of gastroparesis resulting in increased gastric emptying time. around 14–16 years, that is in class C and D of Whites classification or class F.

2) Diabetic cardiomyopathy
3) Preeclampsia 10%
4) Preterm labour
5) Chorioamnionitis
6) Polyhydramnios
7) Urinary tract infections
8) Ketoacidosis- precipating factors : febrile illnesses,dehydration from hype remesis or diarrhoeal disease.

   Maternal mortality rate-4-15%

   It can cause IUFD
9) Recurrent hypoglycaemic episodes requiring hospital admissions.

10) Increased need for instrumental delivery and Caesarean section - macrosomic babies, dysfunctional labour, prolonged
11) Postpartum complications-Postpartum hemorrhage, infections, puerperal sepsis.
12) Long term development of Type2 diabetes mellitus.

EFFECT OF DIABETES ON THE FETUS

1. Growth abnormalities-
    a) Macrosomia
    b) Growth restriction
    c) Congenital Malformations
2. Chemical/ electrolyte imbalances
3. Chronic fetal hypoxia
4. Respiratory distress syndrome
5. Sudden fetal demise.

Macrosomia, defined as fetal weight more than 90th centile for that gestational age or estimated fetal weight equal to or more than 4000g is the commoner abnormality.

PEDERSON HYPOTHESES

- Maternal hyperglycaemia
- Fetal hyperglycaemia
- Fetal hyperinsulinemia (25)(insulin as growth factor)
- Excessive fetal growth and subcutaneous fat deposition

Some suggest that this results in a larger fat pad in the shoulder and trunk region causing shoulder dystocia and subsequent birth trauma to the fetus (like clavicular fracture and brachial plexus injury) as well as the mother. There also occurs fetal hepatomegaly, splenomegaly and cardiomegaly due to hyperinsulinemia. The positive predictive value for detection of macrosomia is greater than 90% when the abdominal circumference is more than 95th centile. ACOG suggests that if gestational diabetes remains undiagnosed or untreated, the risk of macrosomia is as high as 20%.(26) In cases of pregestational diabetes of prolonged duration wherein there is evidence of systemic vasculopathy, there is a risk of development of uteroplacental insufficiency.

Uteroplacental insufficiency leads to intrauterine growth restriction. This vascular insufficiency is accompanied by maternal hypertension. Before 20 weeks, fetal islet cells are not well

developed. Thus, the main culprit is the high glucose levels. Exposure to high glucose levels at the time of organogenesis results in a number of fetal malformations in those with uncontrolled pregestational diabetes. More than 50% of these anomalies affect the central nervous or cardiovascular system. Following anomalies are associated: CNS: neural tube defects including anencephaly, meningomyelocoele, encephalocoele CVS: transposition of great vessels, ventricular and atrial septal defects, hypoplastic left heart and others Skeletal: Caudal regression syndrome, spinal anomalies Renal: hydronephrosis, renal agenesis, cystic kidneys Intestinal: duodenal atresia, anorectal malformation Maternal glycosylated haemoglobin levels in the first trimester may help to predict the risk of occurrence of congenital anomalies in the fetus in cases of pregestational diabetes(27). Studies show that: l l l HbA1c less than 7% – no greater risk for anomalies than nondiabetic mothers 7-8.5% – risk of 5% for anomalies .10% – risk of anomalies rises to 22% Periconceptionally, the patient should be counselled regarding the risk of anomalies on the basis of her glycemic control. In cases of high HbA1c levels, the decision regarding continuation of pregnancy is at the patient's discretion. Screening for anomalies must be done by ultrasound. Though most congenital anomalies occur early in

gestation, a condition called the small left colon syndrome may be seen in second half of gestation especially in type 1 diabetes. In this, there is uniformly small diameter of the descending and sigmoid colon and the rectum. It may result from wide fluctuations in the maternal and fetal glycemic levels. Hyperglucagonemia occurs in response to decrease in fetal glucose levels causing intestinal hypomotility. Intestinal motility is a main factor for stimulating intestinal growth and differentiation.(28) A neonate with small left colon syndrome may present with intestinal obstruction and may mimic meconium plug syndrome. Chemical Imbalances Fetal hypoglycemia due to maternal hypoglycemia can result in sudden intrauterine fetal death. Neonatal hypoglycemia occurs due to hyper insulinemia in the fetus and removal of the exogenous glucose source (maternal) at the time of delivery.(27).Hence these babies be closely observed. Other chemical imbalances seen in a neonate of a diabetic mother are hypocalcaemia and hypomagnesaemia which occur within 72 hours of birth. Hypocalcaemia occurs due to delayed postnatal parathyroid hormone regulation, pathophysiology of which is still unclear. This effect is independent of birth asphyxia. The cause of hypomagnesaemia is similar to that of calcium metabolism in the neonate. It could also occur due to long

standing diabetic nephropathy in the mother leading to maternal renal magnesium losses and hence reduced availability of magnesium for the fetus.(27). Risk of neonatal hyperbilirubinemia is increased due to preterm delivery, ineffective erythropoiesis, expanded red cell mass and relative immaturity of the hepatic bilirubin conjugation and excretion. Studies have shown that around 65% infants of diabetic mothers have abnormalities of iron metabolism. Due to accelerated erythropoiesis, there is deficiency of iron at tissue level indicated by low serum ferritin levels. Iron deficiency increases the risk for neurodevelopmental and behavioural abnormalities.15 However, these babies are not anaemic and spontaneous recovery of the iron status has been documented. There is also risk of respiratory distress syndrome due to surfactant deficiency. Babies of diabetic mothers are prone to this complication due to increased risk of preterm delivery and also due to late maturation of type II alveolar cells.16 Fetal hyperinsulinemia antagonizes the action of cortisol causing blunted production of surfactant. Fetal Oxygenation Problems Fetal hyperglycemia and hyperinsulinemia increase the rate of oxygen consumption by around 30% in a relatively oxygen limited environment.(25,29).Though the fetus increases substrate intake, there exists some degree of oxygen

deficit. This is further accelerated by placental vasculopathy. Chronic hypoxia results in excessive erythropoietin secretion by fetal kidneys causing accelerated erythropoiesis. This results in neonatal polycythemia and hyper viscosity. This may cause neonatal stroke, seizures, necrotizing enterocolitis and sudden fetal death. The degree of maternal hyperglycemia correlates with the severity of polycythemia. Studies have shown the use of amniotic fluid or cord erythropoietin as a marker of fetal hypoxia. Neonatal hypoglycemia, hypertrophic cardiomyopathy and admission to intensive care unit occur in those with a higher amniotic fluid erythropoietin levels. In a study, the mean amniotic fluid erythropoietin levels at term was found to be 14mU/ml in diabetic pregnancies (range of 2–1975mU/ml), and 6.3mU/ml in controls (range 1.7–13.7mU/ml).

Sudden intrauterine fetal death is associated with diabetic pregnancies which is difficult to predict by any kind of antenatal fetal surveillance. Umbilical artery Doppler can detect placental insufficiency which could be a cause. However in absence of vasculopathy, prediction of sudden fetal death is almost impossible. Though amniotic fluid erythropoietin level can indicate a hypoxic fetus, the feasibility of use of this parameter is still under study.

Moreover, it may not predict when a fetus may succumb. Various explanations for sudden fetal demise have been proposed which include maternal hypoglycemia, ketoacidosis, chronic hypoxia, placental villus edema impairing nutrient transfer. Long-Term Sequelae Babies born to diabetic mothers have the risk of developing obesity, type 2 diabetes, cardiovascular disease and impaired cognitive and motor function. This can be due to a combination of factors including genetic inheritance, intrauterine or perinatal asphyxia, abnormal glucose, calcium, magnesium metabolisms and iron deficiency.

SCREENING:

Evolution of diagnostic criteria for GDM over the years:

100-g OGTT

O'Sullivan and Mahan

The original diagnostic criteria, based on blood glucose testing before and hourly for 3 hours after 100-g glucose intake for GDM, were proposed by O'Sullivan and Mahan in 1964,based on a series of 752 women who underwent OGTT during pregnancy [30]. Means and standard deviations (SDs) were derived for each of four whole blood glucose values (defined as mean plus 1, 2, or 3 SD). O'Sullivan and Mahan decided that two

abnormal values would be needed for GDM diagnosis. The decision was based upon the desire to avoid misclassification due to laboratory error or the occasional individual with a single high glucose peak due to rapid absorption. Approximately 2% of pregnant women fulfilled the criteria of mean plus 2 SD, and these criteria became the basis for the diagnosis of GDM in the USA. The diagnostic criteria were applied to a separate group of 1,013 women who underwent 100-g OGTTs during their pregnancy. The risk of subsequent diabetes was 27% after a follow-up period of 8 years, when values at 2 SD were used as the diagnostic threshold during pregnancy.

National Diabetes Data Group criteria (NDDG)

In 1979, the National Diabetes Data Group criteria (NDDG) converted the whole blood glucose thresholds to the plasma values (approximately 14% higher as compared with the original O'Sullivan and Mahan criteria), in response to the general change in laboratory standards from whole blood to plasma or serum [31].

Carpenter and Coustan criteria.

Subsequently, new laboratory technology for glucose measurements using glucose oxidase and hexokinase methods, led to the formulation of

the Carpenter and Coustan criteria.The original O'Sullivan and Mahan criteria were established using Somogyi–Nelson technology. The Somogyi–Nelson method is not specific for glucose, and also measures approximately 0.27 mmol/L (5 mg/dL) of non-glucose reducing substances[32]. Glucose oxidase and hexokinase methods, on the other hand, measure only glucose. In 1982, Carpenter and Coustan used the glucose oxidase method to derive a set of criteria by first subtracting 5 mg/dl from O'Sullivan and Mahan's original values and then adding 14% to each (to account for the conversion from whole blood to plasma glucose values), and, finally, rounding to the nearest 5 mg/dl (0.27 mmol/L). This formulated the Carpenter and Coustan criteria [33].

75-g OGTT

WHO 1999 criteria

The initial recommendation for using 75-g OGTT in pregnancy was from the World Health Organization (WHO). The WHO used the same criteria for diagnosing diabetes both during and outside of pregnancy. This approach was criticized, as it ignored the physiological changes in carbohydrate metabolism that occurs during pregnancy. In 1999, the WHOlowered the threshold for FPG from 7.8 mmol/L (140 mg/dl) to 7.0 mmol/L (126 mg/dl) and recommended that pregnant women meeting the

criteria for diabetes mellitus or impaired glucose tolerance (IGT) be classified as having GDM [34].

DIPSI criteria

As per DIPSI criteria, 2-hour value ≥ 140 mg/dl after 75-gm OGTT done irrespective of fasting state is diagnostic of GDM [9]. It was found to have 100% sensitivity and specificity against WHO 1999 criteria [35].

IADPSG or WHO 2013 criteria

On this basis of HAPO study, the IADPSG recommended a fasting glucose level of 5.1 mmol/L (92 mg/dL), a 1-hour level of 10.0 mmol/L (180 mg/dL), or a 2-hour value of 8.5 mmol/L (153 mg/dL) as diagnostic for GDM.The IADPSG criteria have been endorsed by WHO, ADA, and the Endocrine Society of USA [36,37,38].

The HAPO study was a large, multicentre, multinational, epidemiologic study in which 23,316 women (30 times larger than the O'Sullivan cohort) underwent blinded 2-hour, three-sample, 75-g OGTTs at 24–32 weeks of gestation [39]. All women with a fasting plasma glucose (FPG) <105 mg/dl and 2 hours value up to 200 mg/dl were included. The HAPO clearly established a linear relationship between each of the glucose

values (fasting, 1 hour, and 2 hours) on OGTT and a broad range of predefined pregnancy outcomes. The primary outcomes in the HAPO study were the frequency of largeforgestationalage (LGA≥90th centile) babies, primary caesarean section, clinical neonatal hypoglycaemia, and neonatal hyperinsulinemia. All of these primary outcomes as well as secondary outcomes like foetal adiposity, preeclampsia, and birth trauma/shoulder dystocia were related to each of the maternal OGTT glucose results in a continuous fashion. The independent associations of hyperglycemia with pregnancy outcomes persisted after extensive adjustment for potential confounders including maternal body mass index (BMI), age, height, mean arterial pressure, and parity.

In 2010, the IADPSG recommended the establishment of new diagnostic criteria for GDM based on data from the HAPO study [4]. The diagnostic thresholds that were decided by the IADPSG consensus panel were based on glucose levels that gave 75% increased risk of birth weight ≥ 90th centile, cord C peptide ≥90th centile, and percentage body fat ≥90th centile as compared with mean glucose levels of women of HAPO cohort. On this basis, the IADPSG recommended a fasting glucose level of 5.1 mmol/L (92 mg/dL), a 1-hour level of 10.0 mmol/L (180 mg/dL), or a 2-hour value of 8.5 mmol/L (153 mg/dL) as diagnostic for GDM.

Limitations of HAPO study

The major limitations of HAPO study were

- No South Asian country was included in HAPO study.
- HAPO study was done in women enrolled at 24-32 weeks of gestation. We still donot have outcome-based criteria for early pregnancy.
- IADPSG criteria used internationally (adopted by WHO) have been derived based on HAPO study and HAPO study is based on short-term pregnancy outcomes.

IADPSG criteria

To convert the findings of the HAPO study into practical guidelines, the International Association of Diabetes in Pregnancy Study Groups (IADPSG) conducted a 2-day workshop that carefully examined the results of the HAPO study, and other studies consistent with the HAPO results [40]. A consensus panel was then formed and, after additional analyses on the HAPO study data, at another face- to-face meeting it developed the recommendations for the diagnosis of GDM. In a time frame that was concurrent with the HAPO study, two randomized control trials comparing standard obstetric care with active treatment for women with “mild GDM” were published [41,42]. In the trial conducted

by Landon et al. (the NHCHD- MFMU trial), pregnant women found positive on the glucose challenge test (GCT), and with an OGTT test result with two or three abnormal plasma glucose levels, but a fasting plasma glucose < 95 mg/dl were enrolled [41]. In the study reported by Crowther et al. (ACHOIS), participants were pregnant women with a positive glucose challenge test result and plasma glucose on OGTT < 140 mg/dl under fasting conditions, and between 140 and 198 mg/dl at 2 h after a 75 g glucose load [42]. The results of these trials show that the women treated with a controlled diet, plus insulin if the treatment goals were achieved, had better maternal and fetal outcomes in terms of a lower frequency of LGA babies, shoulder dystocia, gestational hypertension or preeclampsia than the women given standard prenatal care. Although patient selection for these trials differed from that of the HAPO study (i.e., with a two-step instead of a one-step diagnosis of GDM), the overlap in the characteristics (OGTT results, age, BMI) of the women taking part in the three studies justified considering the results of the two trials as complementary to the HAPO study findings [39]. A key strength of the IADPSG criteria lies in having used the HAPO and other data to derive thresholds of maternal glycemia associated with adverse neonatal outcomes in a large, blinded cohort prospectively tested with a 75 g OGTT. As for its potential weaknesses, the rise in the number of pregnant

women with GDM as a consequence of using the new criteria means that more women are treated with medical and obstetrical interventions, with a consequent increase in the medicalization of pregnancy, and in the related health care costs [43-45].

(Pros and Cons)

IADPSG criteria-Pros

- *Large cohort:* IADPSGcriteria has been derived from a blinded multinational cohort of 23,316 women enrolled from 15 centres from 9 countries, as against the traditional US definition of GDM based on the risk of maternal progression to diabetes mellitus in the postpartum period with the use of data that were derived from a small cohort of 752 women who were recruited by O'Sullivan in Boston in the late 1950s, which were later reanalysed to provide basis for current "2-step" testing.
- *Validation in terms of pregnancy outcomes*: All previous criteria had a common problem, namely, they were validated for predicting the future risk of diabetes only in the mother.The current 75-g IADPSG criteria have been devised after evaluating evidence that associates abnormal glucose tolerance in pregnancy with adverse perinatal outcomes.

- *Cost-effectiveness*: A large Spanish cohort study (St. Carlos study) assessed 1750 pregnant women using the two-step approach (Carpenter and Coustan criteria, CC), and 1526 pregnant women using the one-step (IADPSG) criteria. The results showed that the latter one-step approach generated a higher frequency of GDM than the two-step method (35 vs 10.6%), but was associated with a lower rate of adverse pregnancy outcomes (gestational hypertension—14.6%, $p < 0.021$; pre- maturity—0.9%, $p < 0.039$; Caesarean section—23.9%, $p < 0.002$; SGA—6.5%, $p < 0.042$; LGA—20%, $p < 0.004$; and admission to neonatal intensive care unit -24.4%, $p < 0.001$) [45]. Using the IADPSG criteria did not change the proportion of women needing insulin therapy to achieve good metabolic control. These data thus confirm the benefits of adopting the IADPSG criteria, in terms of maternal and fetal outcomes, in a setting of "standard care", with no overtreatment of patients.
- *Adverse outcomes even at values missed by other criteria(s)*:A well-conducted retrospective study compared the accuracy of different GDM screening procedures and diagnostic criteria (NICE, ADA, Irish, IADPSG) in the pregnant women taking part in the ATLANTIC DIP program [46]. The results showed that IADPSG

criteria enabled more cases of GDM to be diagnosed (prevalence 12.4%). When the NICE, Irish and ADA guidelines were applied, 20, 16 and 5% of the cases of GDM identified by the IADPSG criteria would have been overlooked because the women concerned had no risk factors. These women, nonetheless, had more adverse pregnancy outcomes than the women with a normal glucose tolerance. These findings provide a strong argument in favor of adopting the IADPSG detection strategy and diagnostic criteria

- *Reflects true background picture of prediabetes/ overweight/ obesity*:The increase in GDM prevalence reflects the increasing background prevalence of prediabetes/diabetes in reproductive age group as reflected from figures in USA and even from India [47, 48].
- *Opportunity to improve long term outcomes for mother and her offspring:* The recent publication of the HAPO Follow up study provides a long-term view (median follow up of 11.4 years) of the maternal and offspring consequences of pregnancy hyperglycemia [24]. HAPO FUS included 4747 mothers and 4834 infants from the original study, drawn from 10 of the 15 initial HAPO Field Centers. Overall, 52.2% of mothers with GDM, based on IADPSG criteria, who were blinded and untreated during their index

pregnancy experienced prediabetes (composite of impaired glucose tolerance and impaired fasting glucose) or type 2 diabetes mellitus at follow up as compared with 20.1% of those with normoglycemia. Thus, a diagnosis of GDM based on IADPSG criteria at the index pregnancy carried a very strong risk for future metabolic abnormalities. IADPSG GDM in the mother was also associated with offspring overweight or obesity (39.5% vs 28.6%), with a stronger trend for obesity alone (19.1% vs 9.9%). The data suggests an opportunity to improve long term outcomes for both mother and her offspring.

- IADPSG criteria adopted by WHO 2013 and have been endorsed by ADA, and the Endocrine Society of USA.

