A STUDY ON
"CORRELATION OF HBA1C AND MACROSOMIA IN INFANTS OF DIABETIC MOTHER"

A Dissertation Submitted In

Partial Fulfilment of the Requirements

For The Degree of

DOCTOR OF MEDICINE (M.D)

BRANCH VII - PAEDIATRIC MEDICINE

GOVT. KILPAUK MEDICAL COLLEGE

THE TAMILNADU DR. M.G.R MEDICAL UNIVERSITY

CHENNAI, TAMILNADU

MAY 2018

BONAFIDE CERTIFICATE

This is to certify that dissertation named **"CORRELATION OF HBAIC AND MACROSOMIA IN INFANTS OF DIABETIC MOTHER"** is a bonafide original research work carried out by **Dr.ANWIKA KIRTI KUJUR**, post graduate student, Department of Paediatrics, Govt. Kilpauk Medical College Chennai - 10 under our direct supervision and guidance in partial fulfilment of the requirements for the award of the degree of Doctor of Medicine (M.D Paediatrics) Branch VII Paediatric Medicine during the academic year 2014-2018.

Prof. Dr. DEVIMEENAKSHI, M.D.,
Professor ,
Department of Paediatrics,
Govt. Kilpauk Medical College & Hospital
Chennai-10

Prof Dr.K.SUGUNA, M.D.,DCH.,
Professor and Head of Department
Department of Paediatrics
Govt. Kilpauk Medical College & Hospital
Govt. Royapettah Hospital
Chennai-10

Prof. Dr.P.VASANTHAMANI,
MD, DGO, MNAMS, DCPSY, MBA
Dean,
Govt. Kilpauk Medical College & Hospital,
Chennai-10

DECLARATION

I **Dr. ANWIKA KIRTI KUJUR,** hereby solemnly declare that this dissertation entitled **"CORRELATION OF HBA1C AND MACROSOMIA IN INFANTS OF DIABETIC MOTHER"** has been conducted by me at Government Kilpauk Medical College and Hospital, Chennai, under the guidance and supervision of **PROF. DR. K. SUGUNA M.D.**, **D.C.H**., Professor& HOD Department of Paediatrics, Govt. Royapettah Hospital / Govt. Kilpauk Medical College & Hospital, Chennai. This dissertation is submitted to **The Tamil Nadu Dr. M.G.R. Medical University, Chennai** in partial fulfillment of the University rules and regulations for the award of the degree of **M.D. Branch VII (Paediatrics)**.

This has not previously been submitted by me for the award of any degree diploma from any other university.

(Dr. ANWIKA KIRTI KUJUR)

CERTIFICATE – II

This is to certify that this dissertation work titled entitled dissertation **"CORRELATION OF HBA1C AND MACROSOMIA IN INFANTS OF DIABETIC MOTHER"** of the candidate **Dr.ANWIKA KIRTI KUJUR** with Registration Number **201417154** for the award of **M.D** degree in the branch of **PEDIATRICS**. I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows 7**%** of plagiarism in this dissertation.

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INSTITUTIONAL ETHICS COMMITTEE
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CERTIFICATE OF APPROVAL

The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval " **Correlation of HbA1c and macrosomia in infant of diabetic mother** " - For Project Work submitted by **Dr.Anwikakirtikujur**, MD Pediatrics, Govt. Kilpauk Medical College, Chennai – 10.

The Proposal is APPROVED.

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information /informed consent and asks to be provided a copy of the final report

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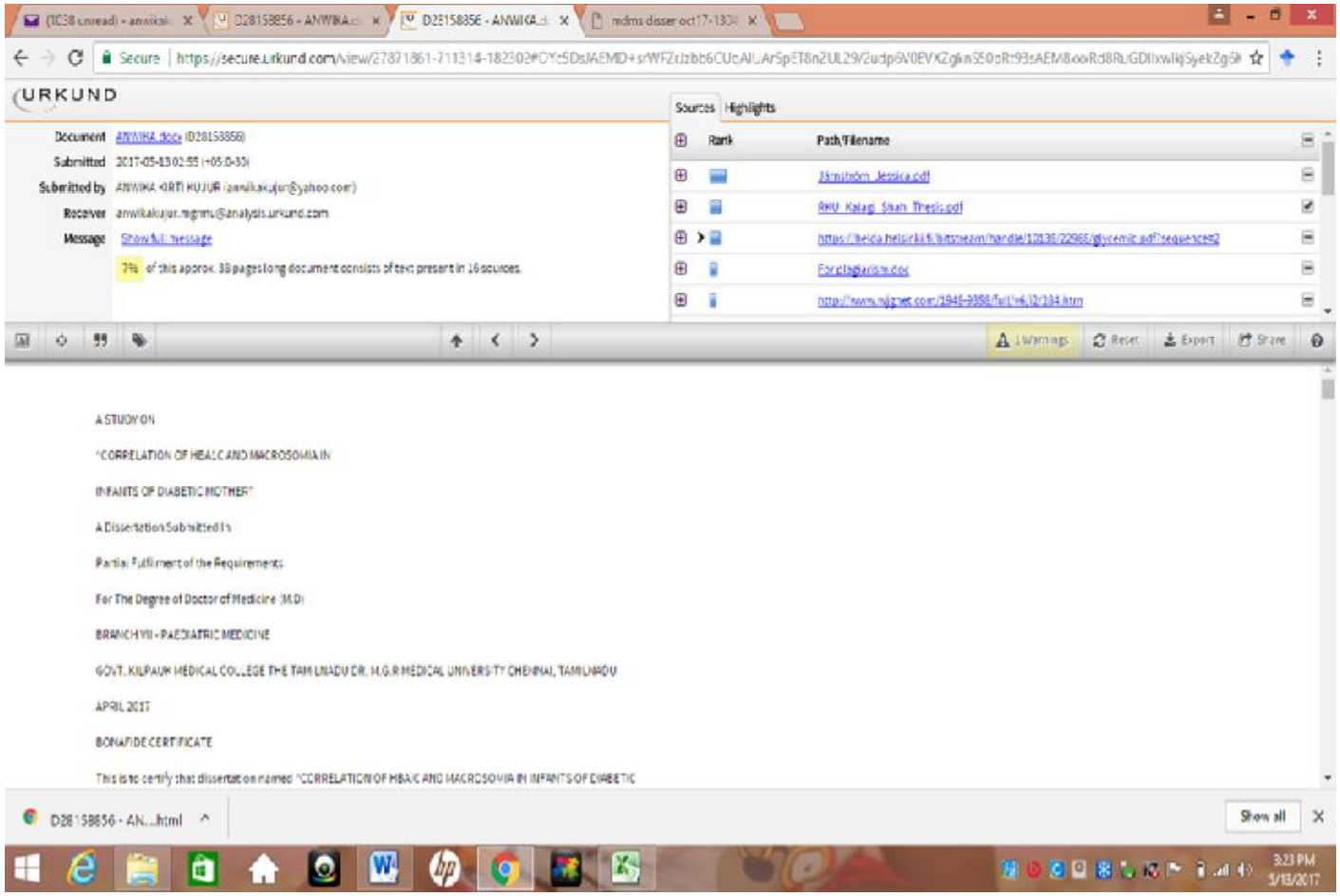
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CONTENTS

INTRODUCTION

Diabetes mellitus is a disease whose prevalence has increased dramatically over the past decades and is predicted to increase furthermore in the for seeable future, with the increasing obesity and more sedentry lifestyles. It has a varied geographical distribution. The global report on diabetes by World Health Organisation, 2016 states that the number of diabetic adults has almost quadrupled since 1980 and now there are 422 million adults living with diabetes[1]. The International Diabetes Federation has predicted that the number of adults with diabetes will increase to 642 million by the year 2040[2].

According to Global Report on diabetes by World Health Organisation estimated prevalence of diabetes in South East Asia is 8.6% i.e. 96 million adults[1]. In India there are 69.2million people with diabetes (8.7%) according to World Health Organisation data[3]. Prevalence of diabetes in Tamil Nadu is 10.4% according to ICMR-INDIAB study- 7.8% in rural population and 13.7% in urban population[4].

1 person dies due to diabetes every 6 second[5]. A disease with a rising trend and having a massive impact on our lifestyle inspite of the

available treatments it needs to be studied over and over again to put a halt to its unwanted effects on human life.

Diabetes mellitus is a chronic metabolic disorder having hyperglycemia as the cardinal feature[6]. It is a heterogenous group of disorders characterized by glucose intolerance. Uncontrolled diabetes follows a vicious cycle. Early imprinting, even the in-utero environment is responsible for causing diabetes in later life[7]. Maternal hyper glycemia causes fetal hyperinsulinemia leading to insulin resistance in childhood, which leads to impaired glucose tolerance in adulthood[8].In non-pregnant individuals, depending upon the various genetic patterns, etiologies and pathophysiology it can be classified into various types.

Broadly it is classified into

TYPE 1- beta cell destruction leading to absolute insulin deficiency

TYPE 2- insulin resistance, relative insulin deficiency, insulin defect alongwith insulin resistance

In pregnancy it is the most common medical complication[8]. Diabetes mellitus is a complication in about 1-14% of all pregnancies[9]. It is divided into :

PREGESTATIONAL or OVERT DIABETES – those having diabetes before Pregnancy.

GESTATIONAL DIABETES – diabetes having onset or first diagnosed during pregnancy.

CLASSIFICATION OF DIABETES MELLITUS[12]

TYPES	
TYPE 1	Beta cell destruction leading to absolute insulin deficiency
TYPE 2	Inadequate insulin secretion and increased insulin resistance
GESTATIONAL DIABETES MELLITUS	Diabetes first diagnosed during pregnancy
OTHERS	Genetic origin, chemical induced, drug induced or due to pancreatic disease

PREGESTATIONAL DIABETES MELLITUS:

As the number of diabetic patients is increasing, the number of pregnancies being affected by it is also on the rise. Women who are

diagnosed as diabetic during their pregnancy may well be having diabetes even before conceiving, although not diagnosed. In our set up, where people do not go for regular health check-ups, many women visit the hospital for the first time during their pregnancy. Number of women having pregestational diabetes is increasing these days.

EFFECT ON PREGNANCY

Outcome of pregnancy is related to the degree of glycemic control and also to the degree of underlying disease. Due to overt diabetes during pregnancy there are serious complications for the fetus as well as the mother.

These complications are as follows :

Spontaneous Abortion:

It is the expulsion of an embryo or fetus which weighs less than or equal to 500gm, at around 20-22wk gestation.[9] Poorly controlled diabetes mellitus causes increased number of spontaneous abortions. In an analysis of 126women there is almost 5 fold increase in pregnancy loss rate in the poor glucose control group as compared with group with fair glucose control[10].

Malformations:

Overt diabetes is an important cause of malformations in the fetus. And these malformations are often responsible for perinatal death in infant of diabetic mothers. It is thought that the poor control of diabetes in the periconceptional period is responsible for these malformations as organogenesis takes place in the first 3 months of pregnancy. Poor glycemic control in the mother causes alteration in lipid metabolism, production of excess superoxide radicals which is toxic and programmed cell death activation. All these may explain the malformations caused in the fetus.

Preterm Delivery:

Delivery of the baby before the 37^{th} completed week of gestation[9] is termed Preterm Delivery. It is an important outcome of overt diabetes. Some are spontaneous but most of them are indicated preterm deliveries due to medical or obstetrical complications. In women with diabetes mellitus, rate of preterm delivery is 38%[11].Another problem is that the antenatal steroids given in preterm delivery in diabetic patientslead to deterioration in glycaemic control thus requiring an increased dose of insulin[12].

Altered Fetal Growth:

Fetal overgrowth is typically found in pregestational diabetes.

However, there may be diminished fetal growth due to congenital anomalies or advanced maternal vascular disease causing substrate deprivation. Due to maternal hyperglycemia, there is fetal hyperinsulinemia, which in turn stimulates somatic growth in excess and results in macrosomia. Macrosomia is characteristic of the fetus of a diabetic mother. MACROSOMIA – birth weight which is more than the 90^{th} percentile or fetus weighing >4000gm[13]. These infants are anthropometrically different from large for gestational age babies[14]. Most fetal organs are affected by it except brain. Those infants with diabetic mother have excess fat deposition on shoulders and trunk. This predisposes to primary caesarean section and obstetric trauma in the form of fractured clavicle or phrenic nerve palsy or Erb's palsy due to shoulder dystocia. Macrosomia is said to be associated with third trimester maternal hyperglycemia, fetal and neonatal hyperinsulinemia and neonatal hypoglycemia. In diabetic group there was disproportionate growth between head circumference and abdominal circumference which ultimately led to macrosomia[15]. And this accelerated growth of the fetus is more evident in women who show poor glycemic control.

Unexplained Fetal Demise:

In diabetic women there is risk of fetal death three to four times higher than in general obstetrical population[16]. There is no identifiable cause for it hence unexplained. Common causes of fetal death like hydramnios, placental insufficiency, abruption placenta or fetal growth restriction are not identified. These are large for gestational age infant who typically die before labour starts, generally after 35 weeks of gestation or later[17]. Lauenborg et al (2003) discovered suboptimal glucose control in 2/3 of diabetic women who had unexplained stillbirth. They also have increased lactic acid levels. In diabetic women the infants had a lower umbilical venous blood pH and it was related to fetal insulin levels significantly[18]. There is chronic alteration in oxygen and metabolite transport in the fetus due to hyperglycemia [19]. It may be one of the causes of stillbirth. Apart from this, women having overt diabetes have increased association with pre-ecclampsia. Pre-ecclampsia leads to placental insufficiency which may result in fetal death. Women with advanced disease and having vascular complications also have increased incidence of stillbirth. Fetal demise can also be caused by maternal ketoacidosis.

Hydramnios:

Hydramnios is another complication of diabetic pregnancy. It is also known as Polyhydramnios. Hydramnios is defined anatomically as a state in whichliquor amnii is more than 2000ml. Sonographically when amniotic fluid index isgreater than 24 cm and the largest vertical pocket is more than 8 cm, it is diagnosed as hydramnios. Clinically when this excess of liquor amnii causes discomfort to the patient or an imaging is required to confirm the lie and position of the fetus. Only the quantity of liquor amnii is increased but the composition remains normal. It may be either due to excessive production or defective absorption of liquor amnii. It is associated with diabetes in 30% of case[9]. Although not proven, mostly the cause for this is raised maternal glucose level causing an increase in fetal blood sugar. As a result there is fetal diuresis leading tohydramnios. This leads to increased incidence of pre-eclampsia, malpresentation, premature rupture of membrane, preterm labour and accidental haemorrhage. During labour it can result in early rupture of membranes, cord prolapse, uterine inertia, retained placenta or postpartum haemorrhage and shock. It also causes subinvolution& increased morbidity in the puerperium. Perinatal mortality is increased upto 50%.

EFFECT ON THE NEONATE:

Maternal diabetes not only causes neonatal death but alsoincreases neonatal morbidity by a great deal. The following are the effects of overt diabetes on the newborn.

Hypoglycemia:

It is defined as blood glucose level less than 40mg/dl in an infant of any gestational age with or without symptoms[13]. It is seen within 1to 2 hours after birth and mostly in macrosomic babies. Pederson hypothesis of maternal hyperglycemia leading to hyperinsulinemia in the fetus explains the pathophysiology of hypoglycemia in infants of diabetic mother. Correlation is established between fetal macrosomia, neonatal hypoglycemia and elevated maternal and cord blood Hba1c. There is also a correlation between cord bloodinsulin levels and hypoglycemia in newborn. Mother who receive large doses of glucose before or at the time of delivery, cause stimulation of the insulin response in the hyperinsulinemic newborn leading to further hypoglycemia. Diabetic mothers who have vascular disease deliver small for gestational age babies who have inadequate glycogen stores, thereby show hypoglycemia at 12-24hrs of age.

In infants of diabetic mother there is decreased hepatic glucose production and oxygenation of fatty acids is also diminished. As a result there is inadequate substrate mobilization for glucose production. In addition to this, there is decreased catecholamine& glycogen secretion which also contribute to hypoglycemia.

It can present as asymptomatic hypoglycemia or may show symptoms like lethargy, apnea , respiratory distress , shock , cyanosis or seizures. Symptomatic babies are at greater risk for sequelae. Asymptomatic infants of diabetic mother are treated with oral feeding. Symptomatic infants are treated with parentral glucose. Blood glucose is monitored at regular intervals and low blood glucose is subsequently attended to by giving dextrose infusion. Hydrocortisone intramuscularly is used in difficult cases. Although persistent hypoglycemia may occur due to continued hyperinsulinemia , hypoglycemia lasting for more than 7 days warrants the search for other etiologies.

Hypocalcemia :

It is defined as total serum calcium less than 7mg/dl or ionized calcium concentration less than 4mg/dl[13]. It is seen in 25% to50% of infants of diabetes mother if maternal glucose is not under control. It is not related to hypoglycemia. It usually occurs on 2^{nd} or 3^{rd} day after birth.

Calcium homeostasis is regulated primarily by parathyroid hormone and calcitriol i.e. 1,25- dihydroxy vitamin D. when calcium level goes down in extra cellular fluid, parathormone mobilizes calcium from bone, stimulates renal production of calcitriol and increases calcium resorption in renal tubules. Thus calcium level rises. In infants of diabetic mother there is a delay in the rise of parathormone in the postnatal period resulting in hypocalcemia.

Vitamin D stored in the liver is transported to kidney and converted to calcitriol. This calcitriol helps in intestinal absorption of calcium &phosphate and also mobilizes them from bone. In infants of diabetic mother, dueto abnormal vitamin D metabolism, there is hypocalcemia. Other causes of hypocalcemia in infants of diabetic mother are hypercalcitonemia, hyperphosphatemia, hypoparathyroidism, prematurity and asphyxia. Symptomaticinfants of diabetic mother or other sick babies who do not respond to blood glucose correction, need serum calcium assessment. Calcium is supplemented as required.

Respiratory Distress Syndrome :

The incidence of Respiratory Distress Syndrome in infants of diabetic mother has come down from being 28% in 1950s to about 4% in the 1990s. it is due to better management of pregnancies. Thus nowadays

more pregnancies in diabetic women reach term and there are more vaginal deliveries, henceforth reducing the cases of respiratory distress syndrome. Mostly death from respiratory distress syndrome occurs in newborns< 35 week gestation who are delivered by caesarean due to maternal or fetal indication[13]. In infants of diabetic mother, there is delayed lung maturity as hyperinsulinemia blocks the induction of lung maturation by cortisol. Apart from respiratory distress syndrome, there can be other causes of respiratory distress among infants of diabetic mother such as due to cardiac or pulmonary anomaly, transient tachypnea of the newborn, hypertrophic cardiomyopathy or polycythemia. Infants of diabetic mother can alsohave respiratory distress also due to pneumonia and pneumothorax of diaphragmatic hernia.

Polycythemia :

It is commonly seen in infants of diabetic mothers. Glycosylated haemoglobin has a high affinity for oxygen, hence there is reduced oxygen delivery to the tissue. In women with elevated Hba1c levels, there is reduced oxygen supply in both maternal and fetal circulation. This leads to fetal hypoxia. In small for gestational age babies, there is fetal hypoxia due to placentalin sufficiency. This hypoxia as well as insulin like growth factors lead to increase in fetal erythropoietin production in

the fetus and red cell production. Blood shifts from placenta to the fetus in case of fetal distress. All these are causes of polycythemia in the infants of diabetic mother. Hematocrits upto 60-70 volume% have been seen in 40% of infants of diabetic mother[18].

Hyperbilirubinemia:

Hyperbilirubinemia is said when bilirubin level is more than 15mg/dl. As there is polycythemia , there is an increase in bilirubin load in the infants of diabetic mothers resulting in hyperbilirubinemia. Due to glycosylation of red blood cells, the cell membrane of erythrocytes become less deformable &hence their life span decrease. This also contributes to increased hemolysis resulting in increased bilirubin production. Other causes of hyperbilirubinemia can be prematurity, impaired hepatic conjugation of bilirubin and also enterohepatic circulation which increases due to feeding problems in infants of diabetic mother.

Cardiomyopathy :

Infants of diabatic mother have myocardial dysfunction as one of the many problems. There was diastolic dysfunction in the fetus of the diabatic mother, seen in the first trimester of pregnancy. In the third

trimester there was thickening of the right ventricular wall and the interventricular septum in these fetuses[20]. Thus they concluded that there was cardiac dysfunction preceding the structural changes. There is a transient hypertrophic subaortic stenosis in infants of diabetic mothers, which results from the ventricular septal hypertrophy. In severe cases it may result in obstructive cardiac failure. Although most of the affected babies are asymptomatic, they may present with symptoms like poor cardiac output, cardiomegaly and heart failure. This myocardial dysfunction also complicates the management of other illnesses in the neonate like respiratory distress syndrome. On echocardiography it is characterized by hypertrophy of ventricular septum, hypertrophy of the right anterior ventricular wall also left posterior ventricular wall and absence of chamber dilatation. As the septal thickness increases, cardiac output goes on decreasing. As these are transient changes, the symptoms are resolved by two weeks after birth and the hypertrophy takes four months to resolve. Many babies are asymptomatic. Symptomatic babies show response to supportive care like oxygen & furosemide & propranolol. Inotropes are contraindicated unless there is an evidence of myocardial dysfunction on echocardiography. Relief from maternal hyperglycemia is thought to cause the resolution[21]. Although it may progress to cardiac disease in adulthood . Good glucose control during

pregnancy can reduce the incidence and also the severity of this cardiomyopathy.

Cognitive Development :

Metabolic condition in the intra-uterine life has long been linked to neurodevelopment in children. There was correlation found between maternal glucose levels and intellectual performances of children upto the age of 11yrs in a study, despite rigorous antepartum management[22]. Several other studies demonstrated that children of mothers, who had diabetes during pregnancy, had lower intellectual level than the controls[23,24]. Among Autism Spectrum Disorders, children of diabetic mothers had Muller Scales of Early Learning scores lower than those children whose mothers had no metabolic diseases[25]. In the presence of postnatal environment and events, it is difficult to interpret cognitive effects on children due to maternal glucose control during pregnancy, but surely, some studies have data to support a connection between neurocognitive outcome in off springs and maternal diabetes and glycemic control.

Inheritance of Diabetes :

It is of prime concern to the parents whether their childwill have diabetes in future or not. There is 1% to 4% risk of developing diabetes in the child if only mother has type 1 diabetes. If father also has type 1 diabetes, risk to the child is increased to 10%. There is 20% risk of diabetes to the child if both the parents have type 1 diabetes. Type 2 diabetes has a greater genetic component. There is a 30% risk of the disease if one of the parents is diabetic, whereas it rises to 50% to 60% if both the parents have type 2 diabetes. Breast-feeding by diabetic mothers is also said to cause childhood diabetes[26]. However, breastfeeding has been linked to reduced type 2 diabetes risk[27].

Poor Feeding :

It is also seen commonly in infants of diabetic mother. Sometimes it may be related to other problem in the baby like respiratory distress syndrome or prematurity. However, it is also present in the absence of such problems in infants of diabetic mother. It is an important cause for prolonged hospital stay of infants of diabetic mother.