IADPSG criteria-Cons

- The diagnostic thresholds that were decided by the IADPSG consensus panel are based on glucose levels that give 75% increased risk of birth weight ≥ 90th centile, cord C peptide ≥90th centile, and percentage body fat ≥90th centile as compared with mean glucose levels of women of HAPO cohort [40].Since these are not hard clinical outcomes, the criteria need evaluation for

providing benefits for more clinically relevant outcomes, especially in resource constrained settings.

- IADPSG recommended any one value or more; a fasting glucose level of 5.1 mmol/L (92 mg/dL), a 1-hour level of 10.0 mmol/L (180 mg/dL), or a 2-hour value of 8.5 mmol/L (153 mg/dL) as diagnostic for GDM.The OGTT has poor reproducibility with about 25% of patients having a negative test result after a previous positive result [49]. In other words, the stricter the threshold values the more patients will be diagnosed by chance as having GDM.
- A recent study published in Lancet Diabetes have proposed even lower thresholds for South Asian women for diagnosis of GDM as compared to IADPSG criteria [50].Bradford study used data (including results of a 26–28 week gestation oral glucose tolerance test) from a prospective study that recruited women attending the antenatal clinic at the Bradford Royal Infirmary, UK, between 2007 and 2011 [50]. It studied the association between fasting and 2 h post-load glucose and three primary outcomes (LGA [defined as birthweight >90th percentile for gestational age], high infant adiposity [sum of skinfolds >90th percentil], and caesarean section). It established fasting and post-load glucose thresholds that equated to an OR of 1·75 for LGA and high infant adiposity in

each group of women to identify ethnic-specific criteria for diagnosis of gestational diabetes. Of 10353 eligible pregnancies, 4088 women were white British, 5408 were south Asian, and 857 were of other ethnic origin. A fasting glucose concentration of 5·4 mmol/L or a 2 h post-load level of 7·5 mmol/L identified white British women with 75% or higher relative risk of LGA or high infant adiposity; in South Asian women, the cut-offs were 5·2 mmol/L or 7·2 mml/L; in the whole cohort, the cut-offs were 5·3 mmol/L or 7·5 mml/L. Their data supported the use of lower fasting and post-load glucose thresholds to diagnose gestational diabetes in South Asian than white British women. They also suggested that diagnostic criteria for gestational diabetes recommended by IADPSG might underestimate the prevalence of gestational diabetes compared with their criteria, especially in South Asian women.

Therefore, given the concerns in both directions with cut-offs proposed by IADPSG criteria in South Asian women, we may need to derive newer cut-offs for diagnosis of GDM in South Asia.

DIPSI criteria (Pros and Cons)

DIPSI criteria-Pros

- Non-fasting test: Convenient, as it does not require fasting and only single plasma glucose post 2 hours of 75 gm glucose challenge is required [34]. All diagnostic criteria require women to be in the fasting state, but most often pregnant women do notcome for antenatal check up in the fasting state due to the prevailing belief that fasting for long hoursduring pregnancy is not good. Even if women are fasting, the long commute to a health facility andsubsequent wait for blood collection due to overcrowding or unsuitable clinic time makes itinconvenient for women to remain fasting for long hours [51]. Moreover, there is increased likelihood ofvomiting when 75 g glucose load is administered to fasting pregnant women necessitating repeat test ona subsequent day.
- *Lower 2-hour cut-off:*Using the2-hour glucose cut-off value of 153 mg/dl (based on an odds ratio of 1.75 for adverseoutcomes derived from HAPO data) as per IADPSG recommendation may not be as efficient inidentifying women at risk for fetal overgrowth as those identified by having a 2-hour glucosecorresponding to a slightly lower odds ratio e.g., 1.5. The latter corresponds to the older WHO criteria 2hr. value of 140 mg/dl. This is important in the developing

countries particularly in SouthAsia where women are relatively small and a larger baby may pose greater obstetric risk [51, 52].

- *Poor contribution of isolated fasting hyperglycemia:* In the HAPO cohort from Asian centers (Hong Kong, Thailand and Singapore), isolated fasting hyperglycemia contributed very little to the diagnosis of GDM [39,53].

DIPSI criteria-Cons

- Misses women with GDM who can have only fasting hyperglycemia. Current meta-analysis suggests that upto 70% women can be diagnosed based on fasting hyperglycemia alone in India using IADPSG criteria [54].
- The DIPSI criteria are not based on pregnancy related outcomes.
- Poor sensitivity (28%) against IADPSG criteria [55].
- DIPSI criteria were derived from WHO 1999 criteria [35]. WHO has said in its new guidelines in 2013 that WHO 1999 criteria were not evidence based and dropped it, and adopted IADPSG criteria [36]. Therefore, there is need to validate DIPSI criteria against IADPSG / WHO 2013 criteria for pregnancy outcomes.

Management of GDM

Guiding Principles

All Pregnant women who test positive for GDM for the first time should be started on Medical Nutrition Therapy (MNT) and physical exercise for 2 weeks. The woman should walk/exercise for 30 mins a day. After 2 weeks on MNT and physical exercise, 2 hrs PPBS (post meal) should be done. Thus, GDM is managed initially with MNT and physical exercise and if it is not controlled with MNT (lifestyle changes), Metformin or Insulin therapy is added to the MNT.

If 2hr PPBS is < 120 mg/dL, repeat test as per high risk pregnancy protocol i.e. to undertake 8 tests (4 regular tests and 4 additional). It is recommended to conduct at least one test every month during 2nd and 3rd trimester. More follow-up tests can be done as recommended by the treating physician.

If 2hr PPBS is ≥120 mg/dL, medical management (metformin or insulin therapy) to be started as per guidelines

Medical Nutrition Therapy (MNT)

Principles of MNT

Healthy eating during pregnancy

All pregnant women with GDM should get Medical Nutrition Therapy (MNT) as soon as diagnosis is made. MNT for GDM primarily involves a carbohydrate controlled balanced meal plan which promotes,

- Optimal nutrition for maternal and fetal health.
- Adequate energy for appropriate gestational weight gain .
- Achievement and maintenance of normoglycemia.

The importance of the individualised nutrition assessment in GDM Nutrition assessment in GDM should be individualised to allow an accurate appraisal of the woman's nutritional status. This assessment includes defining her Body Mass Index (BMI) or percentage of desirable pre-pregnancy body weight and optimal pattern of weight gain during pregnancy.

Recommended daily nutrition:

An addition of 350 kcal can be made to the energy requirement calculated as for the adults.

Energy requirement as per level of day to day activity:

Level of activity	Energy requirement during pregnancy	Total energy requirement
Sedentary work	1900+350	2250
Moderate work	2230+350	2580
Heavy work	3850+350	3200

Energy requirement based on the BMI(body mass index)

Weight category	BMI (kg/m2)	Energy requirement (kcal/day)
Under weight	<18.5	Energy requirement as per level of activity +500 kcal/day
Normal weight	18.5-22.9	Energy requirement as per level of activity
Over weight	23-24.9	Energy requirement as per level of activity
Obese	>25	Energy requirement as per level of activity-500kcal/day

In obese women it is better to advice moderate caloric restriction than that of hypocaloric diet,since it may adversely impair the fetal growth and may often result in ketosis.

SELECTION OF NUTRITION:

CARBOHYDRATES

- The type, amount and frequency of carbohydrate intakes is directly reflected as in blood sugar readings.
- Splitting the carbohydrate meals over 3 small meals and 2-3 mid meal snacks rather than taking 3 large meals helps to prevent sudden postprandial hyperglycaemia.Split meal plan maintains the blood glucose level in a plateau.
- Complex carbohydrates (like whole grain cereals, oats, bajra, jowar, ragi, whole pulses, vegetables and fruits with skin) should always be over the simple carbohydrates like foods with added sugars/honey, sweets, cakes, soft drinks, pizza etc.

FAT

- Saturated fat intake should be less than 10% of the total calorie intake.
- Dietary cholesterol should be less than 300mg/do.
- Source of saturated fat include- ghee, butter, coconut oil, red meat, organ meat, full cream Milk.

PROTEIN

- Protein requirement in pregnancy is increased to meet the demands of the fetus for its growth.
- Recommended daily allowance for protein 23 g/day. At least 3 servings of protein per day is recommended.
- Source of protein include: Milk and milk products, egg, fish, chicken, pulses (dal), nuts etc.

FIBER

High fibre food especially the soluble fibre helps in controlling the postprandial blood glucose.

It delays the gastric emptying, retards the entry of glucose into blood stream and thus lessens the postprandial blood glucose.

Sources of high fibre include, flax seed, psyllium husk, oat bran, legumes(dried beans of all kinds, peas, lentils)

ORAL HYPOGLYCEMIC MEDICATIONS

Metformin

Metformin is a biguanide.

Mechanism of action:

1) decreases hepatic glucose production
2) Decreases intestinal absorption of glucose
3) Increases peripheral uptake and utilization of glucose.25
4) decreases insulin resistance.

Metformin alone or in combination with insulin has been reported to have similar safety and efficacy to insulin for the treatment of GDM.(56,57)

Advantage over sulfonylurea :

Doesn't cause maternal hypoglycaemia.

Doesn't cause fetal hyperinsulinemia.

A recent randomized clinical trial of 160 women with GDM reported that metformin monotherapy resulted in comparable maternal glycemic control as with insulin.29 With a median daily dose of 1500 mg (range 1000-2500 mg), mean fasting blood glucose < 95 mg/dL was achieved in 74% of subjects receiving metformin vs 79% with insulin. Mean postprandial glucose < 120 mg/dL was achieved in 81% of both metformin and insulin treated groups. In this study, 14% of women in the metformin monotherapy group eventually required supplementation with

insulin. In another study (n=363), Rowan et al. reported that 46% of patients with GDM receiving metformin monotherapy experienced treatment failure requiring supplemental insulin.6However, both fasting blood glucose and mean 2-hour postprandial glucose concentrations were comparable in metformin and insulin streatment groups.6

Glyburide

Glyburide is a second-generation sulfonylurea .

Mechanism of action: Enhancing insulin secretion.

Glyburide is FDA approved for the treatment of patients with type 2 diabetes mellitus.(59)

Early use of first-generation sulfonylureas such as chlorpropamide 8 and tolbutamide 9 resulted in concerns regarding teratogenicity, neonatal hypoglycemia, and fetal hyperinsulinemia, thus limiting their use during pregnancy. Maternal hyperglycemia as well as moderate to high placental transfer and the prolonged fetal/neonatal half-life of the firstgeneration sulfonylureas are the likely causes of these adverse events.

Several randomized controlled studies have compared glyburide to insulin for the treatment of GDM. Glyburide was shown to be comparable to insulin in controlling maternal glucose and decreasing the incidence of macrosomia.

Langer et al. (n=404) reported that 82% of the subjects achieved glycemic control (self-monitored fasting glucose • 95 mg/dL) with glyburide (n=165) compared to 88% with insulin (n=179).3In a later study, the authors compared efficacy of glyburide and insulin for treatment of women with GDM, stratified for severity of disease (fasting plasma glucose • 95 mg/dL vs > 95 mg/dL).4 The authors found that both glyburide and insulin were equally effective in treating GDM at both severity levels (n=404).4A smaller randomized study that compared insulin to glyburide in Asian Indian women with GDM (n=23) reported no significant differences in glycemic control (mean 2-hour postprandial glucose concentrations) between insulin and glyburide treatment.(51).

Indicators for success of treatment with glyburide:

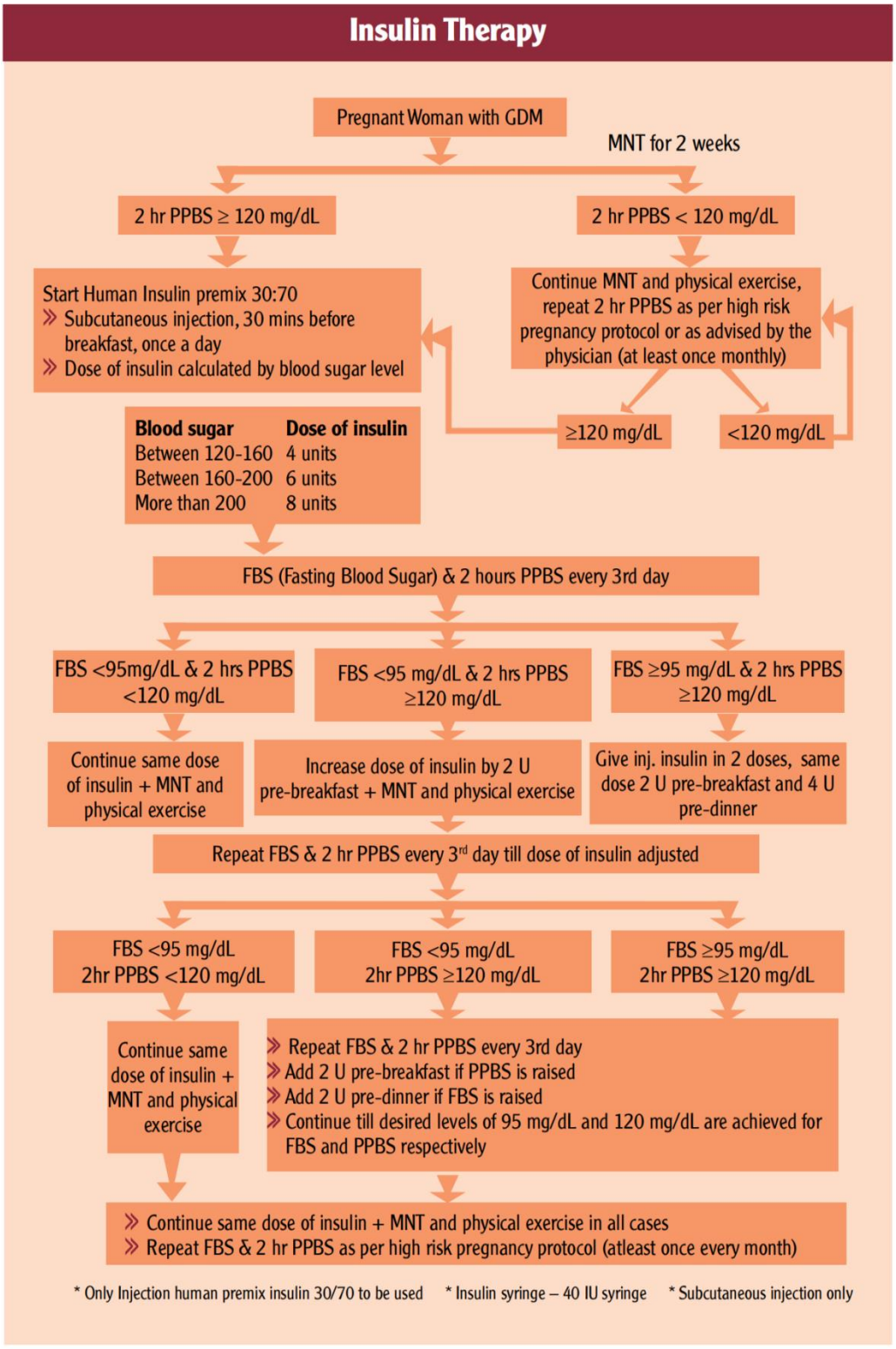
1. Gestational age at the time of delivery
2. Mean fasting blood glucose before initiating therapy.

INSULIN THERAPY:

Insulin pen or syringe can be provided to pregnant women for subcutaneous delivery of injection.

Insulin syringe : 40 IU syringe is to be used.

Insulin vials should not be exposed to direct heat/sunlight and are stable upto 25- 30 degree Celsius.



ANTEPARTUM MANAGEMENT

Recommended Antenatal visits

1) Booking visit –

   counselling

   Retinal assessment in women with prexisting diabetes

   Renal assessment in women with pre existing diabetes

   Measure HbA1c

   Confirm viability and dating.
2) At 16 weeks -offer retinal assessment at 16-20 weeks
3) At 20 weeks-Anomaly scan
4) At 28 weeks-to assess fetal growth and amniotic fluid volume

   Retinal assessment
5) At 32 weeks- fetal interval growth assessment
6) At 36 weeks- fetal monitoring, counselling regarding timing,mode and management of birth.
7) At 37-38+6 days- offer induction of labor or Caesarean section if indicated
8) At 38 weeks-fetal monitoring
9) At 39 weeks-advise termination of pregnancy in uncomplicated gestational diabetes before 40+6 days

INTRAPARTUM CARE

Timing and mode of birth

- Women with Type1 and type 2 diabetes and no other complications to have an elective birth by induced labor or (if indicated) Caesarean section,between 37 and 38+6 days of pregnancy.
- Consider elective birth before 37 weeks for women with type1 or type 2 diabetes who have metabolic or other maternal or fetal complications.
- Advise women with Gestational diabetes to give birth no later than 40 weeks plus 6 days.Offer elective birth by induced labour or if indicated by Caesarean section to women who have not given by this time.
- Consider elective birth before 40weeks plus 6 days for women with Gestational diabetes who have maternal or fetal complications.

POST-PARTUM MANAGEMENT

1. In the postpartum period, the insulin requirements drop and so the dosage of the medications will need to be changed. Most women with GDM can be taken off medications and managed on diet alone. It is important to maintain good glycemic control in the post partum period

2. Life-style modifications and diet are emphasised in the post partum period and at discharge.
3. Breast feeding is the preferred option for all GDM and pregestational diabetes women.
4. Along with nutritional and immunological advantages, breast feeding has been associated in the general population with a reduction in the rates of childhood obesity.

PRECAUTIONS DURING CAESAREAN

Elective caesarean for macrosomia is recommended in a diabetic pregnancy if the estimated fetal weight is >4.5kg (ACOG).

- After appropriate consent and blood availability, a light meal and night dose of insulin are given.
- Elective section of a diabetic patient should preferably be performed as the first case in the morning as the patient is fasting.
- Morning insulin dose is skipped and fasting glucose level should be recorded.
- If required sliding scale of insulin can be started and continued in the postoperative period.
- Severely obese may require thromboprophylaxis.

- Special precautions while performing the caesarean are adequate incision to allow
- delivery of the big baby, use of forceps to deliver high floating head, to check for and suture extensions of the uterine incision which may take place especially while performing a second stage caesarean.
- Postoperative glucose monitoring must be continued and patient must be mobilized as early as possible.

FOLLOW UP

Gestational diabetic women require follow up. Maternal glucose levels usually return to normal after delivery. Nevertheless, a FBS & 2 hr PPBS is performed on the 3rd day of delivery. Glucose tolerance test with 75g oral glucose is later performed at 6 weeks of delivery and if necessary repeated after 6 months and every year to determine whether the glucose tolerance has returned to normal or progressed.

The Cut offs for normal blood glucose values are:

- Fasting plasma sugar: ≥ 126 mg/dl,
- 75 g OGTT 2 hour plasma glucose-
- Normal: < 140 mg/dl,
- Impaired glucose tolerance: 140-199mg/dl,
- Diabetes: ≥ 200 mg/dl.