Renal Vein Thrombosis:

It occurs in infants of diabetic mother and is often seen inthevery first week of life. Thrombosis formation can occur in utero or postpartum.

It usually affects term neonates have predisposing factors like polycythemia, hypercoagulable states and hypovolemia. It can be associated with other illnesses such as perinatal hypoxia , hypotension , cyanotic congenital heart disease. It presents as haematuria with a flank mass, hypertention, thrombocytopenia, proteinuria and renal dysfunction. The neonate may also have vomiting, edema of lower extremeties, abdominal distention and shock. Coagulation studies are deranged and fibrin degradation products are increased. The thrombus may extend into the inferior venecava , or have complications like adrenal haemorrhage.

Bilateral disease is seen in 30% of cases[13]. USG conforms diagnosis showing diffuse homogenous hyperechogenicity in an enlarged kidney. Dopplers study show thrombi causing absent renal flow. Depending upon the extent of thrombosis,its management differs from supportive care , anticoagulation or thrombolysis. Hypertension is also treated. Not only renal vein thrombosis but other thrombosis can also occur in infants of diabetic mother.

Small Left Colon Syndrome:

It is also seen in infants of diabetic mothers. It presents as generalized distention of the abdomen due to inability to pass meconium. When a rectal catheter is passed, meconium is passed. Meglumine Diatrizoate (gastrograffin) given as enema results in the evacuation of colon and conforms the diagnosis. Thereafter in the first week of life , the baby has difficultyin passing stool. It is treated with half-normal saline enemas or glycerine suppositories and it resolves.

EFFECT ON THE MOTHER

During pregnancy carbohydrate metabolism is altered and insulin action is impaired. As a result it becomes very difficult to stabilize the maternal blood glucose level. The human placental lactogen, progesterone , estrogen, free cortisol and insulin degradation by placenta, all these combine to result in insulin antagonism during pregnancy. Blood estimation of glucose is required as urine test for glucose becomes unreliable due to physiological glycosuria during pregnancy. During pregnancy even short duration of fasting can lead to rapid lipolysis. Ketoacidosis can also be precipitated during vomiting in early pregnancy or fasting during labour or any infection. Sometimes corticosteroids or beta-sympathomimetics used during labour can also precipitate

ketoacidosis. All other vascular diseases due to diabetes version during pregnancy as glycemic control keeps fluctuating. Once the baby is delivered insulin requirement decreases drastically. Maternal death due to diabetes in pregnancy is uncommon however has increased incidence. Deaths may be due to hypertention, infection, hypoglycemia or diabetic ketoacidosis. Especially women with ischaemic heart disease have high mortality[8].

GESTATIONAL DIABETES MELLITUS

It is defined as the carbohydrate intolerance of variable severity, with an onset or first recognition duringpregnancy[28]. The word "gestational" means 'induced by pregnancy' i.e. due to physiological changes occurring in glucose metabolism in pregnancy. The women may or may not be on insulin. It also includes women who previously had overt diabetes but it was undiagnosed. It is important to diagnose gestational diabetes mellitus because it results in fetal macrosomia, which can cause birth trauma to both mother as well as the baby. However, on proper management , gestational diabetes mellitus has a good outcome. Women diagnosed with gestational diabetes mellitus, more than half of them , ultimately develop overt diabetes in coming years. Offsprings of gestational diabetes mellitus mothers suffer from obesity and diabetes in

their later life. Gestational diabetes mellitus can be suspected in the following patients[29] :

- those having a family history of diabetes
- history of birth of a big baby
- history of stillbirth which showed pancreatic islet cell hyperplasia on autopsy
- unexplained fetal death
- polyhydramnios in present pregnancy
- presence of recurrent vaginal candidiasis in pregnancy
- persistent glycosuria
- obesity
- more than thirty years of age
- having East Asian , Pacific Island ancestery

EFFECTS ON FETUS AND MOTHER

There is a slight difference in the effects on fetus due to gestational diabetes mellitus from the effects due to pre-gestational diabetes. The incidence of fetal malformations is not so much increased in gestational diabetes mellitus as there is no metabolic disturbance during organogenesis. However, incidence of maternal hypertension and

caesarean delivery is increased similar to pre-gestational diabetes. There is a reccurence of gestational diabetes mellitus in the subsequent pregnancies in around 50% cases[9]. Risk of developing overt diabetes is also increased in 50% of patients in the following years[9].

Fetal Macrosomia :

Macrosomia is defined as birth weight more than 90thpercentile or fetus weighing >4000 grams[13]. The risk factors for fetal overgrowth are obesity, diabetes mellitus, advancing maternal age , multiparity, post term gestation , previous macrosomic baby, large size parents and racial and ethnic factors[8]. The growth and development of the fetus is determined by provision of substrate by the mother, placental transfer of the substrate and the growth potential of the fetus determined by the genome. Insulin like growth factor -1 plays an important role in regulation of the growth of the fetus. In pregnancies complicated by gestational diabetes mellitus , there is an excessive growth of the fetus resulting in macrosomia. Maternal hyperglycemia leads to hyperinsulinemiain the fetus which stimulates excessive growth in the fetus. There is increased neonatal fat mass and also morphological heart changes in infants of diabetic mothers and also they have increased insulin like growth factor-1 in cord blood levels. There are other factors such as fibroblast growth factor , epidermal

growth factor, platelet derived growth factor , leptin and also adiponectin which are implicated in macrosomia[30,31,32]. There is an excessive transfer of lipids to the growing fetus which also results in fetal overgrowth[33]. Increased levels of maternal triglycerides also result in increased birthweight[34,35]. Higher placental levels of omega-3 fatty acids, associated with increase in trophoblastic lipase activity is also seen in overgrown infants[29]. There is excessive fat deposition in shoulder and trunk of macrosomic babies. This predisposes them to birth trauma during delivery such as fractured clavicle , phrenic nerve palsy or Erb'spalsy due to shoulder dystocia. It is also a cause for primary caesareansection delivery in a quite a number of patients. In mothers delivering macrosomic babies there is increased incidence of postpartum haemorrhage , maternal infections and perineal lacerations.

Neonatal Hypoglycemia :

Neonatal hyperinsulinemia leads to neonatal hypoglycemia soon after birth. It is defined as blood glucose level less than40mg/dl with or without symptoms in an infant of any gestational age[13]. The HAPO study – 'Hyperglycemia and Adverse Pregnancy Outcome' Study showed that incidence of neonatal hypoglycemia increased with increase in maternal oral glucose tolerance test values[36]. Likewise , insulin levels in the cord

blood are directly proportional to the maternal glycemic level[37]. Thus mothers having poor glycemic control have more chances of having hypoglycemia in their newborn babies.

Maternal Obesity :

Maternal obesity is an independent risk factor for macrosomiain fetus. It is regardless of the maternal glucose control during pregnancy. Maternal obesity is also related to gestational diabetes. Prevalence of gestational diabetes mellitus increases with increase in body mass index[38]. In women with truncal obesity, there is increased risk of gestational diabetes mellitus, so weight distribution also has a role to play. Measurement of maternal abdominal subcutaneous fat thickness by ultrasound at 18-22 weeks gestation is correlated with body mass index , also a better indicator of gestational diabetes mellitus[39].

There is also excessive weight gain during pregnancy in gestational diabetes mellitus patients and it acts as an additional risk factor for macrosomia.

DIAGNOSIS

Overt Diabetes:

Women having random blood sugar level>200mg/dl in addition to classical signs and symptoms of diabetes like polydipsia, polyuria and unexplained weight loss.

- Women having fasting glucose level>125mg/dl(ADA 2012)
- Women with very high blood glucose levels or
- having glycosuria or having ketoacidosis

The International Association of Diabetes and Pregnancy Study Groups (IADPSG) consensus panel (2010) has recommended the following to diagnose overt diabetes in pregnancy:

PARAMETER	THRESHOLD(12)
Fasting plasma glucose	>=7.0mmol/l (126mg/dl)
Random plasma glucose	>=11.1 mmol/l (200mg/dl)
Hba1c	>=6.5%

Women having family history of diabetes, previously having a large baby or unexplained fetal loss or persistent glucosuria are at high risks of carbohydrate metabolism during pregnancy.

Gestational Diabetes:

There is a lot of disagreement in the optimal screening test for Gestational diabetes. Screening must be performed either at the first antenatal visit or between 24 to28 weeks of gestation. 75 gms 2 hours oral glucose tolerance test (OGTT) is recommended by World Health Organisation (1998) and American Diabetes Association (2013). For low risk women i.e. no risk factors for gestational diabetes , routinely testing of blood glucose is not recommended. For average risk women i.e. presence of some risk factors for gestational diabetes mellitus , either they can take 100grams oral glucose tolerance test (OGTT) – diagnostic test, or , first take a 50 grams glucose challenge test and if they cross the threshold, take the 100 grams OGTT- diagnostic test. In high risk women,100 grams oral glucose tolerance test (diagnostic test) should be performed as soon as possible[8].

Glycosylated Haemoglobin - HBA1C:

When blood glucose enters the red blood cells, it glycosylates E-amino group lysine residues and also amino terminals of haemoglobin. Normally about 5% of haemoglobin is glycosylated. The fraction of haemoglobin which is glycosylated is proportional to glucose concentration in the blood. Half life of a red blood cell is 60 days. So , the level of Hba1c reflects the blood glucose concentration in the preceeding 6-8 weeks[40]. Therefore by measuring the Hba1c level in blood of the patient , we can determine the glucose concentration in blood in the previous 6-8 weeks and manage the diabetic patients appropriately.

Diabetes mellitus has become a common disease these days. One in eleven adults has diabetes[5]. Inspite of effective management tool to monitor and control the blood sugar available, there is a steady rise in the number of pregnancies complicated by diabetes. Among the various diagnostic tools available to diagnose diabetes, estimation of glycosylated haemoglobin (Hba1c) is used in this study. According to “Avery’s Disease of the Newborn” in patients with untreated gestational diabetes upto 20% of infants may be macrosomic. Macrosomia is also, upto certain extent, a cause for primary caesarean section and traumatic vaginal delivery.

Here in this study, we try to find a correlation between glycosylated haemoglobin level in the mother and macrosomia in the infant of diabetic mother.

Hba1c reflects the blood glucose control over the last 4-6 weeks. According to “Textbook of Obstetrics” by D.C.Dutta , Hba1c level of less than or equal to 5.6% is desirable as it indicates adequate glucose control. There have been various studies which show associations between elevated third trimester maternal glucose levels and macrosomia. The cause of macrosomia in infants of diabetic mother is said to be the poor glycemic control in the third trimester of pregnancy. However,there are other studies where no statistical correlation has been found between thefetal body weight and the level of glycosylated haemoglobin in the mother. There has been no such study in this hospital relating macrosomia and Hba1c levels.

Hence, this study is taken up to get an idea of the scenario prevailing here regarding this topic. This will help us to understand the cause of macrosomia and find possible ways to reduce its occurrence.

AIM OF THE STUDY

To study the correlation between HbA1c and macrosomia in infant of diabetic mother.

REVIEW OF LITERATURE

The implications of diabetes in a pregnant mother and the perinatal outcome has been studied since long.

In a retrospective study which compared 287 macrosomic neonates with 284 weight appropriate for age term size babies, **Modanlou et al**[41] found that perinatal morbidity and mortality were found to be higher for macrosomic baby as compared to appropriate weight for age term size babies.

In their study "Macrosomia in Pregnancy Complicated by Insulin Dependent Diabetes Mellitus" **Michael Small et al**[42] found that the maternal glycosylated haemoglobin at the time of delivery correlated with percentile birth weight ratios ($r = 0.43$, $p < 0.001$) and implied that around 18% of variance in birth weight was due to glycemic control of the mother in the third trimester. Pregnancies resulting in macrosomia were also characterized by accelerated fetal growth and biochemical (although asymptomatic) hypoglycemia in the neonate. They concluded that factors other than maternal glycemic control might contribute to occurrence of macrosomia.