GDM recurs approximately in 50% of subsequent pregnancies. The future risk of developing diabetes for a gestational diabetic is twofold, if she becomes overweight. But maintaining ideal weight approximately halves the risk. The requirement of insulin in addition to diet to maintain euglycemia during the index pregnancy is also predictive of future diabetes.

CONTRACEPTIVE ADVICE

Patients must be counselled regarding contraception. Barrier methods are ideal. Progesterone only pills are also safe. Combined oral contraceptive pills may be best avoided, especially when diabetes mellitus is of a long duration but low dose OCPs can be used in well controlled diabetics. Intrauterine devices may predispose to infection. A diabetic patient may undergo tubal sterilization with precaution. Counselling the husband for vasectomy is also a good option.

Thus, diabetes management in pregnancy has evolved over the past years due to changing lifestyles and increase in maternal obesity and age at delivery. Due to this, a thorough knowledge regarding the best possible therapy for the patient is a must. Treatment has to be individualized.

STUDIES RELATED TO THE TOPIC

- In a study by Emmanuel et al,it was found that the mothers with Gestational diabetes (GDM) were four times more likely to have hypertensive disorders (p=0.04).They also found that the indications of Caesarean sections in mother with GDM were more likely to be due to big babies and obstructed labour.(60)
- In a cross sectional study by Zainab Groof et al,it has been found that the prevalence of GDM increased with maternal age and prepregnancy body mass index(BMI).GDM was positively associated with caesarean delivery (aOR= 1.76,95% CI: 1.17,2.66) and fetal macrosomia (aOR = 2.36,95% CI : 1.14,4.89).(61)
- Michelle,A.A.,Olayemi in their study in 694 women found that Gestational diabetes had a higher risk of composite adverse maternal outcome (ARR=1.58, 95% CI: 1.22, 2.04), caesarean delivery (ARR=1.67; 95%: 1.15, 2.44), pregnancy induced hypertension (ARR= 3.32; 95%: 1.55, 7.11), premature rupture of membranes (ARR= 1.83; 95%: 1.02, 3.27), antepartum hemorrhage (ARR= 2.10; 95%: 1.11, 3.98) and postpartum hemorrhage (ARR= 4.85; 95%:2.28, 10.30) compared to women without gestational diabetes mellitus.(62)
- Dr Sri Harsha Lanke,Dr Susmita DeviAgarwal,Dr Lakshmi Kumari Pavuluri in their study at Bhubaneshwar over a period of

2years,concluded that the major outcomes included 83(82.2%) women having gestational diabetes had underwent Cesarean Sections and had 4 macrosomic babies. The babies of these mothers also had the most tendencies to develop hyperbilirubinemia and accounted for a total of 19 NICU admissions with 2 stillbirths.(63)

- In a study by Goedegebure et al, 1386 women with GDM were studied in two cohorts as one diagnosed with WHO 1999 criteria and the other with 2013 criteria.The WHO 2013 cohort has high rate of spontaneous delivery.There were no differences between the cohorts regarding stillbirth, birth trauma,low Appar score and preeclampsia.(64)

METHODOLOGY

1) **Study design:**

Prospective observational study

2) **Study Centre :**

Institute of obstetrics and Gynecology, Egmore, Chennai.

3) **Study Participants:**

All singleton pregnant women attending OPD in Institute of obstetrics and Gynecology at Gestational age of 20-24weeks.

Inclusion criteria:

1. Pregnant women with singleton fetus inutero
2. Age 18 years and above
3. Gestational age 24-28weeks
4. Have undergone OGCT as per IADPSG criteria

Exclusion criteria

1. Early GDM [diagnosed at <24 weeks of gestation]
2. Women coming after 28 weeks
3. Overt or preexisting diabetes mellitus
4. Infection with HIV/Hep B/Hep C

5. Women having PCOS on T.Metformin /had used T.Metformin within 4 weeks of doing OGTT
6. Women uncertain of their LMP and with no USG estimation of Gestational age before 16 weeks

4) **Number of groups studied** :

Single group

Sampling:

Population: The study would include all antenatal women attending OPD in IOG

Sampling method : Random sampling

Sample size:

Sample size N = 3.84 x P x Q /d2

P = 20

Q = 100 – p = 80

d = 5 (absolute precision)

N= 3.84 x 20 x 80 / 5 x 5

= 6144 / 25 = 245

N = 245.

5) METHOD OF STUDY

a. The study participants will be enrolled as per inclusion and exclusion criteria.

b. Informed consent will be taken.

c. At initial prenatal visit fasting blood glucose will be taken and as recommended by IADPSG criteria,75G OGTT was performed.

d. The women with fasting blood glucose>/=126mg/dl,2hr blood glucose >/=200mg/dl are diagnosed with overt diabetes and will be excluded from the study.

e. Women with fasting plasma glucose >/=92 mg/dl but <126mg/dl,1 hr plasma glucose >/=180mg/dl,2 hr plasma glucose >/=153mg/dl are diagnosed as GDM and included in the study.

f. The management of GDM was consistent with standard clinical practice which consisted of dietary control,proper exercise and insulin therapy accordingly.

6) **OUTCOME**:

Maternal outcome variables include the occurrence of preterm delivery.

a. 1.requirement for caesarean section
b. 2.operative vaginal delivery
c. 3.Preeclampsia
d. 4.Miscarriage

7) **Fetal outcome**

a. Large for gestation
b. stillbirth/ IUD
c. clinically significant neonatal hypoglycemia
d. Respiratory distress syndrome
e. perinatal mortality
f. Birth injury /trauma

RESULTS AND OBSERVATIONS

The collected data were analysed with IBM SPSS Statistics for Windows, Version 23.0.(Armonk, NY: IBM Corp).To describe about the data descriptive statistics frequency analysis, percentage analysis were used for categorical variables and the mean & S.D were used for continuous variables.

Table: 1 Age distribution

Age distribution		
	Frequency	Percent
21 - 25 yrs	37	14.8
26 - 30 yrs	192	76.8
Above 30 yrs	21	8.4
Total	250	100.0

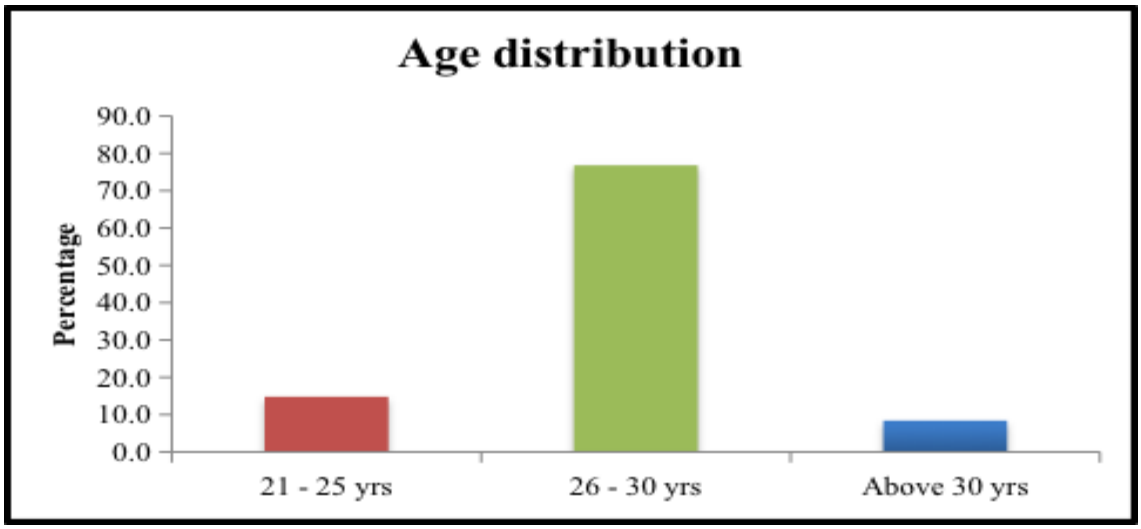


Figure: 1

The above table shows Age distribution were 14.8% is 21-25 years, 76.8% is 26-30 years, 8.4% is Above 30 years.

Table: 2 Parity distribution

Parity		
	Frequency	Percent
Multi	135	54.0
Primi	115	46.0
Total	250	100.0

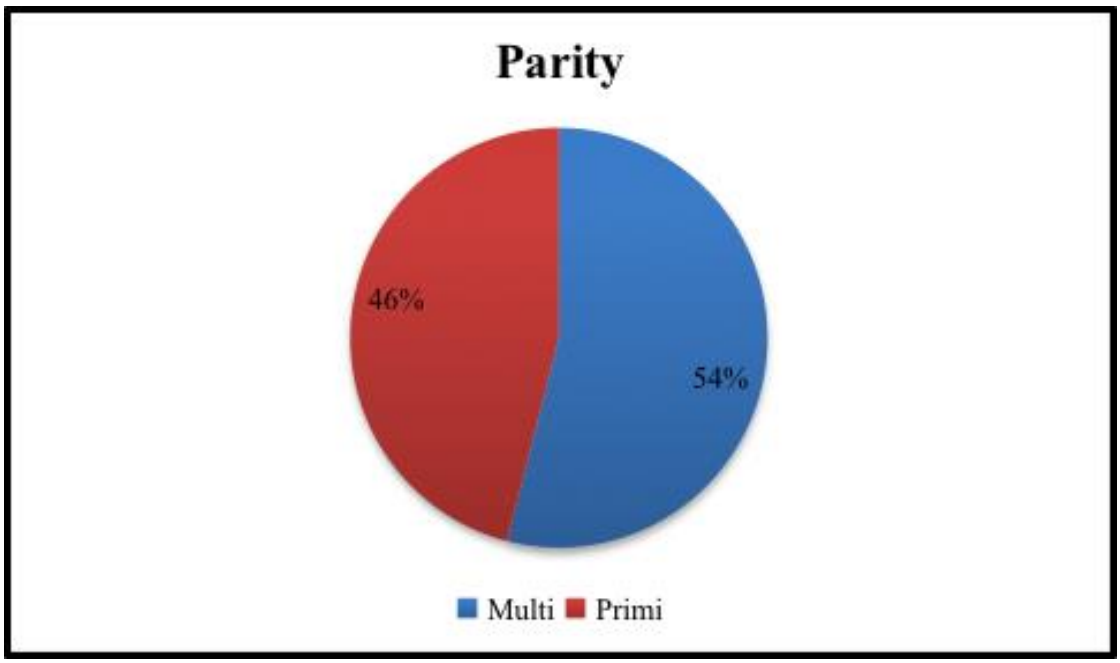


Figure: 2

The above table shows Parity distribution were 54.0% is Multi, 46.0% is Primi.

Table: 3 Gestational Hypertension distribution

GHTN		
	Frequency	Percent
Absent	175	70.0
Present	75	30.0
Total	250	100.0

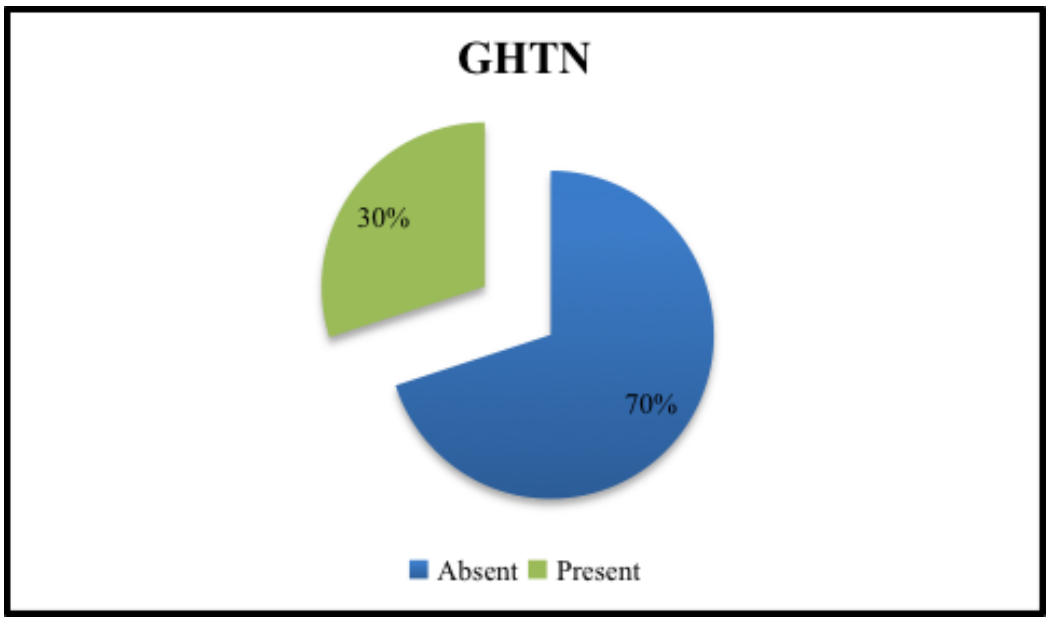


Figure: 3

The above table shows Gestational Hypertension distribution were 70.0% is Absent, 30.0% is Present.

Table: 4 Insulin requirement distribution

Insulin requirement		
	Frequency	Percent
Absent	134	53.6
Present	116	46.4
Total	250	100.0

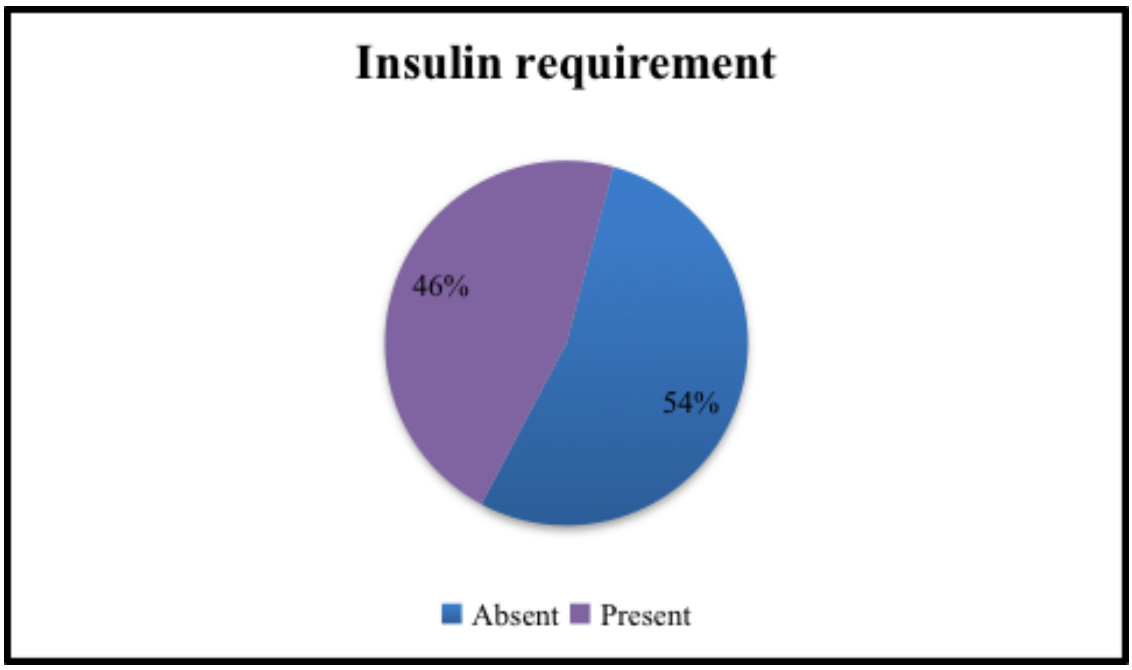


Figure: 4

The above table shows Insulin requirement distribution were 53.6% is Absent, 46.4% is Present.

Table: 5 Mode of delivery distribution

Mode of delivery		
	Frequency	Percent
Elective	25	10.0
Emergency	133	53.2
Natural labour	92	36.8
Total	250	100.0

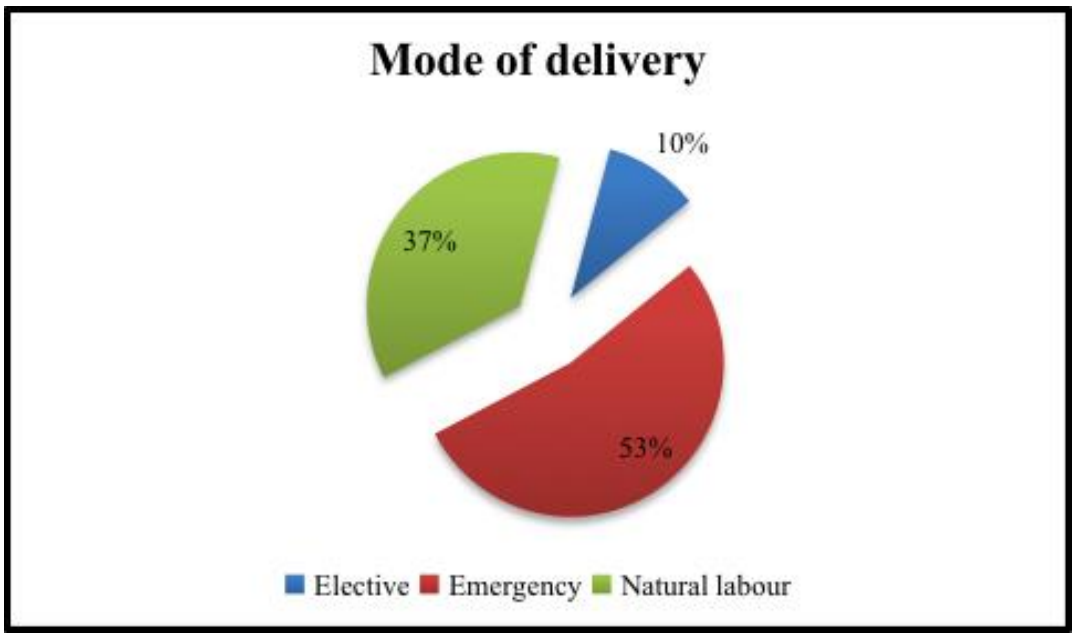


Figure: 5

The above table shows Mode of delivery distribution were 10.0% is Elective, 53.2% is Emergency, 36.8% is Natural labour.