RT Hearty et al[43]in their study conducted in the year 2000 , observed a significant linear trend for the incidence of large for gestational age infants with increasing hyperglycemia.

RiittaLuotoet al[44] conducted a cluster randomized trial in Finland where 2271 women were screened by oral glucose tolerance test at 8-12 week gestation. 399 euglycemic women were included in the study and they had atleast one risk factor for gestational diabetes mellitus. The intervention used by them included individual rigorous counseling on doing physical activity, diet control and weight gain at 5 antenatal visits. It was found that neonatal birth weight was lower in the intervention group than in the other group which had been offered usual care. Proportion of large for gestational age babies was also less 26/216 (12.6%) in the intervention group as compared to 34/179 (19.7%) p=0.042 in the usual care group.

Hayfaa Wahabi et al[45] recently published a study "Prevalence and Complications of Pregestational and Gestational Diabetes in Saudi Women: Analysis from Riyadh Mother and Baby Cohort Study(RAHMA)". A total cohort of 9723 women among which 2353 (24.2%) had gestational diabetes mellitus, 418 (4.3%) had pre-gestational diabetes mellitus and 6951 were non-diabetic. Women having gestational

diabetes mellitus had increased odds of having a macrosomic baby (Odds Ratio : 1.65, 95% Confidence Interval : 1.32 – 2.07). Babies born to mothers with pre-gestational diabetes mellitus were more likely to be Macrosomic(Odds Ratio :2.40, Confidence Interval : 1.50-3.80).

All of the above literature suggests that even though use of insulin for glycemic control in diabetic pregnancies had been started long back and mortality and morbidity of mother greatly reduced , yet for the infant of diabetic mother there were still many problems. Even to this date , the same problems exist although reduced slightly in frequency due to the modern era neonatal intensive care units. Still we need to find the exact cause and ways to prevent these complications in the newborns of diabetic mother, one of them being Macrosomia.

There are numerous articles suggesting that Macrosomia is due to poor glucose control in the third trimester of pregnancy.

R. A. Gandhi et al[46] in their study "HbA1c during pregnancy : Its relationship to meal related glycaemia and neonatal birth weight in patients with diabetes" aimed to find the strength of association between mean home blood glucose levels and HbA1c and whether HbA1c and glycaemic control affects birth weight of the newborn. They observed that neonatal birth weight increased with the higher levels of HbA1c.

Neonatal birth weight (percentiles) +/- SD for HbA1c<6.5% was 78.9% +/- 29.2% as compared to that for HbA1c>6.5% neonatal birth weight percentiles +/-SD was 90.2% +/-18.6%. p=0.02.

The HAPO Study (Hyperglycaemia and Adverse Pregnancy Outcomes) was conducted at 15 centres in nine countries to reach an internationally agreed upon diagnostic criteria for the gestational diabetes mellitus. A total number of 23,316 pregnant women participated in the HAPO study. The authors observed strong , continuous associations between maternalglucose level and increased birth weight.

C Andrew Combs[47] in his longitudinal study on 111 consecutive pregnancies in diabetic women from 13 weeks to 36 weeks of gestation found that macrosomia was related to higher post prandial glucose levels upto 32 weeks gestation although fasting blood glucose was not related at all. Macrosomia was significantly associated with post prandial glucose levels between 29 weeks and 32 weeks of gestation.

BalajiBhavadharini et al[48] from Department of Epidemiology, Madras Diabetes Research Foundation, Chennai, TamilNadu, India conducted a study on 1459 pregnant women from TamilNadu, South India. They diagnosed gestational diabetes mellitus in 195 women following the International Association of the Diabetes and Pregnancy

Study Groups criteria. They found that HbA1c> 5.0% (>= 31 mmol/mol) had a sensitivity of 66.2% and 56.2% specificity for diagnosing gestational diabetes mellitus. In those patients who had HbA1c>5.0% , the adjusted Odds Ratio for Macrosomia was 1.92(CI : 1.24 – 2.97, P=0.003). Those patients with HbA1c >5.0% were also significantly older, had higher body mass index, history of previous gestational diabetes mellitus and more number of Macrosomia.

Even the standard textbooks support this theory that occurrence of macrosomia depends upon the glycaemic control in the third trimester. To quote from 7th edition of "**Manual of Neonatal Care"**[13] by John P Cloherty, Eric C Eichenwald, Anne R Hansen, Ann R Stark which states that associations have been found between macrosomia and third trimester elevated levels of blood sugar in the mother. However, studies by various researchers contradict this theory. They are unable to prove this hypothesis.

A Lapolla et al[49] in their study "Can Plasma Glucose and HbA1c predict Fetal Growth in Mother with Different Glucose Tolerance Levels?" tried to determine whether plasma glucose and HbA1c was able to predict abnormal fetal growth. They concluded that HbA1c at 24-27 weeks of gestation did not predict the fetal overgrowth.

Taylor et al[50] in their study "Clinical Outcomes of Pregnancy in Women with Type 1 Diabetes" evaluated the predictors of macrosomia and neonatal hypoglycaemia in 107 consecutive diabetic pregnancies. They found no relationship between mean glycosylated haemoglobin values and birth weight.($R=0.02$, $p>0.1$)

A Weissman – Brenner[51] conducted a study on non-diabetic pregnant women. The results showed that HbA1c level in women who delivered macrosomic baby was 5.3+/-0.7% and those who delivered non-macrosomic neonates was 5.2+/- 0.5% ($p=0.27$). the area under ROC curve for prediction of macrosomia by HbA1c levels was 0.53 ($p=0.27$). They concluded that HbA1c levels in mother was not useful to predict the birth weight of the baby.

GC Penny et al[52] published an article "The Relationship between Birthweight and Maternal GlycatedHaemoglobin (HbA1c) concentration in pregnancies complicated by Type 1 Diabetes" in 'Diabetic Medicine' a UK diabetes journal. They conducted a prospective study in Scotland on women with pre-existing diabetes. They collected HbA1c levels of women prior to conception and also at the three trimesters of pregnancy and the birth weight of their newborns. Analysis of the data showed that standardized birth weight scores of the newborns of diabetic mothers

were higher than reference population. Between pre-conception HbA1c levels and birth weight , there was a significant negative correlation (Spearman's R: -0.208 ;p=0.016). No statistically significant correlation was found between third trimester HbA1c levels and birth weight of the newborn.

Birol Binbir et al[53] conducted a study to determine the effects of maternal HbA1c levels and umbilical cord thickness on the birth weight of infants born to diabetic pregnant patients. Maternal HbA1c levels and macrosomia did not show any statistically significant relationship (p=0.701).

This study was an effort towards finding a probable cause for macrosomia in infants of diabetic mothers. The parameter used to assess the blood glucose level was glycosylated haemoglobin (HbA1c) as by a single value it gives a picture of the glucose control of the previous three months. It is also not affected by the immediate fasting or eating by the patient at the time of taking the blood sample.

MATERIALS AND METHODS

This study is an observational type of study done at Govt. Kilpauk Medical College and Hospital, Department Of Paediatrics. The study protocol was approved by Ethical Committee for research Studies of Govt. Kilpauk Medical College and Hospital.

STUDY DESIGN:

Cross sectional study

STUDY PERIOD:

February 2016- July 2016

STUDY POPULATION:

Newborn babies of mothers who were already diagnosed as diabetic, and delivered in Government Kilpauk Medical College.

SAMPLE SIZE

Sample size was taken as 230. It was calculated from the following formula:

$$\text{Sample size} = \frac{Z_{1-\alpha/2}^{2} p(1-p)}{d^2}$$

Here

Alpha level is at 5% (Z = 1.96)

P = 65, (proportion of babies with macrosomia having mother's HbA1c levels high).

D = Desired Precision = 6.5, (10% of the proportion)

INCLUSION CRITERIA:

1. Babies born to mothers who are diagnosed as pre gestational diabetes mellitus and gestational diabetes mellitus.
2. Singleton pregnancy.

EXCLUSION CRITERIA:

1) Newborns born with congenital anamolies.
2) Infants of mothers having any other co-morbid conditions apart from diabetes mellitus.

MATERIALS USED IN THE STUDY:

1) HemoCue HbA1c 501 Analyser
2) Electronic Weighing Scale

METHODOLOGY :

After getting the approval from the institution and Ethical Committee of Government Kilpauk Medical College and Hospital, Chennai, the study was started. Permission was sought from the Head of Department of Gynaecology & Obstetrics, Government Kilpauk Medical College and Hospital, Chennai for enrolling the diabetic mothers into study. Pregnant women who were diagnosed as having overt diabetes or gestational diabetes were invited to take part in the study.

This study, done on their newborn babies and them was explained in detail to them. An informed consent was obtained. Pregnant women fulfilling the inclusion criteria were listed. When these mothers delivered, their blood samples were taken to measure HbA1c levels. The women were classified into two categories as having pre-gestational and gestational diabetes mellitus. Their body mass index, weight gain during pregnancy, duration of diabetes mellitus and type of treatment for diabetes were recorded. The newborn's birth order, mode of delivery, gestational age and birth weight were recorded. All this data was then analysed.

RESULTS AND OBSERVATIONS

A total of 230 diabetic pregnant women and their newborns were enrolled for the study. Out of them 2 patients had missing data. So data from a total of 228 diabetic pregnant women were analysed. Bivariate and Multivariate analysis was done. The Probability of <0.05 was taken as significant (alpha= 0.05; C.I.=95%). The discrete variables were analysed by Chi square test and the continuous variables were analysed by t – test. The analysis was done by SPSS 17 software.

AGE GROUP * MACROSOMIA

AGE GROUP	MACROSOMIC BABIES	PERCENTAGE
Upto 25 years	**10**	**55.6%**
25-30 years	**6**	**33.3%**
31 years and above	**2**	**11.1%**

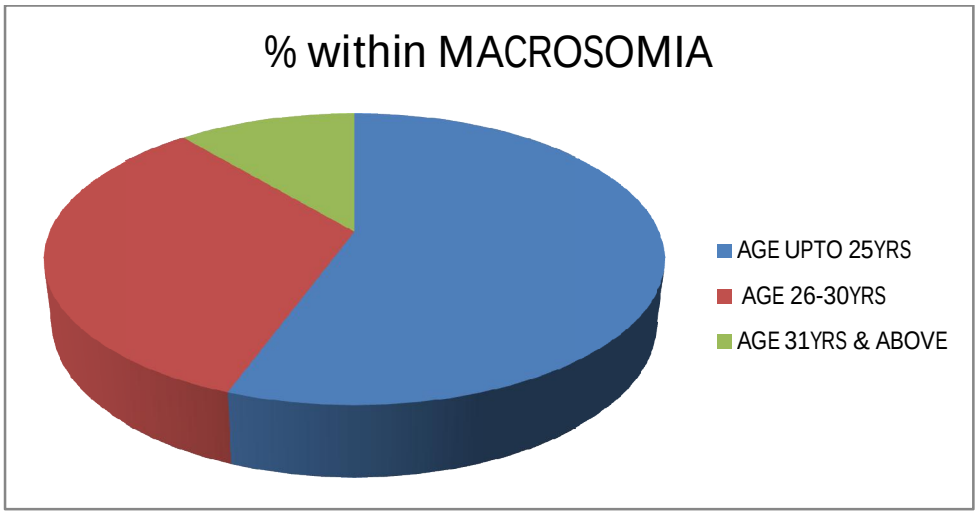


Figure 1 : AGE OF WOMEN HAVING MACROSOMIC BABIES

The women were divided into three groups according to age group.