Table: 6 Indication of primary LSCS distribution

Indication for primary LSCS		
	Frequency	Percent
Breech	4	5.0
CPD	27	33.8
FD	28	35.0
FI	20	25.0
Transverse lie	1	1.3
Total	80	100.0

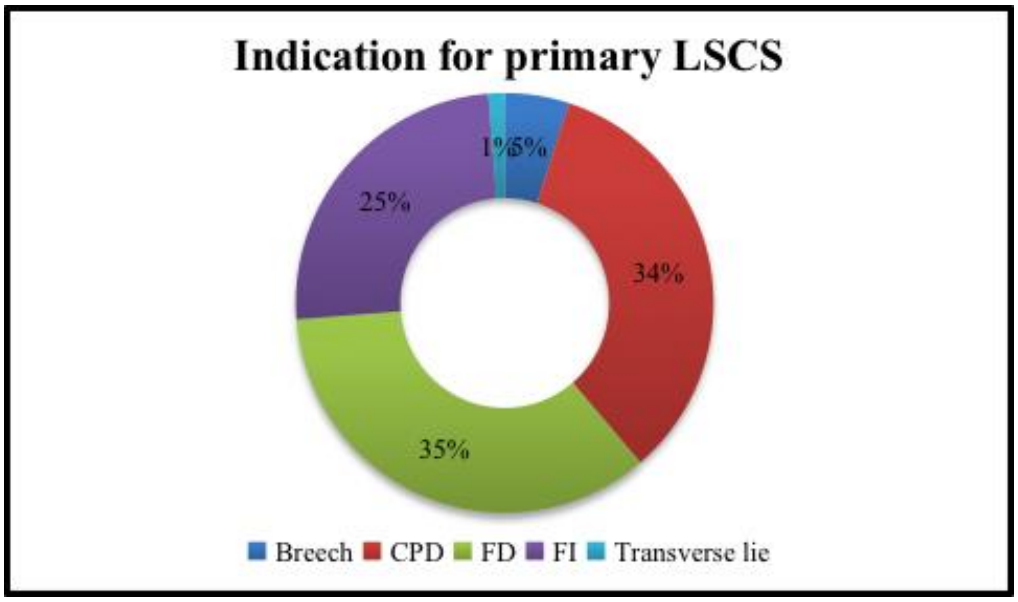


Figure: 6

The above table shows Indication for primary LSCS distribution were 5.0% is Breech, 33.8% is CPD, 35.0% is FD, 25.0% is FI, 1.3% is Transverse lie.

Table: 7 Baby gender distribution

Baby gender		
	Frequency	Percent
Boy	133	53.2
Girl	117	46.8
Total	250	100.0

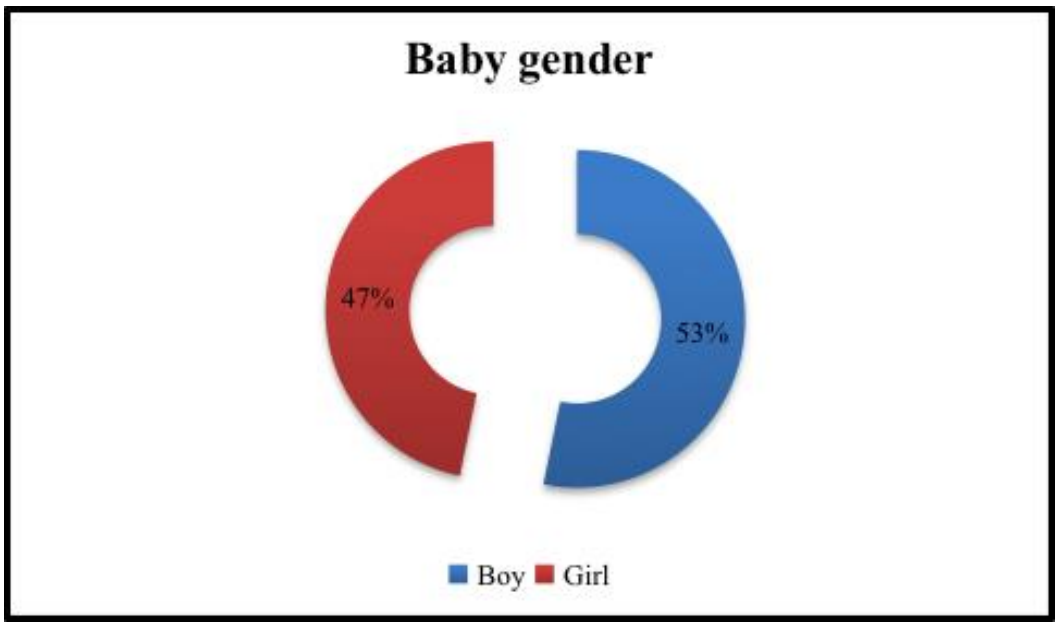


Figure: 7

The above table shows Baby gender distribution were 53.2% is Boy, 46.8% is Girl.

Table: 8 Live/IUD/Abortion distribution

Live/IUD/Abortion		
	Frequency	Percent
D	10	4.0
L	240	96.0
Total	250	100.0

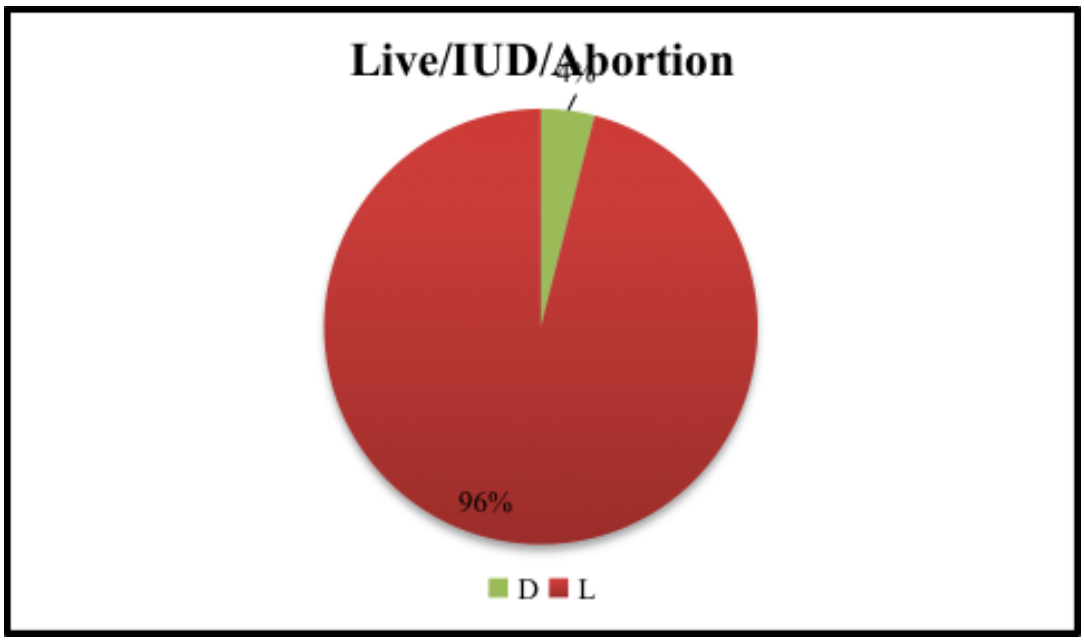


Figure: 8

The above table shows Live/IUD/Abortion distribution were 4.0% is D, 96.0% is L.

Table: 9 APGAR distribution

APGAR		
	Frequency	Percent
6/10,9/10	4	1.6
7/10,9/10	8	3.2
8/10,9/10	228	91.2
Total	250	100.0

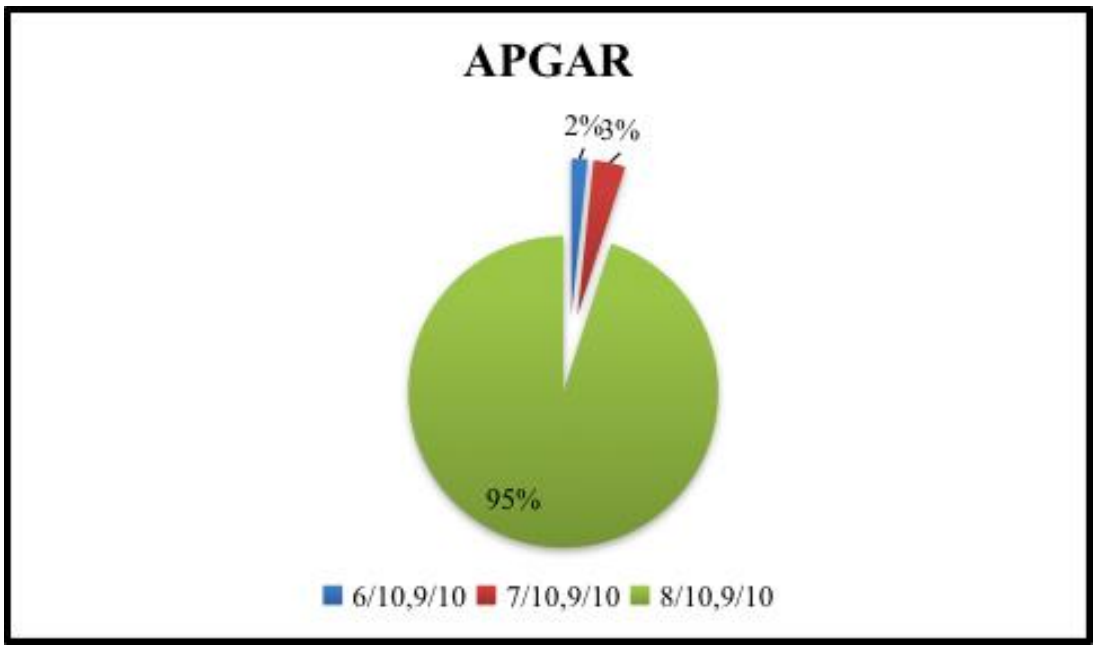


Figure: 9

The above table shows APGAR distribution were 1.6% is 6/10,9/10, 3.2% is 7/10,9/10, 91.2% is 8/10,9/10.

Table: 10 NICU admission distribution

NICU Admission		
	Frequency	Percent
No	158	63.2
Yes	92	36.8
Total	250	100.0

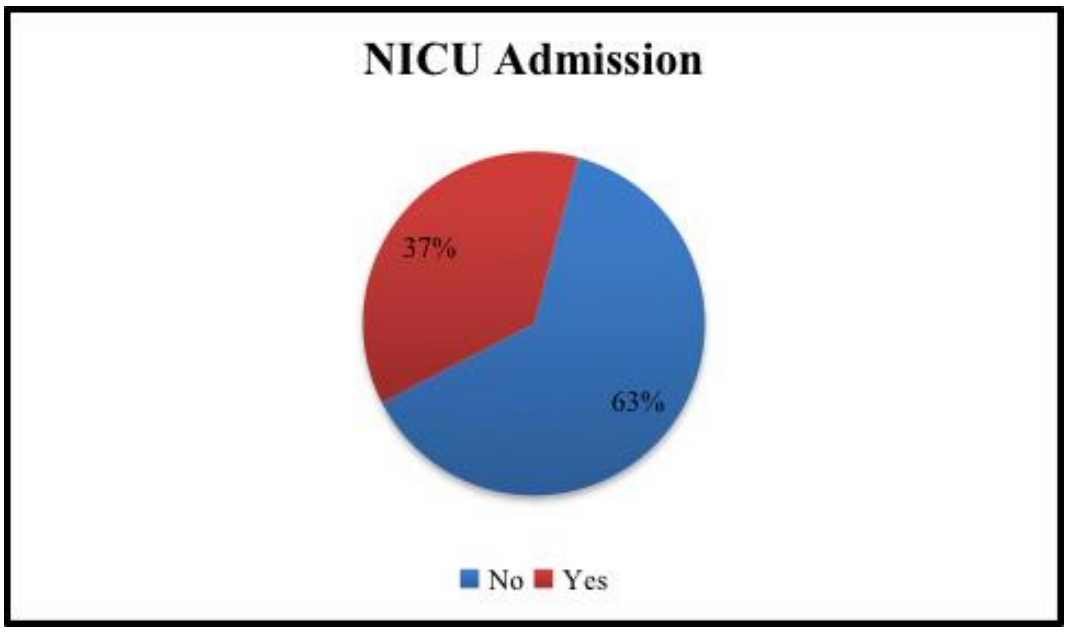


Figure: 10

The above table shows NICU Admission distribution were 63.2% is No, 36.8% is Yes.

Table: 11 Polyhydramnios distribution

Polyhydramnios		
	Frequency	Percent
A	220	88.0
O	5	2.0
P	25	10.0
Total	250	100.0

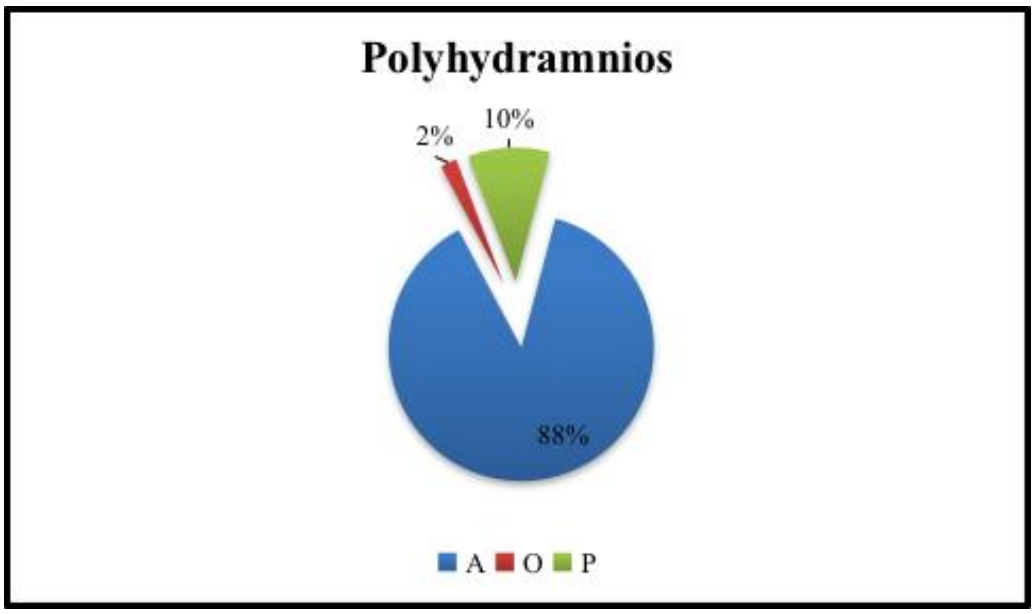


Figure: 11

The above table shows Polyhydramnios distribution were 88.0% is A, 2.0% is O, 10.0% is P.

Table: 12 PPH distribution

PPH		
	Frequency	Percent
No	240	96.0
Yes	10	4.0
Total	250	100.0

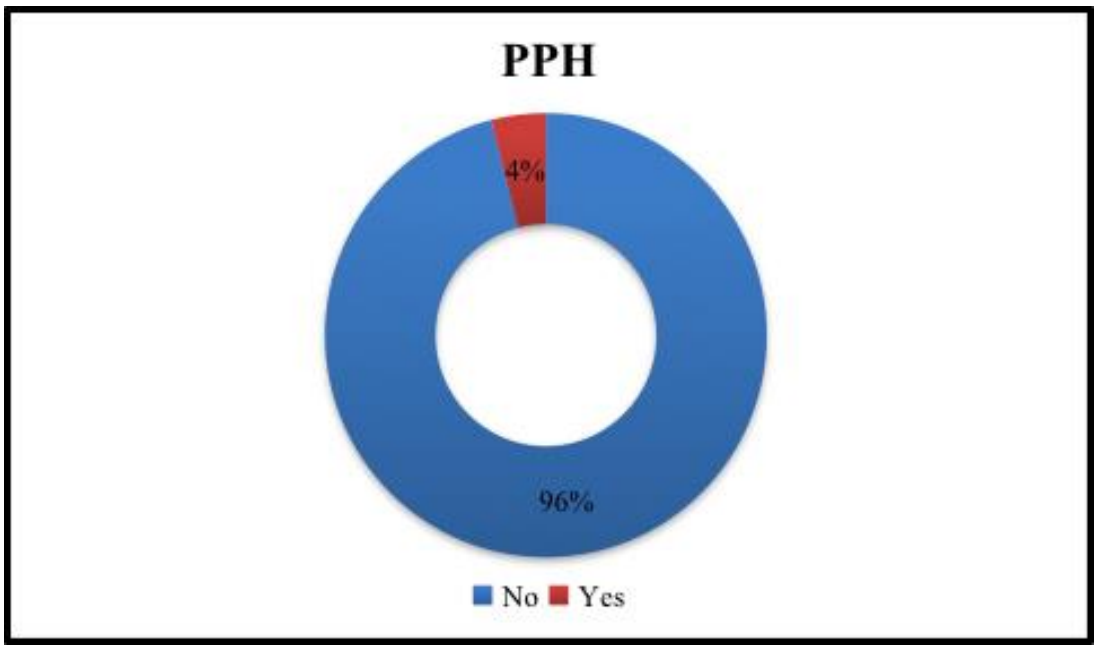


Figure: 12

The above table shows PPH distribution were 96.0% is No, 4.0% is Yes.

Table: 13 Term distribution

Term		
	Frequency	Percent
Preterm	61	24.4
Term	189	75.6
Total	250	100.0

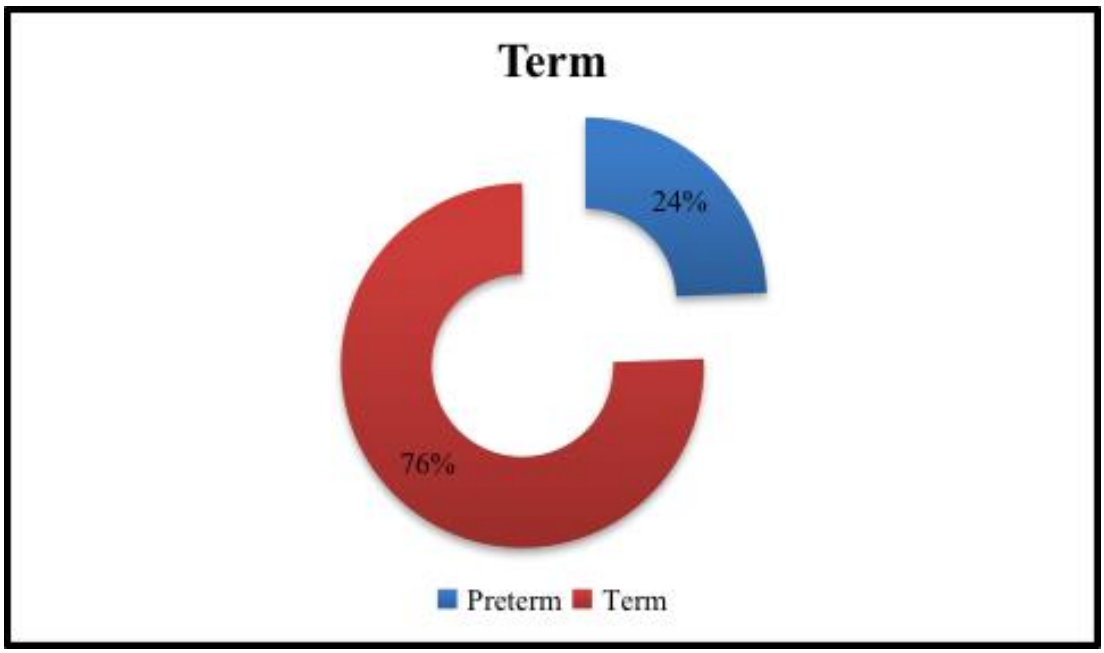


Figure: 13

The above table shows Term distribution were 24.4% is Preterm, 75.6% is Term.

Table: 14 IUGR distribution

IUGR		
	Frequency	Percent
FGR	5	2.0
AGA	245	98.0
Total	250	100.0

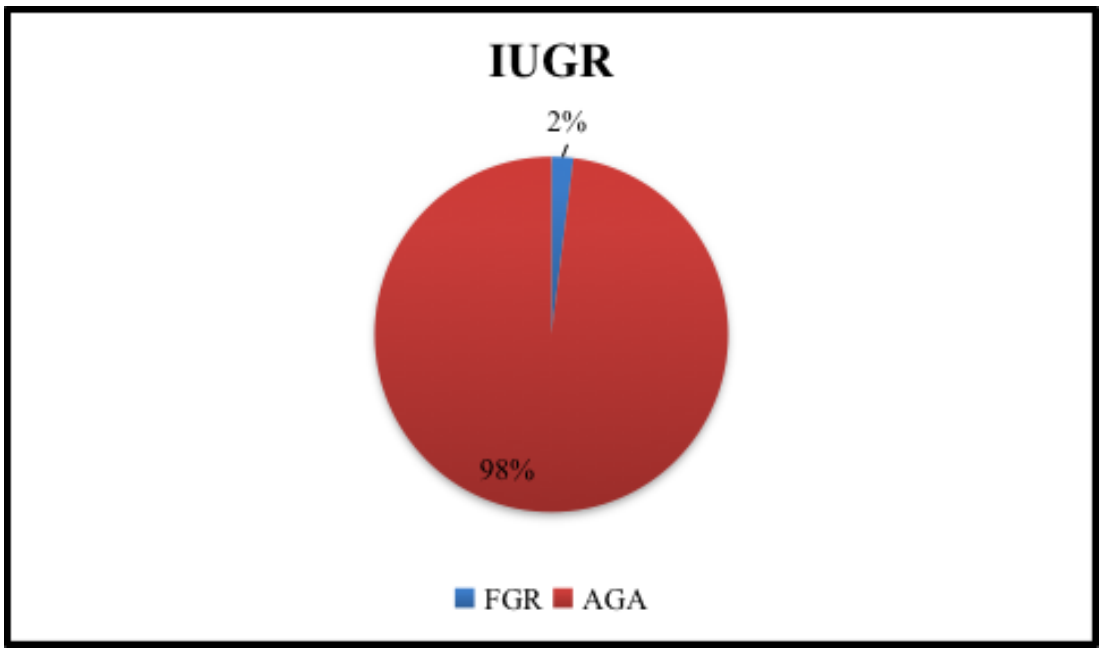


Figure: 14

The above table shows IUGR distribution were 2.0% is FGR, 98.0% is AGA.

Table: 15 Body Mass Index distribution

BMI distribution		
	Frequency	Percent
Normal	143	57.2
Overweight	61	24.4
Obesity Class I	33	13.2
Obesity Class II	12	4.8
Obesity Class III	1	.4
Total	250	100.0

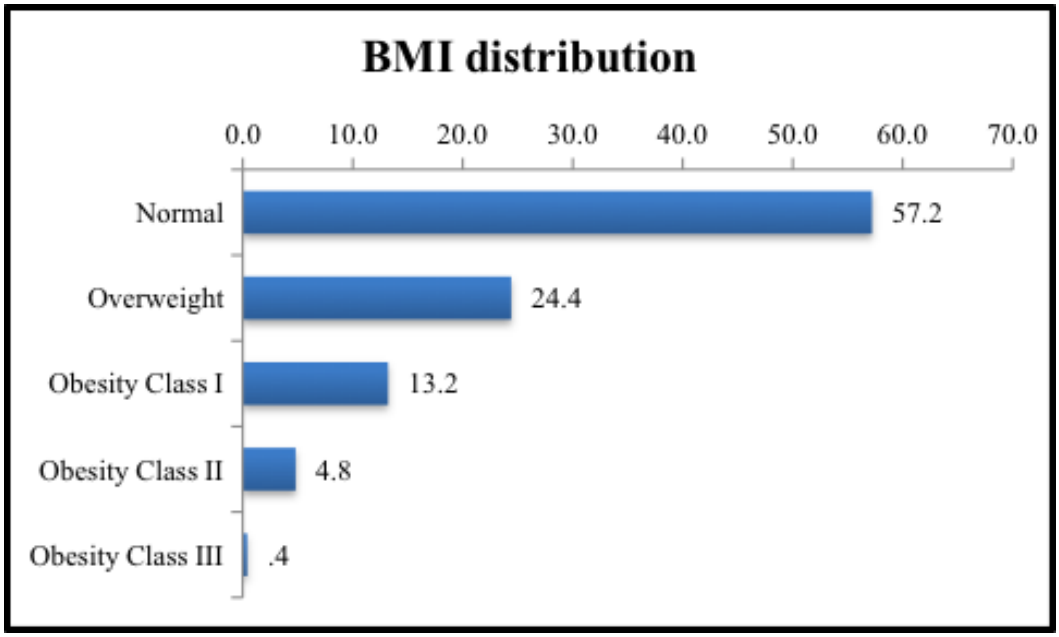


Figure: 15

The above table shows Body Mass Index distribution were 57.2% is Normal, 24.4% is Overweight, 13.2% is Obesity Class I, 4.8% is Obesity Class II, 0.4% is Obesity Class III.

Table: 16 Birth weight/Kilograms distribution

Birth weight distrbution		
	Frequency	Percent
1.5 - 2 kgs	8	3.3
2 - 2.5 kgs	46	18.8
2.5 - 3 kgs	80	32.7
3 - 3.5 kgs	85	34.7
> 3.5 kgs	26	10.6
Total	245	100.0

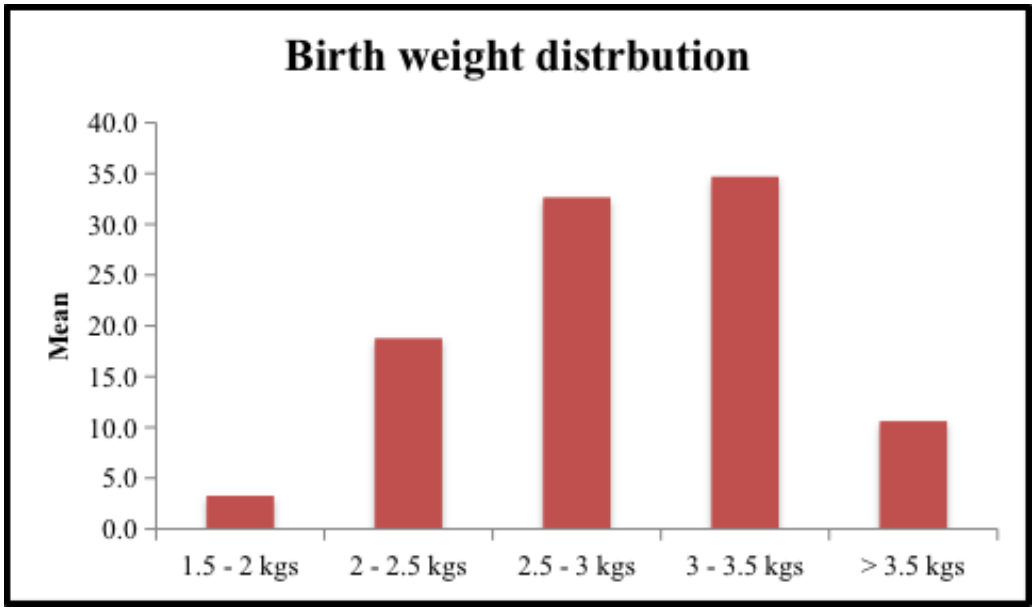


Figure: 16

The above table shows Birth weight distribution were 3.3% is 1.5-2 kgs, 18.8% is 2-2.5 kgs, 32.7% is 2.5-3 kgs, 34.7% is 3-3.5 kgs, 10.6% is > 3.5 kgs.

Table: 17 Descriptive statistics distribution

Descriptive Statistics					
	N	Minimum	Maximum	Mean	SD
Age	250	24.0	36.0	27.8	2.3
Height	250	140.0	166.0	150.9	6.1
Weight	250	40.0	98.0	58.5	12.7
BMI	250	20.0	42.4	25.6	4.8
GA	250	32.00	40.43	37.2	1.1
Birth weight	245	1.540	3.950	2.9	0.5

The above table shows descriptive statistics of continuous variables.

SUMMARY

1. Age distribution were 14.8% is 21-25 years, 76.8% is 26-30 years, 8.4% is Above 30 years.
2. Parity distribution were 54.0% is Multi, 46.0% is Primi.
3. Gestational Hypertension distribution were 70.0% is Absent, 30.0% is Present.
4. Insulin requirement distribution were 53.6% is Absent, 46.4% is Present
5. Mode of delivery distribution were 10.0% is Elective, 53.2% is Emergency, 36.8% is Natural labour.
6. Indication for primary LSCS distribution were 5.0% is Breech, 33.8% is CPD, 35.0% is FD, 25.0% is FI, 1.3% is Transverse lie.
7. Baby gender distribution were 53.2% is Boy, 46.8% is Girl.
8. Live/IUD/Abortion distribution were 4.0% is D, 96.0% is L.
9. APGAR distribution were 1.6% is 6/10,9/10, 3.2% is 7/10,9/10, 91.2% is 8/10,9/10.
10. NICU Admission distribution were 63.2% is No, 36.8% is Yes.
11. Polyhydramnios distribution were 88.0% is A, 2.0% is O, 10.0% is P.
12. PPH distribution were 96.0% is No, 4.0% is Yes.

13. Term distribution were 24.4% is Preterm, 75.6% is Term
14. IUGR distribution were 2.0% is FGR, 98.0% is AGA.
15. Body Mass Index distribution were 57.2% is Normal, 24.4% is Overweight, 13.2% is Obesity Class I, 4.8% is Obesity Class II, 0.4% is Obesity Class III.
16. Birth weight distribution were 3.3% is 1.5-2 kgs, 18.8% is 2-2.5 kgs, 32.7% is 2.5-3 kgs, 34.7% is 3-3.5 kgs, 10.6% is > 3.5 kgs.

DISCUSSION

AGE (refer to table no1)

The present study showed the peak maternal age group of incidence to be between 26-30 years. This finding is in accordance with the study done by Hedderson et al (2003) which had maximum incidence between 25-34 years43.Landon Mark B (2009) study had similar finding of 29 years53.Jacobson John D and Cousins Larry(1989) study also had similar observation of 28 years38. Langer Oded et al 2005) study found mean age of GDM to be 27years50.

PARITY (refer to table no 2)

The present study showed the highest incidence of GDM to be in Multigravida gravidae (54%). This is similar to the finding of Jacobson John D and Cousins Larry (1989)38, which showed it to be >2 parity.

PREMATURITY (refer to table no 13)

The present study showed the incidence of premature labour to be 24.4%. Similar observation 13.9% was made by Ostlund Ingrid et al (2003)45. 7.4% preterm labour was seen in B Dittakaran et al study done in 2006. The incidence of prematurity was high in this study. Coexisting PIH and IUGR with iatrogenic termination might have influenced this

outcome. Gillmer MDG and Hurley PA have mentioned the risk of prematurity to be 20%54 .61

GESTATIONAL HYPERTENTION (GHTN) (refer to table no 3)

The present study found associated PIH in 30% of cases.This is in accordance with many studies done in past.Pennison Erin H and Egerman Robert S (2001) found an incidence of PIH to be 20.9% 42. Gajjar F Maitra N K (2005) found this to as high as 60%49.

POLYHYDRAMNIOS (refer to table no 11)

The present study showed the incidence of polyhydramnios to be 10%. This is relatively high compared to other studies. Jacobson John D et al (1989) found polyhydramnios in 2.1%38 cases while Hedderson Monique M et al (2003) found it in only 0.7% cases43.

IUFD /STILLBIRTH (refer to table no 8)

The present study showed the incidence of intrauterine fetal death to be 4% The stillborn baby was born at 28 weeks. All the patients belonged to insulin group. Odar Emmanuel et al (2004) found 16.7% stillbirth in their study4. Langer Oded et al (2005) observed 5.4%

stillbirth50. The study done by B Dittakaran et al (2006) showed it to as low as 0.6%52.

IUGR (refer to table no 14)

The present study found the incidence of intrauterine growth restriction to be 2%. This case also had PIH. Similar incidence of 1.3% was observed in the study done by Hedderson Monique M et al (2003)43. Gajjar F, Maitra N K (2005) had IUGR in 10% of the cases.The presence of vasculopathy and/ or PIH contribute for the fetal growth restriction.

MODE OF THERAPY (refer to table 4)

The present study had 53.6% of the cases on diet therapy only while the rest 46.4% were on both diet and insulin therapy. This is similar to the study done by Garner Peter et al(1997) where 50% were on insulin41. Jacobson John D et al(1989) had only 13 out of 97 cases on insulin38. Adams Kristina M et al (1997) had 76 cases on insulin and 297 cases only on diet therapy40.This up holds the fact that less than 50% GDM cases require insulin therapy.

MODE OF DELIVERY (refer to table no 5)

36.8% of the cases delivered vaginally and the rest by LSCS in this study. There were no cases of instrumental delivery. Of the Caesarean sections done 10% were done electively and about 53% were done for emergency indications. The incidence of LSCS is relatively high in this study. Previous LSCS was seen in 31.2% cases. Among the primary Caesarean section done,Cephalopelvic disproportion contributed for 33.8% of LSCS. Failed induction contributed for 25% and fetal distress was the indication in 35% . 1.3% had malpresentation. Adam's Kristina M et al(1997) noted similar high rate of LSCS - 41% cases in insulin treated group and 23% in the diet treated group (total:64%)40. Jacobson John D and Cousins Larry (1989) found 30% LSCS in their study38 Langer Oded et al (2005) found total rate of LSCS to be 47%(including both treated and untreated group)50.

POSTPARTUM HAEMORRHAGE (refer to table no 12)

The present study showed the incidence of PPH to be 4%. All were atonic PPH encountered during LSCS and managed medically. Similar incidence of 6% PPH was noticed in the study done by Crowther Caroline A et al(2005)48.An incidence of 10.5% was found in B Dittakarn et al (2006) study52.

NICU ADMISSION (refer to table no 10)

63% of the total neonates were admitted to NICU for various indications like metabolic complications,birth injury and asphyxia. Maresh M et al(1989) observed a comparable incidenceof 54% in classA1 group for similar indications39..Das Vinita et al(2004) study had significant incidence of NICUadmission (p=0.001)46. Ostlund Ingrid et al(2003) noted this to be29%45. Crowther Caroline A et al(2005) found it to be as high as 71%48.

APGAR <7 AT 5 MINUTES (refer to table no 9)

This was noted in 1.6% of the cases. Similar significant observation (p- value=0.001) was made by Das Vinita et al (2004)46.Ostlund Ingrid et al(2003) found it to be 1%45.Barahona Maria Jose et al (2005) found it to be 0.6%47.

BIRTH WEIGHT (refer to table no 16)

The present study showed 8% SGA babies and 34.7% LGA babies. Similar incidence of SGA 9.5% was observed in Hedderson Monique M et al(2003)43 and 7.5% in Landon Mark B(2009)53.Jacobson John D et al(1989) found LGA to be 32%38 and Adams Kristina M et al (1997) found this to be 30% in insulin-group, & 44% in undiagnosed GDM

group40. The incidence of 34.7% in this study correlates with the above studies.

MACROSOMIA (refer to table no 16)

The present study found the incidence of macrosomy to be 10%. Both the patients were on high dose of insulin.Both underwent LSCS for cephalopelvic disproportion. Similar observation of 4% was made by Barahona Maria Jose et al(2005)47, 6% by Landon Mark B(2009)53, and 6% by Garner Peter et al(1997)41.

CONCLUSION

Diabetes mellitus is one of the most common medical disorder complicating pregnancy. If left untreated it may result in various maternal and fetal complications. Treatment includes simple dietary modifications to insulin therapy. Counseling the patients regarding the need for frequent blood glucose monitoring and frequent antenatal visits helps to achieve optimal glycemic control. Early diagnosis and good glycemic control reduces both maternal and fetal complications in GDM.

The present study shows that highest incidence of GDM occurs at around 29 years and that most of the cases can be managed with diet & exercises as first line of therapy. The study also shows that the incidence of pregnancy complications like PIH, IUGR, IUFD & polyhydramnios are increased significantly in these cases. The study confirms the increased rate of LSCS in GDM cases (more than 50%), the indications being not only GDM but also the associated risk factors like PIH and IUGR, big baby,etc.The intra partum complications like asphyxia, birth trauma, shoulder dystocia, postpartum haemorrhage are noted with increased frequency in these cases.

The neonatal metabolic complications like hypoglycemia, hypocalcaemia, RDS, hyperbilirubinemia, low Apgar are increased in babies of GDM mothers. Most of the babies need NICU care either for the morbidity or for observation upto 48-72 hours. Large for gestation babies are common in GDM cases. The incidence of macrosomy is also increased contributing for birth trauma.

Good glycemic control in GDM cases decreases both maternal and fetal morbidity.

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PROFORMA

Name:

Age:

OPD No:

Educational status:

Menstrual History: L.M.P E.D.D

Obstetric History:

Contraceptive History:

Past History:

Medical : Diabetes, Hypertension, Renal disease, Cardiac illness, Asthma, Epilepsy.

Past surgical history:

Family history:

H/O congenital anomalies, H/O twins

H/O Diabetes mellitus, hypertension, tuberculosis, asthma, epilepsy.

Personal history:

General examination:

Weight:

Height:

Weight gain during pregnancy:

BMI:

Systemic Examination:

Cardio vascular system:

Respiratory system:

Per abdomen:

Per Vaginal examination:

PATIENT CONSENT FORM

Patient may check ( ) these boxes:

( ) I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask questions and all my questions and doubts have been answered to my complete satisfaction.

( ) I understand that my participation in the study is voluntary and that I am free to withdraw at anytime without giving reason, without my legal rights being affected.

( ) I understand that sponsor of the clinical study, others working on the sponsor's behalf, the Ethics committee and the regulatory authorities will not need my permission to look at my health records, both in respect of current study and any further research that maybe conducted in relation to it, even if I withdraw from the study I agree to this access.

( )However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study.