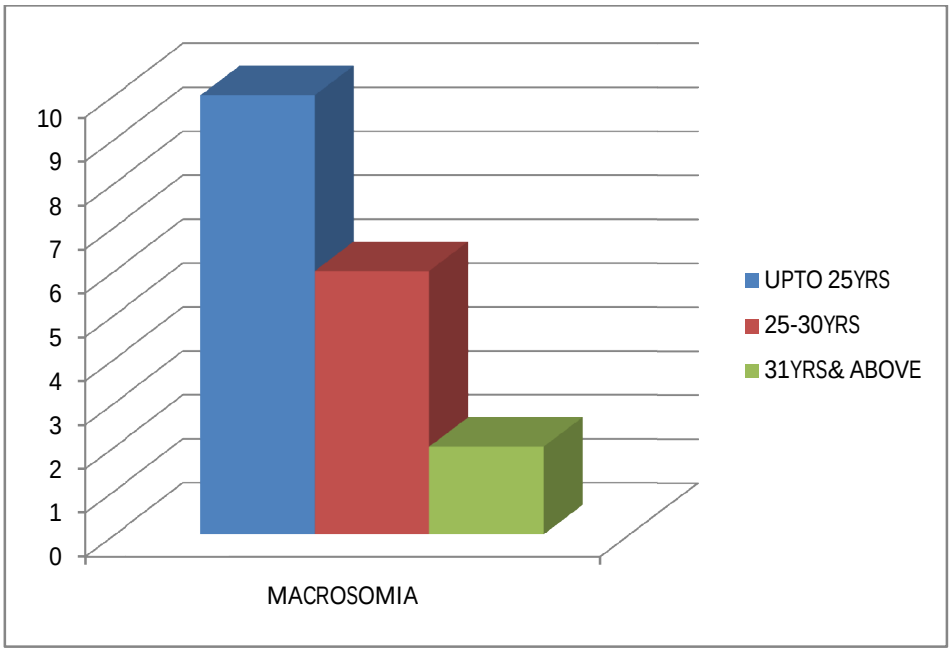
a. Upto 25 yrs of age – there were a total of 117 women in this group Out of them 10 women had MACROSOMIC baby 55.6% of the MACROSOMIA occurred in this group

b. 26yrs – 30yrs of age – there were a total of 100 women in this group Out of them 6 women had MACROSOMIC baby 33.3% of the MACROSOMIA occurred in this group

c. Above 31 yrs of age – there were a total of 11 women in this group Out of them 2 women had MACROSOMIC baby11.1% of the MACROSOMIA occurred in this group PEARSON CHI-SQUARE = 2.163

P VALUE =0.339

Not Significant


10
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MACROSOMIA
UPTO 25YRS
25-30YRS
31YRS& ABOVE

BMI GROUP * MACROSOMIA

BMI	MACROSOMIC BABIES	PERCENTAGE
15 TO 22	2	11.1%
22 TO 25	7	38.9%
BMI>25	9	50%

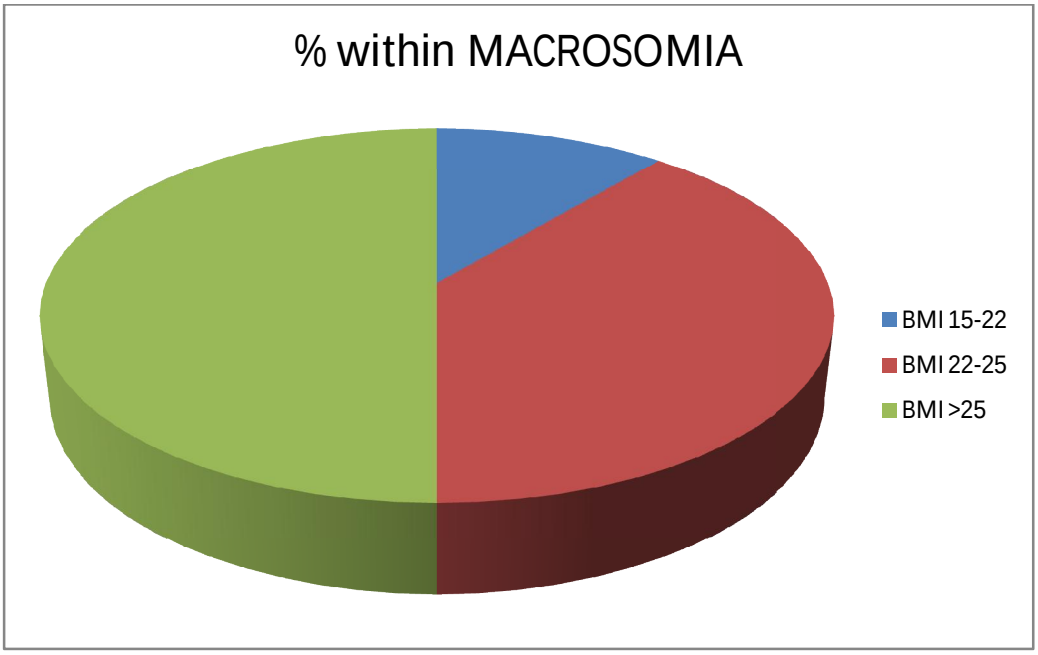


Figure 2: BMI OF WOMEN HAVING MACROSOMIC BABIES

The women were divided into three groups according to BMI group.

a) BMI 15-22–there were a total of 111 women in this group Out of them 2 women had MACROSOMIC baby11.1% of the MACROSOMIA occurred in this group

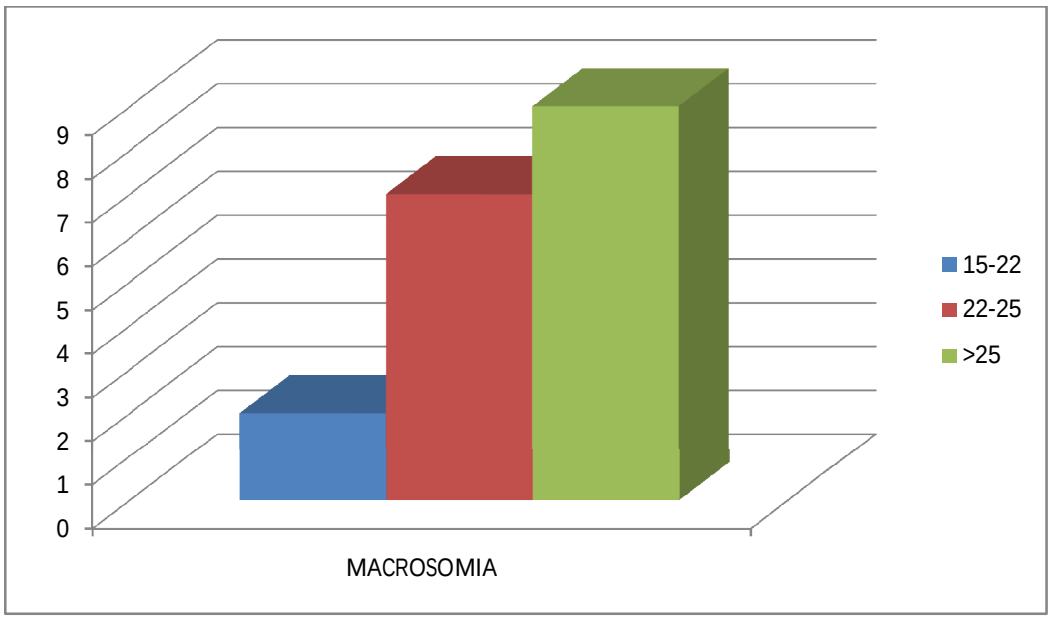
b) BMI 22-25–there were a total of 105 women in this group Out of them 7 women had MACROSOMIC baby 38.9% of the MACROSOMIA occurred in this group

c) BMI > 25– there were a total of 12 women in this group Out of them 9 women had MACROSOMIC baby 50% of the MACROSOMIA occurred in this group

PEARSON CHI-SQUARE = 80.199

P VALUE =0.000 ; P=0.000<0.0001

Significant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MACROSOMIA
15-22
22-25
>25

TYPE OF DIABETES * MACROSOMIA

TYPE OF DIABETES	MACROSOMIC BABIES	PERCENTAGE
GESTATIONAL DM	9	50%
DIABETES MELLITUS	9	50%

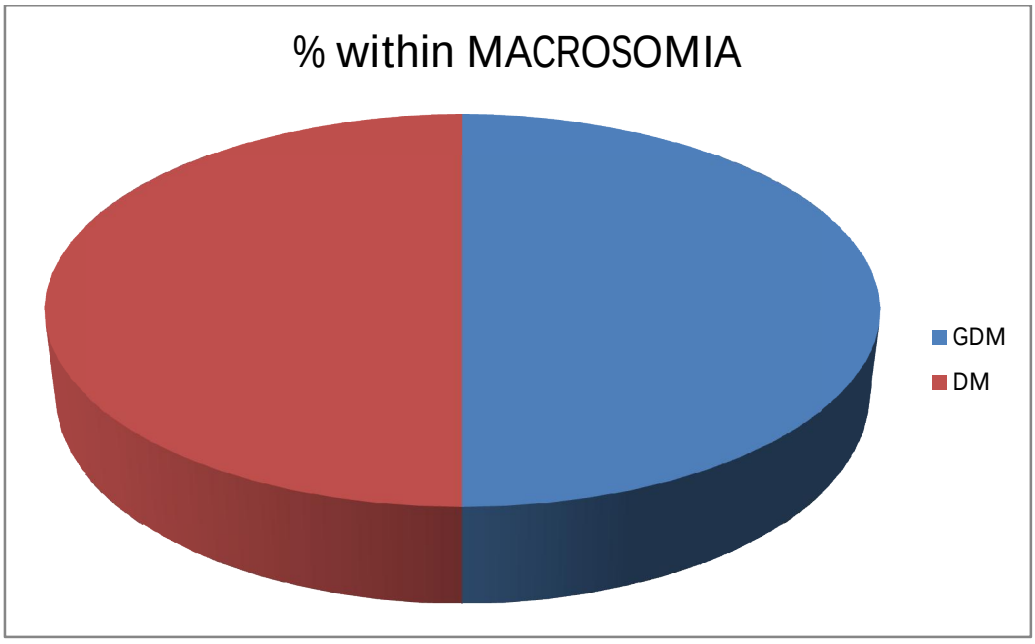


Figure 3: TYPE OF DIABETES

The women were divided into two groups according to type of diabetes.