Study title: "**A STUDY ON FETOMATERNAL OUTCOME IN GESTATIONAL DIABETES MELLITUS DIAGNOSED BY IADPSG (INTERNATIONAL ASSOCIATION OF DIABETES IN PREGNANCY STUDY GROUP) CRITERIA AT TERTIARY CARE HOSPITAL"**

Study Centre: Institute of obstetrics and gynaecology,Egmore, Chennai.

Patient's Name:

Patient's Age:

In/Out Patient Number:

I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately in form the study staff if I suffer from any deterioration in my health or well being or any unexpected or unusual symptoms.

I hereby consent to participate in this study.
I hereby give permission to undergo complete clinical examination and diagnostic tests including hematological, biochemical, radiological tests and to undergo treatment.

Signature/Thumb impression of the patient

Patient’s Name and Address:
Signature of Investigator

INFORMATION SHEET

TITLE: : **“‘A STUDY ON FETOMATERNAL OUTCOME IN GESTATIONAL DIABETES MELLITUS DIAGNOSED BY IADPSG (INTERNATIONAL ASSOCIATION OF DIABETES IN PREGNANCY STUDY GROUP) CRITERIA AT TERTIARY CARE HOSPITAL”**

Name of the investigator: Dr.S. Solairajalakshmi

Name of the Participant

Purpose of Research: To evaluate the maternal and perinatal outcome in women diagnosed with gestational diabetes mellitus according to IADPSG criteria .

Study Design: Prospective Observational study

Study Population : All pregnant women with singleton pregnancy attending AN OPD at IOG.

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:

அனுமதியுடனான ஒப்புதல் படிவம்

-இந்த ஆய்விற்கான செயல்முறையின் நோக்கத்தை நான் புரிந்துள்ளேன் என்பதை உறுதிப்படுத்துகிறேன். எனக்கு கேள்விகளை கேட்க வாய்ப்பு உள்ளது. என்னுடைய எல்லாகேள்விகளும் சந்தேகங்களும் என் முழுதிருப்திக்கு பதில் அளித்துள்ளன.

-ஆய்வில் எனது பங்கேற்பு தன்னார்வமாக இருப்பதையும், என் சட்டஉரிமைகள் பாதிக்கப்படாமல், காரணத்தைத் தெரிவிக்காமல் எப்போது வேண்டுமானாலும் விலக்கிக்கொள்ளலாம் என்பதையும் நான் புரிந்துகொள்கிறேன்.

-ஆய்வில் இருந்து நான் விலகி வந்தாலும் கூட, ஆராய்ச்சிக்கு பொருந்தக்கூடிய என் உடல்நல ஆவணங்களைப் பார்க்க என் நெறிமுறைக்குழு மற்றும் ஒழுங்குமுறை அதிகாரிகளுக்கு எனது அனுமதி தேவையில்லை என்பதை நான் புரிந்து கொள்கிறேன். இந்தஅணுகலை நான் ஏற்கிறேன்.

-இருப்பினும், சட்டத்தின்கீழ் தேவைப்பட்டாலன்றி, மூன்றாம் தரப்பினருக்கு வெளியிடப்பட்ட அல்லது வெளியிட்ட எந்த தகவலிலும் என் அடையாளத்தை வெளிப்படுத்தமுடியாது என்பதை நான் புரிந்து கொள்கிறேன். இந்த ஆய்விலிருந்து எழும் எந்தவொரு தரவு அல்லது முடிவுகளின் பயன்பாட்டைக் கட்டுப்படுத்துவதை நான் ஏற்றுக் கொள்கிறேன்.

-மேலே உள்ள படிப்பில்கலந்துகொள்ளவும், ஆய்வின் போது கொடுக்கப்பட்ட அறிவுறுத்தல்களுக்கு இணங்கவும், ஆய்வுக் குழுவோடு ஒத்துழைக்கவும், என் உடல்நலம் அல்லது நலம் அல்லது எந்தவொரு எதிர்பாராத அல்லது அசாதாரண அறிகுறிகளிலும் நான் பாதிக்கப்படுகையில் உடனடியாக ஆய்வு ஊழியர்களுக்கு தெரிவிக்கவும், இந்த ஆய்வில் பங்கேற்க ஒப்புக் கொள்கிறேன்.
நான் இதனுடன் முழுமையான மருத்துவ பரிசோதனை மற்றும் நோயறிதல் சோதனைகள் இரத்தம், உயிர்வேதியியல், கதிரியக்க சோதனைகள் உட்பட சிகிச்சைக்கு உட்படுத்த அனுமதிக்கிறேன்.

ஆய்வுதலைப்பு:

ஆய்வுமையம்: எம்.எம்.சி, சென்னை

பங்கேற்பாளரின்பெயர்:

பங்கேற்பாளரின்வயது:

நோயாளிஎண்:

நோயாளியின்கையொப்பம்

நோயாளியின்பெயர்மற்றும்முகவரி: ஆராய்ச்சியாளரின்கையொப்பம்:

நோயாளியின் ஒப்புதல் படிவம்

ஆய்வு தலைப்பு : கர்ப்பிணி பெண்களின் கர்ப்பகால சர்க்கரை நோயினை IADPSG CRITERIA மூலம் கண்டறிந்து அதன் விளைவுகளை பற்றி அரசு மருத்துவ கல்லூரி மருத்துவமனையில் ஆய்வு செய்தல்

முக்கிய ஆய்வாளரின் பெயர் : டாக்டர். ச. சோலைராஜலட்சுமி

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 பெண்களுக்கு கர்ப்பகால சர்க்கரை நோயால் குறைப் பிரசவம், அறுவை சிகிச்சையின் தேவை, வாக்குவம் கருவி உதவியுடன் பிரசவம், கருக்கலைப்பு, கர்ப்பகால உயர் இரத்த அழுத்தம், மேலும் வயிற்றில் இருக்கும் சிசுவிற்கு மூச்சுத்திணறல், கர்ப்பத்திற்கு மீறிய பெரிய குழந்தை, அதனால் பிரசவத்தின் போது காயம் ஆகியவை ஏற்பட வாய்ப்புள்ளது.

இந்த ஆய்வில் மேற்கண்ட பாதிப்புகள் எந்த அளவில் ஏற்படுகிறது. மேலும் அதன் விளைவால் தாய் மற்றும் குழந்தைக்குக் ஏற்படும் பாதிப்புகள் பற்றியும் ஆய்வு நடத்தப்படுகிறது. இந்த பரிசோதனையின் மூலம் எந்தவிதமான பாதிப்புகள் அதிக அளவில் ஏற்படுகிறது எனவும், இரத்தத்தில் சர்க்கரை அளவு அதிகரிக்கும்போது பாதிப்புகளின் வீரியமும் அதிக அளவில் உள்ளதா என்பதையும் அறியலாம், இந்த ஆய்வின் பயனாக கர்ப்பிணி பெண்களுக்கு கர்ப்ப கால சர்க்கரை நோயினால் நிகழும் பாதிப் புகளை முன்னரே அறிந்து கொள்ளவும், அதன் வீரியத்தை குறைக்கவும், மேலும் முதல் அறுவை சிகிச்சையின் எண்ணிக்கையை குறைக்கவும் சுகபிரசவத்திற்கு ஊக்குவிக்க வேண்டும் எனவும், அதனால் மேற்கண்ட பாதிப்புகள் ஏற்படாமல் எவ்வாறு தவிர்க்கலாம் என்பதையும் அறியலாம்.

உங்கள் தகவல் குறித்த நம்பிக்கை

உங்களை பற்றிய தகவல் (பரிசோதனைவிவரங்கள்) 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 சர்க்கரை நோயினை IADPSG CRITERIA மூலம் கண்டறிந்து அதன் விளைவுகளை பற்றி அரசு மருத்துவ கல்லூரி 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 STUDY ON FETOMATERNAL OUTCOME IN GESTATIONAL DIABETES MELLITUS DIAGNOSED BY IADPSG (INTERNATIONAL ASSOCIATION OF DIABETES IN PREGNANCY STUDY GROUP) CRITERIA AT TERTIARY CARE HOSPITAL”** of the candidate **DR. S. SOLAIRAJALAKSHMI** with **Registration Number: 221916898** for the award of **M.S OBSTETRICS AND GYNECOLOGY (BRANCH II)**. I personally verified that the urkund.com website for the purpose of checking plagiarism. I found that the uploaded thesis file contains contents from introduction to conclusion and result shows **SEVEN Percent** of plagiarism in the dissertation.(D124450534)

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Date :

Dr.VIJAYA, M.D DGO,
Professor,
Institute of Obstetrics and Gynaecology
Egmore, Chennai – 600 008

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.SOLAIRAJALAKSHMI S,
II year MS (OG),
Madras Medical College,
Chennai - 600003.

Dear Dr. SOLAIRAJALAKSHMI S,

The Institutional Ethics Committee has considered your request and approved your study titled **"A STUDY ON FETOMATERNAL OUTCOME IN GESTATIONAL DIABETES MELLITUS DIAGNOSED BY IADPSG (INTERNATIONAL ASSOCIATION OF DIABETES IN PREGNANCY STUDY GROUP) CRIETIA AT TERTIARY CARE HOSPITAL"- NO.14022021.** 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
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MASTER CHART KEY

COLUMN HEADING	KEY	
A	SERIAL NUMBER	
B	NAME	
C	AGE	
D	IP/OP NUMBER	
E	HEIGHT	
F	WEIGHT	
G	BMI-BODY MASS INDEX	
H	BOOKING STATUS	B-BOOKED, UB- UNBOOKED
I	GESTATIONAL AGE IN WEEKS	
J	OBSTETRIC CODE	
K	OGTT- FASTING BLOOD SUGAR	IN MG/DL
L	OGTT-1HR BLOOD SUGAR	IN MG/DL
M	OGTT 2HR BLOOD SUGAR	IN MG/DL
N	ASSOCIATED GESTATIONAL HYPERTENSION	Y- YES,N-NO
O	INSULIN REQUIREMENT	Y-YES,N-NO, ONLY MNT
P	MODE OF DELIVERY- LN	LN- LABOR NATURAL
Q	MODE OF DELIVERY -LSCS	EM-EMERGENCY,EL -ELECTIVE

MASTER CHART KEY

R	INDICATION FOR LSCS	PREV LSCS, FD -FETAL DISTRESS, FI-FAILED INDUCTION,CPD-CEPHALO PELVIC DISPROPOTION
S	SEX OF THE BABY	B-BOY,G-GIRL
T	BIRTH WEIGHT	IN KG
U	OUTCOME	L- LIVE BABY,D-DEAD BORN
V	APGAR	AT 1 MIN, AT 5 MINS
W	NICU ADMISSION	Y-YES,N-NO
X	LIQUOR STATUS	A-ADEQUATE,O-OLIGOHYDR AMNIOS,P-POLY HYDRAMNIOS
Y	PPH-POSTPARTUM HEMORRHAGE	Y-YES,N-NO
Z	FETAL OUTCOME	PT-PRETERM,T-TERM
AA	PRESENCE OF IUGR	NIL,IUGR- INTRAPARTUM UTERINE GROWTH RESTRICTION

S.No	Name	Age	IP.NO	Height	Weight	BMI	BOOKEDunbooked	Gestational age	Obscode	OGTT-FASTING	1HR	2HR	GHTN	INSULIN REQUIREMENT	LN	LSCS	INDICATION OF LSCS	SEX OF BABY	BIRTH WEIGHT	LIVE/IUD/ Abortion	APGAR	NICU ADMISSION	Polyhydramnios	PPH	TERM/ PRETEERM	IUGR
1	Pushpa	24	25655	149	51	23	B	36+6	G2P1L1	79	154	170	N	Y		Em	Prev Lscs in	B	2.615	L	8/10,9/10	Y	A	N	PT	
2	Faritha	26	25942	149	74	33.3	B	37+3	Primi	74	180	180	N	Y		Em	FD	B	3.38	L	8/10,9/10		A	N	Term	
3	Sowmiya	28	25842	152	98	42.4	B	38	G2P1L1	64	172	155	Y	N		Em	Prev Lscs in	B	3.55	L	6/10,9/10	Y	A	N	Term	
4	Deepavath	24	25542	152	53	22.9	B	38	Primi	70	184	174	N	N	Ln			G	2.67	L	8/10,9/10		A	N	Term	
5	Mageshwa	30	25642	160	86	33.6	B	37+1	G2P1L1	72	166	165	N	Y		Em	Prev Lscs in	G	3.84	L	8/10,9/10	y	A	N	Term	
6	Laxmi	29	25666	145	70	33.3	B	36+3	Primi	86	145	156	Y	N	Ln			B	2.4	L	8/10,9/10	Y	A	N	PT	
7	Reshma	30	25365	150	76	33.8	B	39+3	Primi	100	190	151	Y	N		Em	CPD	B	2.725	L	8/10,9/10		A	N	Term	
8	Shailaja	26	25122	142	50	24.8	B	40+3	Primi	73	162	179	N	N		Em	FI	G	3.45	L	8/10,9/10		A	N	Term	
9	Sumathi	28	25321	145	51	24.3	B	36+4	G2P1L1	74	123	161	N	Y	Ln			G	2.2	L	8/10,9/10	Y	A	N	PT	
10	Priya	29	25222	150	54	34	B	37+4	G2P1L1	64	161	163	N	N	Ln			B	2.615	L	8/10,9/10		A	N	Term	
11	Gayathri	25	25489	152	64	33.3	B	38+2	G2P1L1	70	139	170	Y	Y		El	Prev LSCS	B	3.38	L	8/10,9/10		A	N	Term	
12	Divya	27	25488	155	49	20.4	B	36+5	Primi	98	172	186	N	Y		Em	FD	G	2.18	L	8/10,9/10	Y	A	N	PT	
13	Suganya	34	25411	147	51	23.6	B	38+4	G3P1L1A1	69	149	177	Y	Y		El	Prev Lscs	G	2.725	L	8/10,9/10	Y	A	N	Term	
14	Krishnaver	27	25400	148	65	29.7	B	37+5	Primi	60	196	165	N	N		Em	CPD	B	2.75	L	7/10,9/10		P	Y	Term	
15	Kavitha	27	25500	154	49	27.8	B	37+3	G2P1L1	78	200	180	Y	N		Em	Prev Lscs	B	3.1	L	8/10,9/10		A	N	Term	
16	Lakshmi	27	25100	145	48	22.8	B	37	Primi	72	159	165	N	N		Em	FI	G	3.25	L	8/10,9/10		A	N	Term	
17	Amirthava	30	25633	159	53	21	B	37+4	Primi	86	166	174	N	N	Ln			G	2.65	L	8/10,9/10		P	N	Term	
18	Amuthaval	26	25963	155	49	20.4	B	38+1	G2P1L1	94	147	179	Y	Y		El	Prev Lscs	G	2.85	L	8/10,9/10		A	N	Term	
19	Sangeetha	28	25896	147	51	23.6	B	34+1	G2P1L1	110	161	159	N	Y	Ln			B		D			A	N	PT	
20	Kaviya	29	25356	156	68	27.9	B	37+5	G2P1L1	73	139	165	N	N		Em	FI	B	3.38	L	8/10,9/10		A	N	Term	
21	Vijayalaksh	25	25650	152	64	27.7	B	37+3	Primi	78	188	165	N	N		Em	FD	G	3.55	L	8/10,9/10	Y	A	N	Term	
22	Kanika	27	25965	158	66	26.4	B	37	G2P1L1	74	172	156	Y	Y		Em	Prev Lscs	B	3.45	L	8/10,9/10		A	N	Term	
23	Lakskhmi	27	25865	140	44	22.4	B	36	Primi	64	149	156	N	Y	Ln			G	2.22	L	8/10,9/10	Y	A	N	PT	
24	Andal	27	25150	142	50	24.8	B	37+4	Primi	70	189	185	N	N		Em	CPD	G	2.615	L	7/10,9/10		A	N	Term	
25	Pandishwa	35	25455	155	49	20.4	B	38	G3P1L1A1	73	133	177	Y	Y		Em	Prev Lscs	B	3.38	L	8/10,9/10		P	N	Term	

26	Mohana	30	25130	147	51	23.6	B	37+4	Primi	107	194	156	N	N	Ln			B	3.55	L	8/10,9/10		A	N	Term	
27	Tamilselvi	26	25220	150	48	21.3	B	36+3	Primi	69	159	179	N	N	Ln			B	2.4	L	8/10,9/10	Y	A	N	PT	
28	Hindhuja	28	25360	159	53	21	B	37+4	Primi	60	166	165	N	N	Ln			G	2.75	L	8/10,9/10		A	N	Term	
29	Dhivya	29	25930	142	50	24.8	B	38+1	G2P1L1	78	147	154	Y	Y	Ln			G	3.1	L	8/10,9/10		A	N	Term	
30	Gayathri	25	25830	155	49	20.4	B	37+4	Primi	79	199	170	N	N		Em	FI	G	3.25	L	8/10,9/10	Y	A	N	Term	
31	Nithya	27	25836	148	65	29.7	B	38	G2P1L1	103	172	186	N	N		Em	Prev Lscs	B	2.65	L	8/10,9/10		A	N	Term	
32	Manisha	27	25789	154	66	27.8	B	37+4	G2P1L1	72	149	156	N	Y	Ln			G	2.85	L	8/10,9/10		A	N	Term	
33	Elangai	27	25333	156	68	27.9	B	37+4	Primi	86	180	174	N	N		Em	FD	B	2.725	L	8/10,9/10	Y	A	N	Term	
34	Thirumaga	30	25001	155	49	20.4	B	37+4	G3P1L1A1	69	161	165	Y	Y	Ln			G	2.195	L	6/10,9/10	Y	O	N	PT	FGR
35	Kanniga	26	25088	147	51	23.6	B	37	G2P1L1	60	139	165	N	N	Ln			B		D			P	N	Term	
36	Palaniamm	28	25726	150	53	23.6	B	38	G2P1L1	78	132	156	N	Y		Em	Prev Lscs	B	2.615	L	8/10,9/10		A	N	Term	
37	Sailakshmi	29	25378	150	48	21.3	B	38	G2P1L1	108	182	170	N	N	Ln			G	3.38	L	8/10,9/10		A	N	Term	
38	Kannamma	25	25678	155	52	21.6	B	37+4	Primi	74	134	186	N	Y		Em	CPD	B	3.55	L	8/10,9/10	Y	A	N	Term	
39	Abirami	28	25170	155	49	20.4	B	35+4	Primi	64	161	156	Y	Y	Ln			B	2.28	L	8/10,9/10	Y	A	N	PT	
40	Nithya	24	25773	150	49	21.8	B	37+5	Primi	70	139	165	N	N	Ln			G	2.78	L	8/10,9/10		A	N	Term	
41	Aparna	26	25888	152	64	27.7	B	37+5	Primi	72	199	170	N	N	Ln			G	3.256	L	8/10,9/10		A	N	Term	
42	Gayathrii	25	25009	158	66	26.4	B	37+4	G2P1L1	86	141	186	N	N		Em	Prev Lscs	G	3.1	L	8/10,9/10		A	N	Term	
43	Monisha	27	25047	157	80	32.5	B	37+4	Primi	69	200	159	N	Y		Em	FD	B	3.45	L	8/10,9/10	Y	A	N	PT	
44	Karthiga	30	25617	159	53	21	B	38+6	G2P1L0	60	169	174	N	Y		El	Prev Lscs	G	3.95	L	8/10,9/10	Y	A	N	Term	
45	Malliga	26	25999	155	49	20.4	B	37+5	Primi	78	178	179	Y	Y		Em	CPD	G	2.75	L	8/10,9/10		A	N	Term	
46	Sinolasi	28	25015	150	53	23.6	B	37+5	G3P1L1A1	114	196	158	N	N	Ln			B	3.1	L	8/10,9/10		P	Y	Term	
47	Priya	29	25019	147	51	23.6	B	38+1	G3P1L1A1	74	201	156	N	N	Ln			B	3.25	L	8/10,9/10		A	N	Term	
48	Mullai	25	25700	159	53	21	B	37+4	Primi	79	172	165	N	N		Em	FI	G	2.65	L	8/10,9/10		A	N	Term	
49	Lilly	27	25777	150	48	21.3	B	37	G2P1L1	73	149	156	Y	Y		Em	Prev Lscs	G	2.85	L	8/10,9/10		A	N	Term	
50	Jennifer	27	25997	150	49	21.8	B	37+5	G2P1L1	74	148	168	N	N	Ln			B	3.125	L	8/10,9/10		A	N	Term	
51	Jameera B	27	26654	148	65	29.7	B	38+2	G2P1L1	64	201	170	N	N		El	Prev Lscs	B	2.725	L	8/10,9/10		A	N	Term	
52	Faritha	30	26546	154	66	27.8	B	36	Primi	70	144	186	N	Y	Ln			G	2.35	L	8/10,9/10	Y	P	N	PT	
53	Kalaiselvi	26	26540	156	68	27.9	B	37+5	Primi	69	180	180	N	N	Ln			B	2.615	L	8/10,9/10		A	N	Term	
54	Jasmeen	28	26453	153	81	34.6	B	37+5	Primi	60	161	162	N	N		Em	FD	G	3.38	L	8/10,9/10	Y	A	N	Term	
55	Vanishree	29	26410	149	79	35.6	B	37+4	G2P1L1	78	139	174	Y	Y		Em	Prev Lscs	B	3.55	L	8/10,9/10		A	N	Term	
56	Santhiya	25	26520	142	50	24.8	B	37+5	G2P1L1	100	131	170	N	N	Ln			G	2.615	L	8/10,9/10		A	N	Term	
57	Vanathi	27	26580	147	51	23.6	B	37+4	Primi	72	194	186	N	Y		Em	CPD	B	3.38	L	8/10,9/10		A	N	Term	
58	Asmitha	26	26490	159	53	21	B	35+3	Primi	86	172	179	Y	Y	Ln			G	2.15	L	8/10,9/10	Y	A	N	PT	

59	Nisha	28	26580	145	48	22.8	B	38	Primi	73	149	168	N	N	Ln			G	2.85	L	8/10,9/10		P	N	Term	
60	Ponmayil	32	26548	155	49	20.4	B	37+5	G3P1L1A1	69	149	169	N	N		Em	Prev Lscs	B	2.725	L	8/10,9/10		A	N	Term	
61	Vinitha	26	26598	152	64	27.7	B	34	G2P1L1	60	195	170	Y	Y	Ln			B	2	D			A	N	PT	
62	Indrani	28	26458	158	66	26.4	B	37+5	Primi	78	161	186	N	N		Em	FI	G	2.615	L	8/10,9/10		A	N	Term	
63	Tamilarasi	27	26453	155	85	35.4	B	37+5	Primi	73	139	153	N	N		Em	FD	G	3.38	L	8/10,9/10	Y	A	N	Term	
64	Balkiyarasi	27	26540	156	88	36.2	B	38+2	G2P1L1	109	181	174	Y	Y		El	Prev Lscs	B	3.55	L	8/10,9/10		A	N	Term	
65	Chandra	34	26533	140	44	20.4	B	38	G3P1L1A1	74	169	170	N	Y		Em	Prev Lscs	B	3.45	L	8/10,9/10		A	N	Term	
66	Ezhilarasi	30	26400	140	40	20.4	B	36+4	G2P1L1	64	178	186	Y	N	Ln			B	2.4	L	7/10,9/10	Y	A	N	PT	
67	Leelavathi	26	26154	142	50	24.8	B	37+5	Primi	70	180	165	N	N		Em	CPD	G	2.78	L	8/10,9/10		A	N	Term	
68	Dhanalaksl	28	26312	155	49	20.4	B	38	Primi	98	169	150	N	Y	Ln			B	3.256	L	8/10,9/10		A	N	Term	
69	Parvathi	29	26123	159	53	21	B	37+5	Primi	73	178	150	N	N	Ln			G	3.1	L	8/10,9/10		A	N	Term	
70	Oorvasi	25	26895	150	48	21.3	B	37+5	Primi	72	198	170	N	N		Em	FD	G	3.125	L	8/10,9/10	Y	A	N	Term	
71	Pooja	30	26985	157	65	26.4	B	36	G2P1L1	86	169	186	Y	Y		Em	Prev Lscs	G	2.36	L	8/10,9/10	Y	A	N	PT	
72	Amrutha	26	26151	150	62	27.6	B	37+5	Primi	73	178	155	N	Y		Em	FI	G	2.85	L	8/10,9/10		P	Y	Term	
73	Shame a	28	26141	154	66	27.8	B	38	G2P1L1	97	200	168	Y	Y		Em	Prev Lscs	B	2.725	L	8/10,9/10		A	N	Term	
74	Sangeetha	29	26150	155	75	31.2	B	36+2	G2P1L1	74	161	185	N	N	Ln			B	2.2	L	8/10,9/10	Y	A	N	PT	
75	Manjari	25	26250	154	74	31.2	B	38+3	Primi	64	139	177	N	N		El	Breech	B	2.615	L	8/10,9/10		A	N	Term	
76	Pradeepa	27	26352	140	44	22.4	B	37+5	G2P1L1	70	180	160	N	Y		Em	Prev Lscs	G	3.38	L	8/10,9/10		A	N	Term	
77	Pavithra	27	26523	140	40	20.4	B	37+3	Primi	79	145	170	N	N		Em	CPD	B	3.9	L	8/10,9/10	Y	A	N	Term	
78	Banupriya	33	26951	142	50	24.8	B	37	G3P1L1A1	73	172	186	N	Y		Em	Prev Lscs	G	2.615	L	8/10,9/10		A	N	Term	
79	Sumathi	30	26520	155	49	20.4	B	37+5	G2P1L1	95	149	168	N	N	Ln			G	3.38	L	8/10,9/10		A	N	Term	
80	Kamatchi	26	26500	159	53	21	B	38+3	G2P1L1	69	190	174	Y	Y		El	Prev Lscs	B	3.55	L	8/10,9/10		A	N	Term	
81	Karthika	28	26890	149	62	27.9	B	35	G2P1L1	60	169	170	N	N	Ln			B	2.02	L	8/10,9/10	Y	A	N	PT	
82	Sumithra	29	26982	153	64	27.3	B	37+5	G2P1L1	78	178	186	Y	Y		Em	Prev Lscs	B	3.45	L	8/10,9/10		A	N	Term	
83	Rita	25	26999	152	68	29.4	B	38	Primi	72	199	166	N	N		Em	FD	B	2.75	L	8/10,9/10	Y	A	N	Term	
84	Lordu Mar	26	26853	156	78	32.1	B	37+5	G2P1L1	86	161	169	N	N	Ln			G	3.1	L	8/10,9/10		P	N	Term	
85	Kumudha	28	26333	145	70	33.3	B	37+5	G2P1L1	105	139	170	N	N		Em	Prev Lscs	B	3.25	L	8/10,9/10		A	N	Term	
86	Shaziya	29	26352	140	44	22.4	B	37	Primi	74	154	186	N	N		Em	CPD	G	2.65	L	8/10,9/10		A	N	Term	
87	Yamuna	25	26986	142	50	24.8	B	37+5	Primi	64	194	156	Y	Y	Ln			B	2.85	L	8/10,9/10		A	N	Term	
88	Prameshw	27	26669	140	40	20.4	B	38	Primi	70	162	155	N	N	Ln			G	3.125	L	7/10,9/10	Y	A	N	Term	
89	Jeeva	27	26848	155	49	20.4	B	33+6	Primi	103	172	151	N	Y	Ln			B	2	D			A	N	PT	
90	Kalaiarasi	33	26898	159	53	21	B	37+5	G3P1L1A1	73	149	170	N	N		Em	Prev Lscs	G	2.615	L	8/10,9/10		A	N	Term	
91	Bhavani	27	26111	155	63	26.2	B	37+5	G2P1L1	78	200	186	N	N	Ln			B	3.38	L	8/10,9/10		A	N	Term	

92	Narmadha	27	26600	155	64	26.6	B	37+5	Primi	69	161	166	N	N		Em	FD	G	3.55	L	8/10,9/10	Y	A	N	Term	
93	Rajeshwar	30	26775	162	84	32	B	37	Primi	60	139	174	N	Y		Em	FI	B	3.45	L	8/10,9/10		A	N	Term	
94	Juliet Rani	26	26757	160	83	32.4	B	35+4	G2P1L1	78	201	169	Y	Y		Em	Prev Lscs	G	2.35	L	8/10,9/10	Y	O	N	PT	FGR
95	Sameera	28	26773	142	50	24.8	B	37+5	Primi	72	162	165	N	N		Em	CPD	B	3.64	L	8/10,9/10		P	Y	Term	
96	Kalaiarasi	29	26737	155	49	20.4	B	37+5	Primi	102	185	163	Y	Y	Ln			G	3.125	L	8/10,9/10		A	N	Term	
97	Valarmathi	25	26173	140	40	20.4	B	36	G2P1L1	74	175	161	N	N		Em	Prev Lscs	B	2.24	L	8/10,9/10	Y	A	N	PT	
98	Shanthi	27	26711	159	53	21	B	38	G2P1L1	64	163	179	N	Y	Ln			B	2.78	L	8/10,9/10		A	N	Term	
99	Janaki	35	26577	140	44	22.4	B	37+2	G3P1L1A1	70	188	160	N	Y		Em	Prev lscs	B	3.256	L	8/10,9/10		A	N	Term	
100	Bhavani	30	26377	142	50	24.8	B	37+5	Primi	69	161	159	N	N		Em	FD	G	3.1	L	8/10,9/10	Y	A	N	Term	
101	Valarmathi	26	26799	148	65	29.7	B	37+5	G2P1L1	60	139	170	N	N	Ln			G	3.45	L	8/10,9/10		A	N	Term	
102	Suganthi	28	26758	154	66	27.8	B	35+1	G3P1L1A1	78	188	186	Y	Y		Em	Prev lscs	B	2.05	L	8/10,9/10	Y	A	N	PT	
103	Maheshwa	29	26375	158	66	26.4	B	37+5	G2P1L1	72	175	156	N	N	Ln			G	2.85	L	8/10,9/10		A	N	Term	
104	Divya	25	26710	149	75	33.8	B	37+2	G2P1L1	86	163	174	N	N		Em	Prev Lscs	G	2.725	L	8/10,9/10		P	N	Term	
105	Karpagam	27	26730	155	80	33.3	B	36+3	G2A1	100	184	185	Y	Y	Ln			B	2.34	L	8/10,9/10	Y	A	N	PT	
106	Sameena	27	26700	142	50	24.8	B	37+2	Primi	79	172	177	N	Y		Em	CPD	G	2.615	L	8/10,9/10		A	N	Term	
107	Tamilarasi	26	27123	155	49	20.4	B	37+2	Primi	74	149	160	N	N		Em	FI	G	3.38	L	8/10,9/10		A	N	Term	
108	Nandhini	34	27213	140	44	22.4	B	36+5	G3P1L1A1	64	185	179	Y	Y		Em	Prev Lscs	B	2.4	L	8/10,9/10	Y	A	N	PT	
109	Tamilselvi	26	27452	159	53	21	B	37+5	G2P1L1	70	161	161	N	N		Em	Prev Lscs	G	3.125	L	7/10,9/10		A	N	Term	
110	Divya	28	27321	140	40	20.4	B	37+3	Primi	78	139	160	Y	Y		Em	FD	B	2.75	L	8/10,9/10	Y	A	N	Term	
111	Dhanalaksl	29	27322	142	50	24.8	B	37	G2A1	94	200	165	N	N	Ln			B	3.1	L	8/10,9/10		A	N	Term	
112	Pavithra	25	27235	156	68	27.9	B	37+2	G2P1L1	72	195	162	N	Y		Em	Prev Lscs	G	3.25	L	8/10,9/10		P	N	Term	
113	Amala	27	27152	152	64	27.7	B	37+2	Primi	86	159	169	N	N		Em	CPD	B	2.65	L	8/10,9/10		A	N	Term	
114	KLpana	36	27145	156	90	37	B	38+3	G4P1L1A2	73	166	170	Y	Y		El	Prev Lscs	B	2.85	L	8/10,9/10		A	N	Term	
115	Kalpana	30	27111	166	92	33.4	B	38+1	Primi	69	147	186	N	N		El	Breech	G	2.615	L	8/10,9/10		A	N	Term	
116	Balasangee	26	27632	142	50	24.8	B	37+2	Primi	60	196	159	Y	Y		Em	FI	B	3.38	L	8/10,9/10		A	N	Term	
117	Monica	28	27163	155	49	20.4	B	38	G2P1L1	78	161	155	N	N		Em	Prev Lscs	G	3.55	L	8/10,9/10		A	N	Term	
118	Saranya	29	27362	140	44	22.4	B	34	G2A1	73	139	180	Y	Y	Ln			B	2.05	D		Y	A	N	PT	
119	Gunapoora	25	27522	159	53	21	B	36	Primi	105	199	158	N	N		Em	FD	B	2.5	L	8/10,9/10	Y	A	N	PT	
120	Nazira	26	27500	140	40	20.4	B	37+5	G2A1	74	172	174	N	N	Ln			G	3.45	L	8/10,9/10		A	N	Term	
121	Vennila	27	27367	142	50	24.8	B	36+6	G2P1L1	64	149	185	Y	Y		Em	Prev Lscs	G	2.45	L	8/10,9/10	Y	A	N	PT	
122	Kameshwa	27	27987	154	66	27.8	B	37+2	Primi	70	186	177	N	N		Em	CPD	B	2.65	L	8/10,9/10		A	N	Term	
123	Kanchana	34	27985	156	68	27.9	B	38+3	G3P1L1A1	72	159	169	N	Y		El	Prev Lscs	B	3.85	L	8/10,9/10		A	N	Term	
124	Sathya	30	27159	158	66	26.4	B	37+5	Primi	86	166	165	N	N		Em	FI	B	2.615	L	8/10,9/10		A	N	Term	

125	Radhika	26	27489	157	80	32.5	B	37+3	Primi	99	147	168	Y	Y		Em	FD	G	3.38	L	8/10,9/10	Y	A	N	Term	
126	Amudha	28	27980	153	81	34.6	B	37	Primi	69	185	162	N	N		Em	CPD	G	3.55	L	8/10,9/10		P	Y	Term	
127	Anandhi	29	27577	155	49	20.4	B	37+2	G2P1L1	60	161	170	N	N		Em	Prev lscs	G	3.125	L	8/10,9/10		A	N	Term	
128	Yashodha	25	27333	142	50	24.8	B	35+5	G2P1L1	78	139	186	Y	Y	Ln			B	2.2	L	8/10,9/10	Y	A	N	PT	
129	Tamilselvi	27	27484	140	44	22.4	B	37+5	Primi	73	166	163	N	N		Em	Breech	G	2.88	L	8/10,9/10	Y	A	N	Term	
130	Sudha	30	27977	140	40	20.4	B	37+3	Primi	108	186	174	N	N		Em	FD	G	2.615	L	8/10,9/10	Y	A	N	PT	
131	Revathi	26	27800	155	49	20.4	B	37	G2P1L1	74	172	179	Y	Y		Em	Prev lscs	B	3.38	L	8/10,9/10		A	N	Term	
132	Anjali devi	28	27969	148	65	29.7	B	37+2	Primi	64	149	163	N	Y		Em	CPD	G	3.55	L	8/10,9/10		P	N	Term	
133	Kalarani	29	27635	152	64	27.7	B	35+4	G2P1L1	70	200	166	Y	Y	Ln			B	2.14	L	8/10,9/10	Y	A	N	PT	
134	Lavanya	25	27956	149	79	35.6	B	37+5	G2P1L1	73	204	170	N	N		Em	Prev lscs	G	2.75	L	8/10,9/10		A	N	Term	
135	Rekha	27	27976	155	85	35.4	B	37+2	Primi	78	164	186	Y	N		Em	FD	B	3.1	L	8/10,9/10	Y	A	N	Term	
136	Shanthi	27	27831	140	40	20	B	37+5	G2P1L1	104	159	165	N	N	Ln			G	3.25	L	8/10,9/10		A	N	Term	
137	Saranya	31	27907	142	50	24.8	B	38	G3P1L1A1	72	166	169	N	Y		Em	Prev Lscs	B	2.65	L	6/10,9/10	Y	A	N	Term	
138	Sanju	30	27507	145	48	22.8	B	37+2	Primi	86	147	180	N	N		Em	CPD	B	2.85	L	8/10,9/10		A	N	Term	
139	Jayanthi	26	27733	159	53	21	B	36	G2A1	73	195	156	Y	Y	Ln			B	2	L	8/10,9/10	Y	O	N	PT	FGR
140	Kala	28	27199	155	49	20.4	B	38+3	G2P1L1	104	161	174	Y	Y		El	Prev Lscs	B	2.615	L	8/10,9/10	Y	A	N	PT	
141	Kalaivani	29	27186	142	50	24.8	B	37+5	Primi	72	139	175	N	N	Ln			G	3.38	L	8/10,9/10		A	N	Term	
142	Soundhara	25	27136	154	66	27.8	B	37+2	Primi	86	199	188	N	N		Em	CPD	B	3.55	L	8/10,9/10		P	N	Term	
143	Reshma	30	27158	156	68	27.9	B	36	G2P1L1	69	175	165	Y	Y	Ln			G	2.02	L	8/10,9/10	Y	A	N	PT	
144	Mohana	26	27509	152	64	27.7	B	37+2	Primi	60	163	166	N	N		Em	FD	B	2.78	L	8/10,9/10	Y	A	N	Term	
145	Siva Nandh	28	27971	156	88	36.2	B	38	G2P1L1	78	201	164	Y	Y		Em	Prev Lscs	G	3.256	L	8/10,9/10		A	N	Term	
146	Devagi	29	27833	155	75	31.2	B	37+3	Primi	78	172	186	N	Y	Ln			B	3.1	L	8/10,9/10		A	N	Term	
147	Priyanka	25	27666	140	40	20.4	B	38	Primi	72	149	154	N	N	Ln			G	3.86	L	8/10,9/10	Y	A	N	Term	
148	Ramya	27	27447	150	48	21.3	B	37+1	Primi	86	144	145	N	Y	Ln			B		D			A	N	Term	
149	Manju	27	27770	155	49	20.4	B	37+2	Primi	109	210	169	N	N		Em	FI	G	2.615	L	8/10,9/10		A	N	Term	
150	Anitha	27	28011	142	50	24.8	B	37+3	G2A1	74	175	180	N	N	Ln			B	3.38	L	8/10,9/10		A	N	Term	
151	Bhavani	30	28026	148	65	29.7	B	37+2	Primi	64	163	166	N	N		Em	CPD	G	3.55	L	8/10,9/10		A	N	Term	
152	Devi Rajesl	26	28048	158	66	26.4	B	36+4	Primi	70	180	174	Y	Y		Em	FD	B	2.38	L	8/10,9/10	Y	A	N	PT	
153	Sundari	28	28098	154	74	31.2	B	38+1	G2P1L1	73	161	185	N	N		El	Prev Lscs	G	2.78	L	8/10,9/10		A	N	Term	
154	Gayathri	29	28012	156	78	32.1	B	37+2	G2P1L1	112	139	177	N	Y		Em	Prev Lscs	B	3.256	L	8/10,9/10		P	N	Term	
155	Jayanthi	25	28015	155	49	20.4	B	38	G2A1	74	182	159	Y	Y	Ln			G	3.1	L	8/10,9/10		A	N	Term	
156	Hamsaven	27	28019	145	48	22.8	B	34	G2A1	69	172	179	Y	Y	Ln			G	2.2	L	8/10,9/10	Y	A	N	PT	
157	Praveenap	27	28024	159	53	21	B	37+2	Primi	60	149	169	N	N		Em	FI	B	3.45	L	8/10,9/10		A	N	Term	