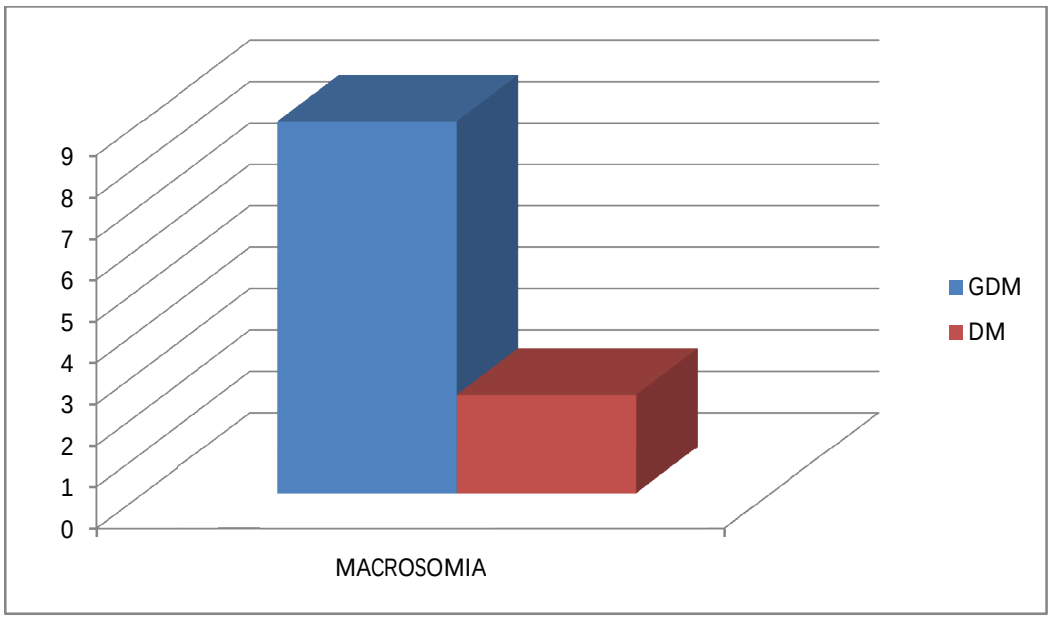
a) Gestational Diabetes Mellitus – there were a total of 99 women in this groupOut of them 9 women had MACROSOMIC baby 50% of the MACROSOMIA occurred in this group

b) Pre-Gestational Diabetes Mellitus – there were a total of 129 women in this group Out of them 9 women had MACROSOMIC baby50% of the MACROSOMIA occurred in this group

PEARSON CHI-SQUARE = .344

P VALUE =0.557

Not Significant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MACROSOMIA
GDM
DM

MODE OF TREATMENT * MACROSOMIA

MODE OF TREATMENT	MACROSOMIC BABIES	PERCENTAGE
MEAL PLAN	**7**	**38.9%**
INSULIN	**11**	**61.1%**

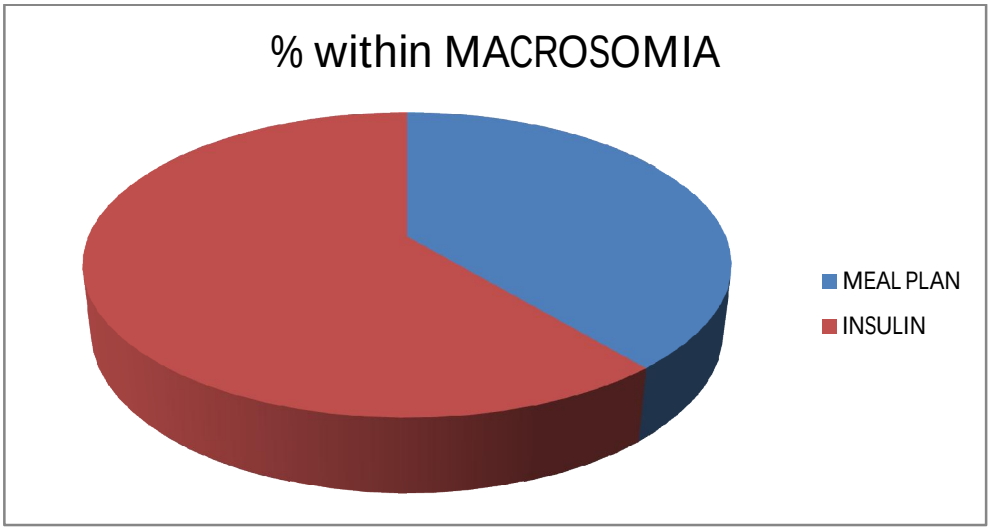


Figure 4: MODE OF TREATMENT

The women were divided into two groups according to treatment taken for diabetes.

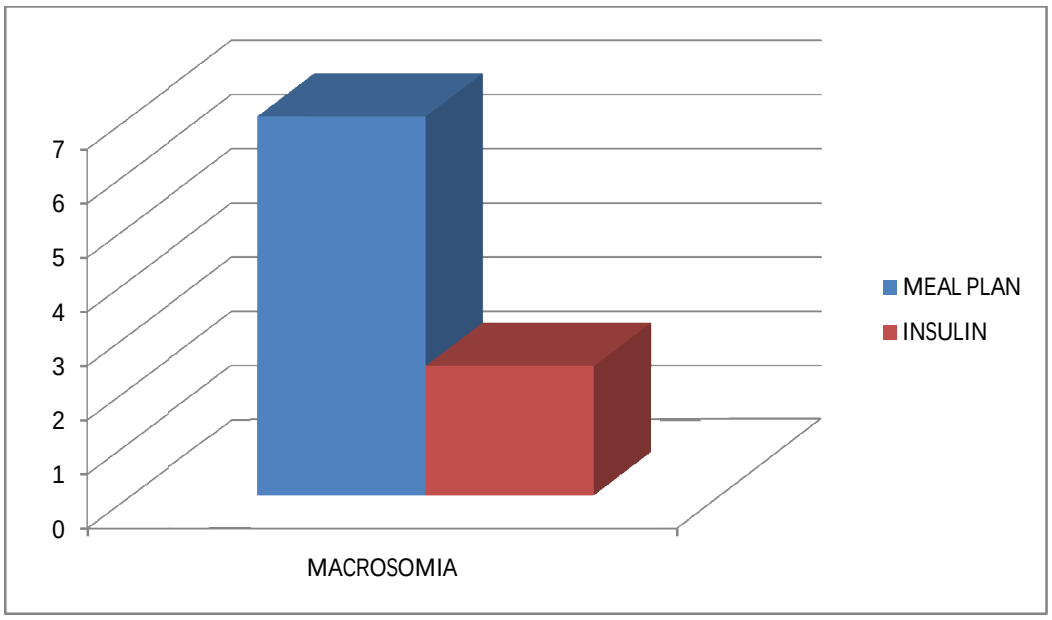
a) MEAL PLAN – there were a total of 81 women in this group Out of them 7 women had MACROSOMIC baby38.9% of the MACROSOMIA occurred in this group

b) INSULIN– there were a total of 147 women in this group Out of them 11 women had MACROSOMIC baby61.1% of the MACROSOMIA occurred in this group

PEARSON CHI-SQUARE = 0.096

P VALUE =0.756

Not Significant


7
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MACROSOMIA
MEAL PLAN
INSULIN

BIRTH ORDER * MACROSOMIA

BIRTH ORDER	MACROSOMIC BABIES	PERCENTAGE
1st BABY	7	38.9%
2ND BABY	7	38.9%
3RD BABY	3	16.7%
4TH BABY	1	5.6%

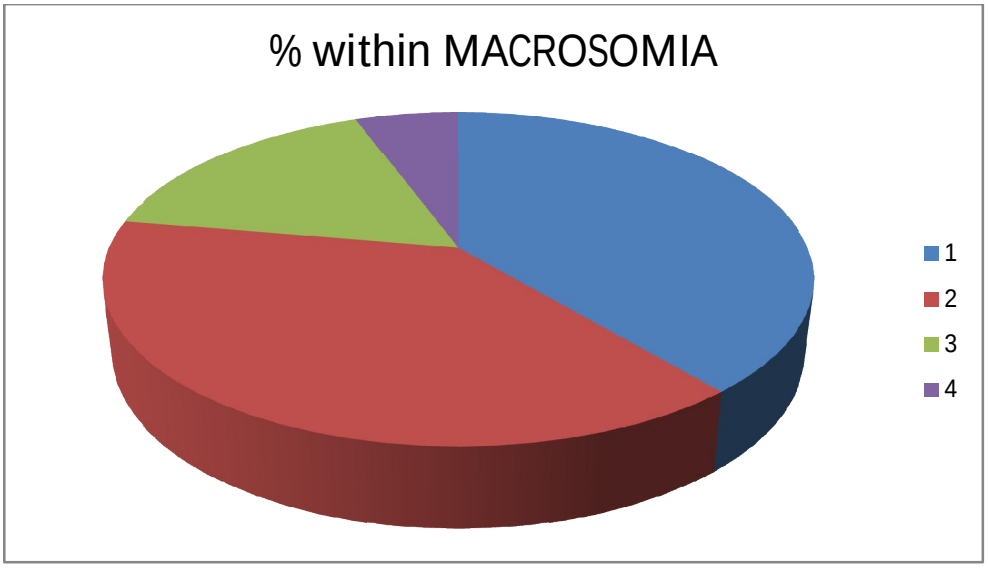


Figure 5: BIRTH ORDER OF THE MACROSOMIC BABY

The women were divided into four groups according to birth order of the baby.

a) 1st baby – there were a total of 53 women in this group Out of them 7 women had MACROSOMIC baby38.9% of the MACROSOMIA occurred in this group

b) 2nd baby – there were a total of 115 women in this group Out of them 7 women had MACROSOMIC baby38.9% of the MACROSOMIA occurred in this group

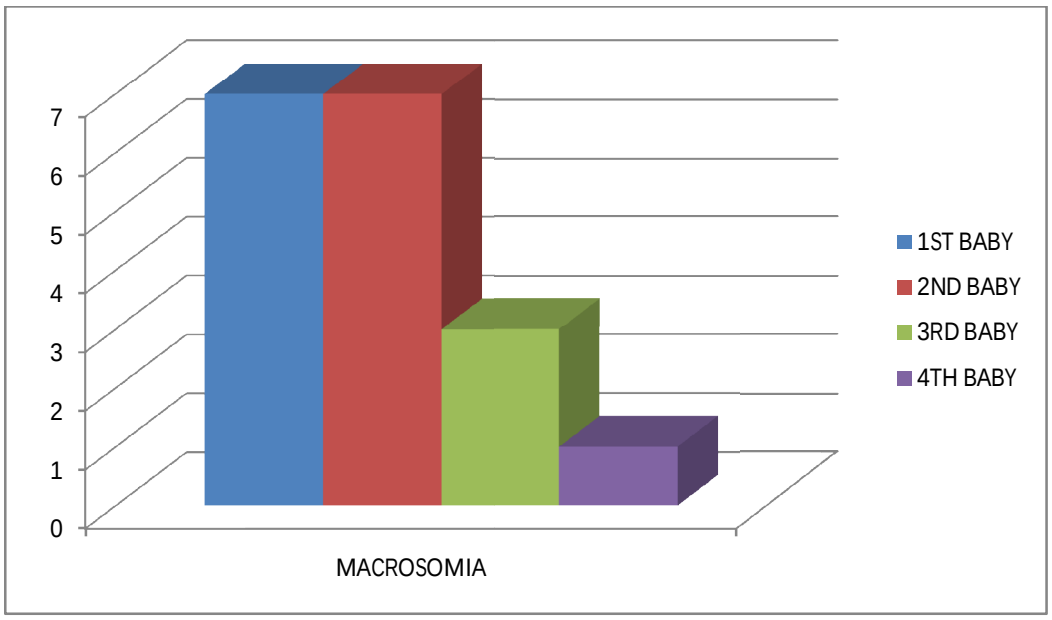
c) 3rd baby – there were a total of 54 women in this group Out of them 3 women had MACROSOMIC baby 16.7% of the MACROSOMIA occurred in this group

d) 4th baby -- there were a total of 4 women in this group Out of them 1 women had MACROSOMIC baby 5.6% of the MACROSOMIA occurred in this group

PEARSON CHI-SQUARE = 4.761

P VALUE =0.313

Not Significant


7
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MACROSOMIA
1ST BABY
2ND BABY
3RD BABY
4TH BABY

MODE OF DELIVERY * MACROSOMIA

MODE OF DELIVERY	MACROSOMIC BABIES	PERCENTAGE
LSCS	18	100%

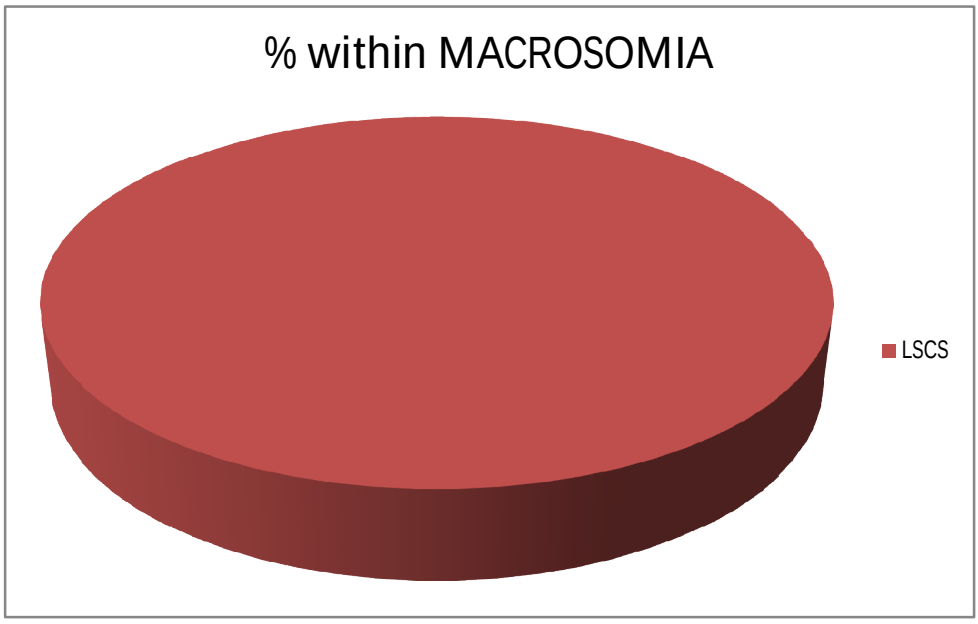


Figure 6: MODE OF DELIVERY OF THE MACROSOMIC BABY

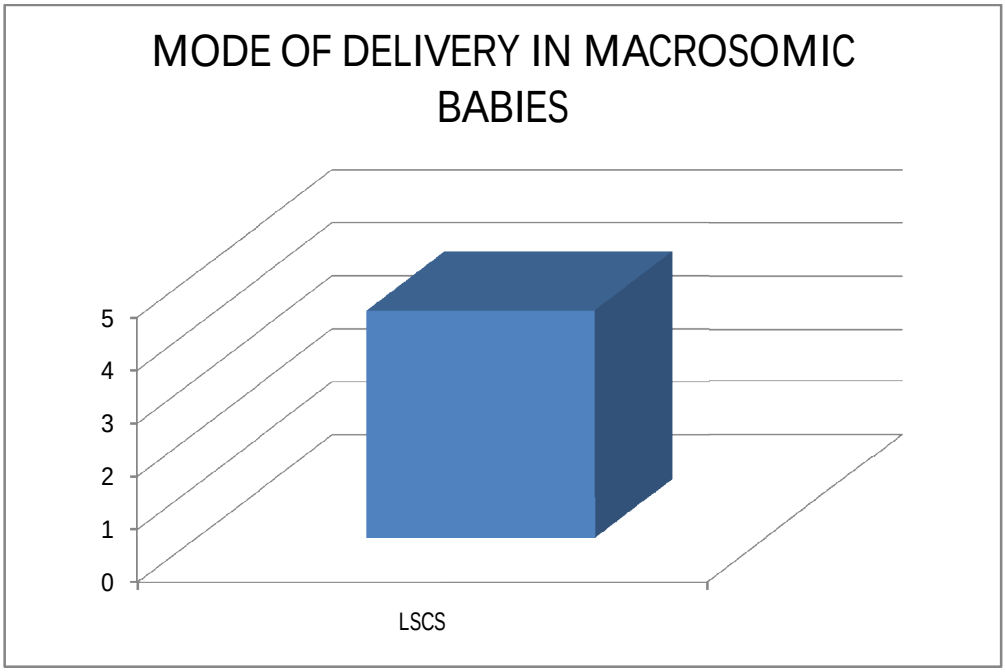
The women were divided into two groups according to Mode of Delivery.

All macrosomic babies were delivered by Caesarean Section.

PEARSON CHI-SQUARE = 6.514

P VALUE =0.011

Significant



INDEPENDENT SAMPLES T TEST

AGE

	MACROSOMIA	N	Mean	Std. Deviation	Std. Error Mean
AGE	YES	18	25.56	3.382	.797
	NO	210	25.54	2.967	.205

TABLE : 1

The total number of women who gave birth to MACROSOMIC babies was 18, and their mean age was 25.56 years(Standard Deviation= 3.382).(TABLE :1) By comparison the women who gave birth to non-macrosomic babies had a mean age of 25.54 years (Standard Deviation=2.967).(TABLE : 1)

		t- test for equality of Means		
		t	**df**	**P value**
AGE	Equal variances assumed	.024	226	.981
	Equal variances not assumed	.021	19.311	.983

TABLE : 2

In order to test the hypothesis, that there is no statistically significant difference between the age of women giving birth to macrosomic babies and the age of women giving birth to non-macrosomic babies, an independent samples t-test was done. The independent samples t – test showed that t value (with Degree of Freedom=226) = 0.024 and P value = 0.981, thus not significant. Thus there is no statistically significant difference between age of women who gave birth to macrosomic babies and age of women giving birth to non-macrosomic babies.(TABLE : 2)

BMI- BODY MASS INDEX

Group Statistics

	MACROSOMIA	N	Mean	Std. Deviation	Std. Error Mean
BMI	YES	18	24.72	2.469	.582
	NO	210	21.73	2.865	.198

TABLE : 3

The total number of women who gave birth to MACROSOMIC babies was 18, and their mean BMI was 24.72 (Standard Deviation= 2.461). By comparison the women who gave birth to non-macrosomic babies had a mean BMI of 21.73 (Standard Deviation=2.865) (TABLE:3).

		t-test for Equality of Means		
		t	df	P value
BMI	Equal variances assumed	4.296	226	.000
	Equal variances not assumed	4.871	21.129	.000

TABLE : 4

In order to test the hypothesis, that there is no statistically significant difference between the BMI of women giving birth to macrosomic babies and the BMI of women giving birth to non-macrosomic babies , an independent samples t-test was done. The independent samples t – test showed that t value (with Degree of Freedom=226) = 4.296 and P value = 0.000 , thus **significant.** Thus there is a statistically significant difference between BMI of women who gave birth to macrosomic babies and BMI of women giving birth to non-macrosomic babies.(TABLE : 4)

WEIGHT GAIN during pregnancy

Group Statistics

	MACROSOMIA	N	Mean	Std. Deviation	Std. Error Mean
WEIGHT GAIN	YES	18	13.72	.895	.211
	NO	210	11.73	1.224	.084

TABLE : 5

The total number of women who gave birth to MACROSOMIC babies was 18, and their mean weight gain was 13.72 (Standard Deviation= 0.895). By comparison the women who gave birth to non-macrosomic babies had a mean weight gain of 11.73 (Standard Deviation=1.224).(TABLE : 5)

		t-test for Equality of Means		
		t	df	P value
WEIGHT GAIN	Equal variances assumed	6.736	226	.000
	Equal variances not assumed	8.754	22.842	.000

TABLE : 6

In order to test the hypothesis , that there is no statistically significant difference between the weight gain of women giving birth to macrosomic babies and the weight gain of women giving birth to non-macrosomic babies, an independent samples t-test was done. test.The independent samples t – test showed that t value (with Degree of Freedom=226) = 6.736 and P value = 0.000 , thus **significant.** Thus there is a statistically significant difference between weight gain during pregnancy in women giving birth to macrosomic babies and weight gain during pregnancy in women giving birth to non-macrosomic babies.(TABLE :6)

DURATION of DIABETES MELLITUS

Group Statistics

	MACROSOMIA	N	Mean	Std. Deviation	Std. Error Mean
DURATION OF DM	YES	18	14.89	14.768	3.481
	NO	210	21.81	25.463	1.757

TABLE : 7

The total number of women who gave birth to MACROSOMIC babies was 18, and their mean duration of diabetes was 14.89months (Standard Deviation= 14.768). By comparison the women who gave birth to non-macrosomic babies had a mean duration of diabetes was 21.81 (Standard Deviation=25.463).(TABLE : 7)

		t-test for Equality of Means		
		t	df	P value
DURATION OF DM	Equal variances assumed	-1.136	226	.257
	Equal variances not assumed	-1.776	26.626	.087

TABLE : 8

In order to test the hypothesis , that there is no statistically significant difference between the duration of diabetes in women giving birth to macrosomic babies and the duration of diabetes in women giving birth to non-macrosomic babies , an independent samples t-test was done. The independent samples t – test showed that t value (with Degree of Freedom=226) = -1.136 and P value = 0.257 , thus not significant. Thus there is no statistically significant difference between duration of diabetes in women giving birth to macrosomic babies and duration of diabetes in women giving birth to non-macrosomic babies.(TABLE : 8)

HBA1C AT DELIVERY

Group Statistics

	MACROSOMIA	N	Mean	Std. Deviation	Std. Error Mean
HBA1C @ DELIVERY	YES	18	5.978	.2365	.0558
	NO	210	4.853	.4825	.0333

TABLE : 9

The total number of women who gave birth to MACROSOMIC babies was 18, and their mean HBA1C at delivery was 5.978 (Standard Deviation= 0.2365). By comparison the women who gave birth to non-macrosomic babies had a mean HBA1C at delivery = 4.853 (Standard Deviation=0.4825).(TABLE : 9)

		t-test for Equality of Means		
		t	df	P value
HBA1C @ DELIVERY	Equal variances assumed	9.772	226	.000
	Equal variances not assumed	17.316	30.971	.000

TABLE : 10

In order to test the hypothesis, that there is no statistically significant difference between the HBA1C at delivery of women giving birth to macrosomic babies and the HBA1C at delivery of women giving birth to non-macrosomic babies , an independent samples t-test was done.