158	Valarmathy	34	28028	140	40	20.4	B	38	G3P1L1A1	78	199	162	N	Y		Em	Prev Lscs	B	2.615	L	8/10,9/10		A	N	Term	
159	Baby	30	28065	155	49	20.4	B	37+3	Primi	70	175	165	N	N	Ln			G	3.38	L	8/10,9/10		A	N	Term	
160	Neelavathy	26	28089	142	50	24.8	B	37+2	Primi	98	163	166	N	N		Em	FD	G	3.55	L	8/10,9/10	Y	A	N	Term	
161	Subbulaxm	28	28095	157	65	26.4	B	34+6	G2A1	74	186	168	Y	Y	Ln			G	2.18	L	8/10,9/10	Y	A	N	PT	
162	Jayabharat	29	28154	150	62	27.6	B	37+2	G2P1L1	64	175	186	N	N		Em	Prev Lscs	G	2.78	L	8/10,9/10		A	N	Term	
163	Shobarani	25	28198	154	66	27.8	B	38	Primi	70	163	165	N	Y		Em	CPD	B	3.256	L	8/10,9/10		P	Y	Term	
164	Ramya	27	28245	145	70	33.3	B	38+1	G2P1L1	73	200	160	N	N		El	Prev Lscs	B	3.1	L	8/10,9/10		A	N	Term	
165	Vijayalaksh	27	28298	162	84	32	B	35+6	G2A1	100	210	174	Y	Y	Ln			G	2.36	L	8/10,9/10	Y	A	N	PT	
166	Vidhya	35	28290	142	50	24.8	B	37+2	G3P1L1A1	72	161	168	N	N		Em	Prev Lscs	B	2.85	L	8/10,9/10		A	N	Term	
167	Finoliya	30	28210	147	51	23.6	B	37+3	Primi	86	139	177	N	Y	Ln			B	2.725	L	8/10,9/10		A	N	Term	
168	Angel papa	26	28345	159	53	21	B	37+2	Primi	69	172	156	Y	Y	Ln			B		D			A	N	Term	
169	Roobini	28	28365	142	50	24.8	B	36	G2P1L1	60	149	156	Y	Y		Em	Prev Lscs	G	2.5	L	8/10,9/10	Y	A	N	PT	
170	Geetha	29	28398	155	49	20.4	B	37+2	Primi	78	195	179	N	N		Em	FD	G	2.615	L	8/10,9/10	Y	A	N	Term	
171	Sowmiya	25	28390	149	62	27.9	B	38+1	G2P1L1	72	142	154	N	N		El	Prev Lscs	B	3.38	L	8/10,9/10		A	N	Term	
172	Sundhari	27	28412	153	64	27.3	B	37+5	Primi	86	181	155	Y	Y		Em	FI	G	3.55	L	8/10,9/10	Y	A	N	PT	
173	Anitha	31	28436	160	83	32.4	B	37+3	G3P1L1A1	104	161	186	N	Y		Em	Prev Lscs	B	3.125	L	8/10,9/10		A	N	Term	
174	Banumathy	30	28445	149	75	33.8	B	37	Primi	73	139	160	N	N		Em	CPD	G	3.68	L	8/10,9/10		A	N	Term	
175	Mythili	26	28440	140	40	20.4	B	37+2	G2A1	69	175	180	N	Y	Ln			B	3.45	L	8/10,9/10		A	N	Term	
176	Revathy	28	28459	142	50	24.8	B	36+1	G2P1L1	60	163	162	Y	N		Em	Prev lscs	G	2.28	L	8/10,9/10	Y	A	N	PT	
177	Basheera	29	28499	147	51	23.6	B	37+2	G3P1L1A1	78	180	174	N	Y	Ln			B	2.85	L	8/10,9/10		A	N	Term	
178	Jasmine	25	28512	150	48	21.3	B	38+1	G2P1L1	73	145	166	N	N		El	Transverse	B	2.725	L	8/10,9/10		P	N	Term	
179	Jeny	27	28520	159	53	21	B	36+2	Primi	105	161	186	Y	Y	Ln			G	2.2	L	8/10,9/10	Y	A	N	PT	
180	Kanchana	30	28569	155	49	20.4	B	37+2	Primi	78	139	155	N	N	Ln			G	2.78	L	8/10,9/10		A	N	Term	
181	Pooja	26	28588	155	63	26.2	B	37+2	Primi	74	185	165	N	N		Em	FD	B	3.256	L	8/10,9/10	Y	A	N	Term	
182	Renuka	28	28594	155	64	26.6	B	36	G2A1	64	199	151	Y	Y	Ln			G	2.14	L	7/10,9/10	Y	A	N	PT	
183	Selvi	29	28580	148	65	29.7	B	37+2	G2P1L1	70	159	152	N	Y		Em	Prev Lscs	B	3.25	L	6/10,9/10	Y	A	N	Term	
184	Sumiya	25	28599	155	80	33.3	B	36+4	G2A1	73	166	179	Y	Y	Ln			G	2.03	L	8/10,9/10	Y	A	N	PT	
185	Vaidegi	33	28610	156	90	37	B	38+1	G3P1L1A1	98	147	166	N	Y		El	Prev Lscs	B	2.78	L	8/10,9/10		A	N	Term	
186	Rani	30	28618	150	53	23.6	B	37+5	Primi	78	194	161	N	N		Em	CPD	G	3.256	L	8/10,9/10		P	Y	Term	
187	Khatambar	26	28624	150	48	21.3	B	37+3	G2P1L1	74	172	180	N	N		Em	Prev Lscs	B	3.1	L	8/10,9/10		A	N	Term	
188	Vaishnavi	28	28639	142	50	24.8	B	37	Primi	64	149	160	N	N		Em	FI	G	3.45	L	8/10,9/10		A	N	Term	
189	Jayashri	29	28640	147	51	23.6	B	36	Primi	70	149	174	Y	Y		Em	FD	B	2.05	L	8/10,9/10	Y	O	N	PT	FGR
190	Chitra	25	28644	155	49	20.4	B	37+2	G2P1L1	69	195	185	N	N		Em	Prev Lscs	G	2.78	L	8/10,9/10		A	N	Term	

191	Vanitha	27	28699	154	66	27.8	B	37+2	G2A1	60	175	154	Y	Y	Ln			G	3.256	L	8/10,9/10	Y	A	N	PT	
192	Lakshmi	32	28812	156	68	27.9	B	38	G3P1L1A1	78	163	155	N	N		Em	Prev Lscs	B	3.1	L	8/10,9/10		A	N	Term	
193	Poongodha	28	28900	152	64	27.7	B	37+2	Primi	96	199	162	N	Y		Em	FD	B	3.125	L	8/10,9/10	Y	P	N	Term	
194	Pratheepa	29	28855	157	80	32.5	B	33+4	G3P1L1A1	74	161	165	N	N	Ln			G	1.9	D		Y	A	N	PT	
195	Elakiya	25	28816	153	81	34.6	B	37+2	G2P1L1	64	139	165	Y	Y		Em	Prev Lscs	G	2.85	L	8/10,9/10		A	N	Term	
196	Gowsalya	27	28893	150	53	23.6	B	34+1	G3P1L1A1	70	201	169	N	N	Ln			G	2.3	L	8/10,9/10	Y	A	N	PT	
197	Jeyashree	30	28914	159	53	21	B	38+6	G2P1L1	73	172	158	N	N		El	Prev Lscs	B	3.45	L	8/10,9/10		A	N	Term	
198	Dhatchayir	26	28956	155	49	20.4	B	37+2	Primi	92	149	186	N	N		Em	CPD	B	3.9	L	8/10,9/10		A	N	Term	
199	Yogeshwar	28	28936	150	48	21.3	B	35+6	Primi	73	200	169	Y	Y	Ln			B	2.06	L	8/10,9/10	Y	A	N	PT	
200	Priya	29	28963	147	51	23.6	B	37+5	Primi	69	159	168	N	N		Em	FI	B	2.78	L	8/10,9/10		A	N	Term	
201	Jeyaselvi	25	28940	158	66	26.4	B	37+3	G2P1L1	60	199	158	N	N		Em	Prev lscs	G	3.256	L	8/10,9/10		A	N	Term	
202	Kalyana Su	27	29011	157	65	26.4	B	37	Primi	78	159	186	N	Y		Em	CPD	B	3.1	L	8/10,9/10		P	Y	Term	
203	Baby Shalir	30	29035	149	79	35.6	B	37+2	Primi	104	166	158	N	N		Em	FD	G	3.125	L	8/10,9/10	Y	A	N	Term	
204	Janet rani	26	29054	155	85	35.4	B	34+4	Primi	78	147	154	N	Y	Ln			B	2.05	L	8/10,9/10	Y	A	N	PT	
205	Malini	28	29090	145	48	22.8	B	34+3	Primi	74	185	174	Y	N	Ln			B	2	L	8/10,9/10	Y	A	N	PT	
206	Indrani	29	29028	142	50	24.8	B	38	Primi	64	161	185	Y	Y	Ln			G	2.75	L	8/10,9/10		A	N	Term	
207	Sumathy	25	29061	159	53	21	B	37+2	Primi	70	139	177	N	N		Em	FI	B	3.1	L	8/10,9/10		A	N	Term	
208	Chithikame	27	29066	150	48	21.3	B	37+6	G2P1L1	78	186	160	N	N	Ln			B	3.25	L	8/10,9/10		A	N	Term	
209	Divya laksh	27	29112	150	49	21.8	B	37+2	G2P1L1	98	159	165	N	Y		Em	Prev Lscs	B	2.65	L	7/10,9/10		A	N	Term	
210	Nirmala	35	29145	155	49	20.4	B	38+1	G3P1L1A1	72	166	160	N	N		El	Prev Lscs	B	2.85	L	8/10,9/10		A	N	Term	
211	Nandhini	29	29154	150	62	27.6	B	36	Primi	86	147	169	Y	Y		Em	FD	G	2.3	L	8/10,9/10	Y	A	N	PT	
212	Kanmani	25	29158	154	66	27.8	B	37+1	G2A1	69	180	186	N	Y	Ln			B		D			P	N	Term	
213	Kalpana	27	29168	149	62	27.9	B	37+5	G2P1L1	60	172	164	N	N		Em	Prev Lscs	G	3.256	L	8/10,9/10		A	N	Term	
214	Indhumath	32	29198	156	88	36.2	B	37+3	G3P1L1A1	78	149	180	Y	N		Em	Prev Lscs	B	3.1	L	8/10,9/10		A	N	Term	
215	Malathy	30	29190	150	53	23.6	B	37	Primi	72	132	165	N	N		Em	CPD	B	2.85	L	8/10,9/10		A	N	Term	
216	Jana Nayag	26	29214	159	53	21	B	37+2	G2P1L1	86	180	174	Y	Y		Em	Prev Lscs	G	2.725	L	8/10,9/10	Y	A	N	Term	
217	Anitha	28	29218	142	50	24.8	B	34+3	G2P1L1	97	161	166	N		Ln			B	2.2	L	8/10,9/10	Y	A	N	PT	
218	Sowmiya	29	29210	145	48	22.8	B	37+2	Primi	78	139	187	N			Em	FI	B	2.85	L	8/10,9/10		A	N	Term	
219	Chitra	25	29225	150	48	21.3	B	35	G2A1	74	200	179	Y	Y	Ln			B	2.04	L	8/10,9/10	Y	A	N	PT	
220	Nithya	27	29228	150	49	21.8	B	38+6	G2P1L1	64	201	155	N	N		El	Prev lscs	G	3.45	L	8/10,9/10		A	N	Term	
221	Agilamdesl	27	29223	152	64	27.3	B	34+6	G2A1	70	145	165	Y	Y	Ln			B	2	L	8/10,9/10	Y	A	N	PT	
222	Indhra	30	29235	155	63	26.2	B	37+2	G2P1L1	72	199	186	N	Y		Em	Prev lscs	B	2.85	L	8/10,9/10		A	N	Term	
223	Sneha	26	29238	155	85	35.4	B	38	Primi	108	175	169	N	N		Em	FD	B	2.725	L	8/10,9/10	Y	P	Y	Term	

224	Nithya Gar	28	29248	155	49	20.4	B	36	Primi	69	163	165	Y	Y	Ln			G	2.14	L	8/10,9/10	Y	A	N	PT	
225	Dhivya	29	29345	145	48	22.8	B	37+2	Primi	60	172	168	N	N		Em	CPD	B	3.125	L	8/10,9/10		A	N	Term	
226	Priya	25	29200	159	53	21	B	38+6	G2P1L1	78	149	155	N	Y		El	Prev lscs	G	3.95	L	8/10,9/10	Y	A	N	Term	
227	Kamatchi	27	29384	142	50	24.8	B		Primi	73	199	180	N	N	Ln			B	2.78	L	8/10,9/10		A	N	Term	
228	Velvizhi	31	29368	150	48	21.3	B	37+2	G3P1L1A1	105	161	165	N	N		Em	Prev lscs	B	3.256	L	8/10,9/10		A	N	Term	
229	Chandraka	27	29412	150	53	23.6	B	37+6	Primi	73	139	174	Y	Y		Em	FI	G	3.1	L	8/10,9/10		A	N	Term	
230	Khateeja	27	29486	147	51	23.6	B	35+6	G2A1	69	194	185	Y	Y	Ln			G	2.33	L	8/10,9/10	Y	A	N	PT	
231	Maheshwa	30	29512	155	64	26.6	B	37+5	G2P1L1	60	175	177	N	N		Em	Prev lscs	B	2.615	L	8/10,9/10		A	N	Term	
232	Kannagi	26	29524	148	65	29.7	B	37+3	Primi	78	163	168	N	Y		Em	FD	G	3.38	L	8/10,9/10	Y	A	N	Term	
233	Jayarani	28	29565	154	66	27.8	B	37	G2P1L1	73	195	155	N	N		Em	Prev lscs	B	3.55	L	8/10,9/10		A	N	Term	
234	Tamilarasi	29	29548	155	75	31.2	B	35	G2P1L1	96	161	187	Y	Y	Ln			B	2.09	L	8/10,9/10	Y	A	N	PT	
235	Suganya	25	29590	154	74	31.2	B	37+6	Primi	73	139	179	N	N		Em	CPD	G	2.85	L	8/10,9/10		A	N	Term	
236	Vinothini	27	29612	159	53	21	B	38+6	G2P1L1	74	166	161	N	Y		El	Prev lscs	G	2.725	L	8/10,9/10		P	Y	Term	
237	Angajala I	27	29632	142	50	24.8	B	35+4	Primi	64	180	168	Y	N	Ln			B	2.13	L	8/10,9/10	Y	A	N	PT	
238	Vinoliya	30	29654	150	48	21.3	B	32	Primi	70	172	185	Y	Y	Ln			G	1.54	D		Y	O	N	PT	FGR
239	Santhiya	26	29658	155	49	20.4	B	38+4	G2P1L1	72	149	177	N	Y		El	Prev lscs	B	2.75	L	8/10,9/10		A	N	Term	
240	Annalakshı	28	29712	145	48	22.8	B	37+6	G2P1L1	86	182	164	N	N	Ln			G	3.1	L	8/10,9/10		A	N	Term	
241	Shantha	29	29735	156	68	27.9	B	37+5	Primi	99	175	199	N	N		Em	FD	B	3.25	L	8/10,9/10	Y	A	N	Term	
242	Jenita	25	29769	152	64	27.7	B	37+3	Primi	73	163	165	N	N		Em	FI	B	2.65	L	8/10,9/10		A	N	Term	
243	Dhanalaksl	34	29812	156	78	32.1	B	37	G3P1L1A1	74	180	169	Y	Y		Em	Prev lscs	G	2.85	L	8/10,9/10		A	N	Term	
244	Vanaja	26	29829	145	70	33.3	B	35+4	Primi	64	161	164	N	Y	Ln			G	2.04	L	8/10,9/10	Y	A	N	PT	
245	Anandhi	28	29865	155	49	20.4	B	38+2	Primi	70	139	156	N	N	Ln			B	3.86	L	8/10,9/10		A	N	Term	
246	Rani	29	29856	142	50	24.8	B	37+6	G2A1	69	201	180	N	N		Em	CPD	B	2.85	L	8/10,9/10		A	N	Term	
247	Kasiammal	25	29884	147	51	23.6	B	34+5	G2P1L1	60	172	156	Y	Y	Ln			B	2	L	8/10,9/10	Y	A	N	PT	
248	Saraswathy	34	29890	145	48	22.8	B	39	G3A4	78	149	174	N	Y		El	Breech	G	3.45	L	7/10,9/10		P	N		
249	Bharathi	27	29901	159	53	21	B	36	G2A1	104	200	156	N	N	Ln			B	2.1	L	8/10,9/10	Y	A	N	PT	
250	Vidhya	27	29956	155	49	20.4	B	38+4	G2P1L1	73	177	158	Y	Y		El	Prev lscs	G	3.125	L	8/10,9/10		A	N		