The independent samples t – test showed that t value (with Degree of Freedom=226) = 9.772 and P value = 0.000 , thus**significant**. Thus there is a statistically significant difference between HBA1C at delivery of women who gave birth to macrosomic babies and HBA1C at delivery of women giving birth to non-macrosomic babies. Women giving birth to macrosomic babies have more HbA1c values above 5.6 than women giving birth to non-macrosomic babies.(TABLE : 10)

BIRTH ORDER

Group Statistics

	MACROSOMIA	N	Mean	Std. Deviation	Std. Error Mean
BIRTH ORDER	YES	18	1.89	.900	.212
	NO	210	2.08	.775	.053

TABLE : 11

The total number of women who gave birth to MACROSOMIC babies was 18, and their mean birth order was 1.89 (Standard Deviation= 0.900). By comparison the women who gave birth to non-macrosomic babies had a mean birth order of 2.08 (Standard Deviation=0.775). (TABLE : 11)

		t-test for Equality of Means		
		t	df	P value
BIRTH ORDER	Equal variances assumed	-.996	226	.320
	Equal variances not assumed	-.878	19.224	.391

TABLE : 12

In order to test the hypothesis , that there is no statistically significant difference between the birth order of women giving birth to macrosomic babies and the birth order of women giving birth to non-macrosomic babies, an independent samples t-test was done. The independent samples t – test showed that t value (with Degree of Freedom=226) = 0.996 and P value = 0.320 , thus not significant. Thus there is no statistically significant difference between birth order of women who gave birth to macrosomic babies and birth order of women giving birth to non-macrosomic babies.(TABLE : 12)

GESTATIONAL AGE

Group Statistics

	MACROSOMIA	N	Mean	Std. Deviation	Std. Error Mean
GESTATIONAL AGE	YES	18	37.67	1.029	.243
	NO	210	37.69	1.143	.079

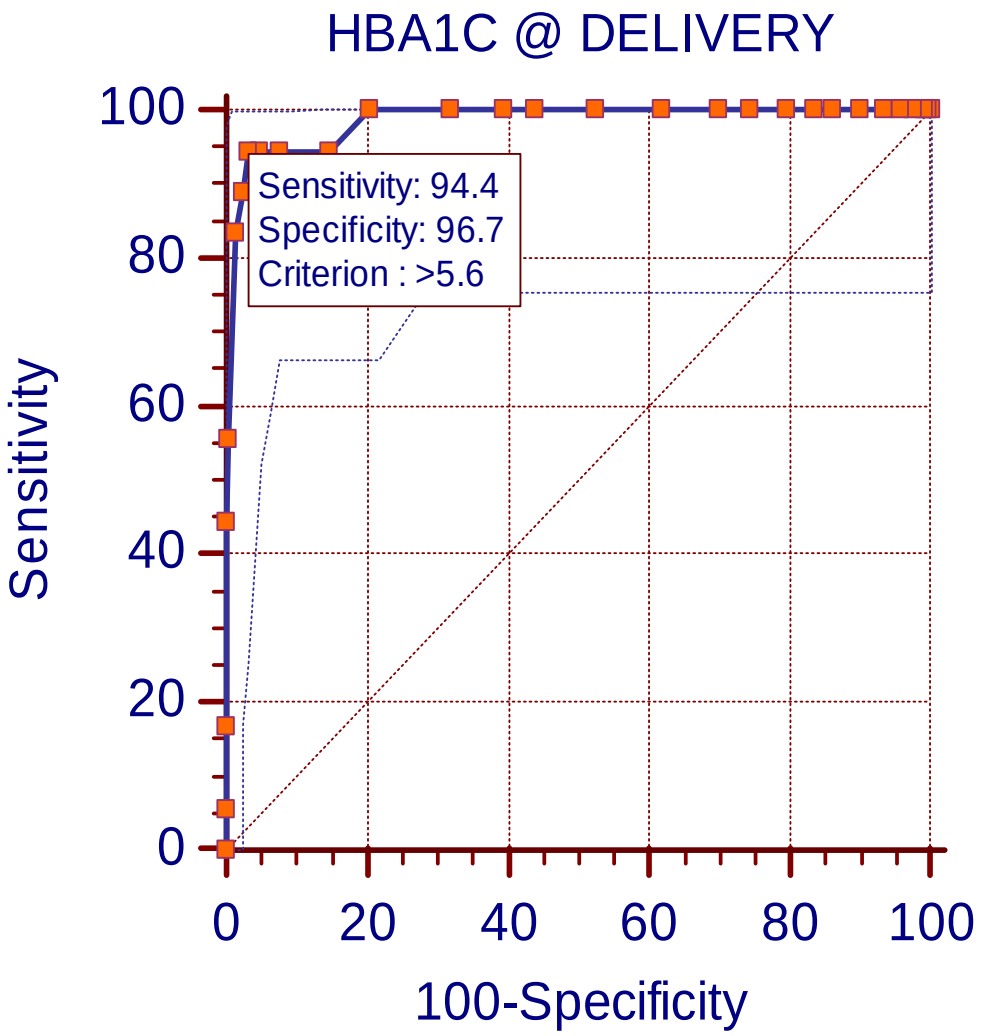
TABLE : 13

The total number of women who gave birth to MACROSOMIC babies was 18, and their mean gestational age was 37.67weeks (Standard Deviation= 1.029). By comparison the women who gave birth to non-macrosomic babies had a mean gestational age of 37.69 weeks (Standard Deviation=1.143(TABLE : 13)

		t-test for Equality of Means		
		t	df	P value
GESTATIONAL AGE	Equal variances assumed	-.068	226	.946
	Equal variances not assumed	-.075	20.770	.941

TABLE : 14

In order to test the hypothesis, that there is no statistically significant difference between the gestational age of women giving birth to macrosomic babies and the gestational age of women giving birth to non-macrosomic babies , an independent samples t-test was done. The independent samples t – test showed that t value (with Degree of Freedom=226) = -0.068 and P value = 0.946 , thus not significant. Thus there is no statistically significant difference between gestational age of women who gave birth to macrosomic babies and gestational age of women giving birth to non-macrosomic babies.(TABLE : 14)


HBA1C @ DELIVERY
Sensitivity: 94.4
Specificity: 96.7
Criterion : >5.6
Sensitivity
100-Specificity
0
20
40
60
80
100

ROC curve

Test Result Variable(s)	Area Under the Curve (AUC)	Std. Error	95% Confidence Interval of AUC		P-value
			Lower Bound	Upper Bound	
Hba1c @ delivery	0.985	0.010	0.958	0.996	<0.0001

As shown above, the ROC curve between the two variables HBA1C at delivery and Macrosomia is depicted. The area under the ROC curve = 0.984656 which signifies that it is an excellent test.

DISCUSSION

In this study , a total of 230 pregnant women with diabetes mellitus and their newborn babies were included. For 2 women some data was missing , hence remaining 228 pregnant diabetic women and their newborn completed the study. In this study, there is a **statistically significant** correlation between **glycosylated haemoglobin (HbA1c)** at delivery and occurrence of Macrosomia in infants of diabetic mothers. Pregnant diabetic women having a value of HbA1c > 5.6 are more likely to have a macrosomic baby. This has a sensitivity of 94.4 and specificity of 96.7. Glycosylated haemoglobin value measured at the time of delivery is a good indicator of delivering a macrosomic baby. HbA1c value at delivery reflects the 24 hour glucose profile[54] during the last 6-8 weeks[48]. As the major weight gain in the fetus occurs in the last trimester of pregnancy[9], it is the period during which high glucose level in the maternal blood favours fetal overgrowth. This is similar to the findings by **MR Mikkelsen et al**[55] in their study that median HbA1c value=5.9 before delivery , had a 3-fold increased risk of having large for gestational age babies.

Depending upon the **age** of the women , 55.6% of Macrosomic babies were born in the younger age group i.e. those below 25yrs of age. The mean age of women having macrosomic babies was 25.56 yrs (S.D.=3.382) although it was not significant. It is in contrast to the study published by N.E. Stotland et al[56] where they found that birth macrosomiawas statistically significantly seen in women of age group: 30yrs- 40yrs($p<0.001$).

50% of the macrosomic babies were born to women with a higher **Body Mass Index** i.e. above 25 and it was statistically **significant**. Thus we can say that Body Mass Index also contributes to having a macrosomic baby in a diabetic women. This is similar to the findings by **I.O. Frederick et al**[57] that body mass index was independently and positively associated with baby's birth weight ($p<0.001$). The mean of **Body Mass Index** of women having macrosomic babies was 24.72(S.D.=2.469) and it was statistically **significant.** This study showed that women with Body Mass Index > 25 were more likely to have a macrosomic baby.

There was no difference due to **type of diabetes mellitus**. Macrosomia occurred with almost equal frequency in women with gestational diabetes mellitus or pre-gestational diabetes mellitus i.e. overt diabetes.

Although 61.1% of the macrosomic babies were born in the group of women who were taking **insulin**, the results were not statistically significantly associated. It implies that women requiring insulin to maintain a stable glucose control have a more deranged carbohydrate metabolism in the body which ultimately leads to fetal overgrowth. Women on meal plan offer a better intra-uterine environment so as not to lead to fetal growth alterations. This is in confirmation of the study done by **L Suhonen et al**[58] in which they found that macrosomia occurred more often in women treated with insulin ($p<0.001$)

Mostly the first born or second born children seem to be macrosomic in our study.

All the macrosomic babies were delivered by **Caesarean Section.** Macrosomia is greater predictor of caesarean delivery[59]. Due to availability of better operating conditions and neonatal intensive care units these days, the obstetricians opt for caesarean section for the

delivery of macrosomic babies in order to have a healthy mother and a healthy baby.

Mean **weight gain** during pregnancy was 13.72 kg (S.D.=0.895). Weight gain during pregnancy is also showed asstatistically **significantly** related to occurrence of Macrosomic babies. Excessive weight gain during pregnancy independently influences the occurrence of macrosomia as shown in the study by **M..M. Heddersonet al**[60]. It should be kept in mind that the total weight gain during pregnancy by the women included the weight of the fetus and the placenta as well.

Women in this study had a mean **duration of diabetes mellitus** of 14.89 months. (S.D.=14.718) with a wide range and women with non-macrosomic babies have a numerically longer duration of diabetes (Mean=21.81 months with S.D.=25.463). This is similar to the findings in the study by **M.A.Berk et al**[61] in which duration of diabetes was inversely related to macrosomia. Possibly women having diabetes since long developed vascular disease which led to intra uterine growth restriction. May be women having diabetes since long were more cautious to take care during pregnancy to control their glucose level .

With respect to **gestational age** all babies were born around 37 weeks of gestation.

LIMITATIONS IN THE STUDY

* This study was done on a small number of diabetic pregnant women.
* Macrosomia was taken as a fixed value as birth weight> 4000grams.
* HBA1c reflects the glucose control of the past 6-8 weeks.

CONCLUSION

Overall, this study suggests that glycosylated haemoglobin (HbA1c) at delivery > 5.6 is statistically significantly related to the women giving birth to Macrosomic babies. Diabetic pregnant women who have a higher value of HbA1 care definitely more likely to have a macrosomic baby.

Thus this study recommends that in order to reduce the occurrence of macrosomia and its associated morbidities, HbA1c level at the time of delivery needs to be reduced.

In order to do so glucose control should be strictly monitored and should be within limits in the third trimester.

Other maternal characteristics related significantly to Macrosomia are BMI, weight gain during pregnancy and delivery by caesarean section. Macrosomia seem to occur more in women who were on treatment with insulin.

Rest of the parameters i.e. age of the mother, type of diabetes mellitus, duration of diabetes mellitus, birth order of the baby, gestational age does not seem to be associated with macrosomia.

However, it should be borne in mind that this study has its own limitations. As HbA1c reflects the blood glucose level of the previous days, nothing can be done for the damage already occurred. Measuring the blood glucose level at the time of delivery gives us no time for interventions to prevent the bad pregnancy outcome.

Hence, further research is needed to evaluate the accurate range of blood glucose levels in the mother to avoid bad prognosis. Also, the timing of the test should be such that we can intervene to alter the perinatal outcome in the infants of diabetic mother.

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ANNEXURES

ANNEXURE 1 : PROFORMA

ANNEXURE 2 : INFORMED CONSENT FORM

PROFORMA

MOTHER'S DETAILS:

Name

Age

BMI

Weight gain during pregnancy

Type of diabetes – pre gestational / gestational

Duration of GDM

Mode of treatment (meal plan/ insulin/ metformin)

HbA1c level at the time of delivery

BABY'S DETAILS:

Birth Order

Gestational age

Mode of delivery

Birth weight

INFORMED CONSENT FORM

STUDY: "CORRELATION OF HBA1C WITH MACROSOMIA

IN INFANTS OF DIABETIC MOTHER"

STUDY CENTRE: GOVT. KILPAUK MEDICAL COLLEGE HOSPITAL,CHENNAI

PATIENT'S NAME :

PATIENT'S AGE :

Patient may check () these boxes

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

I understand that my participation in the study is purely voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected. ☐

I understand that the Ethics Committee members and the regulatory authorities will need not my permission to look at my health records, both in respect of the current study and any further research that may be 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 the study.

I agree to take part in the above study and to comply with the instructions given during the study and faithfully co operate with the study team and to immediately inform 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.

Signature / thumb impression:

Patient’s name and address: Place: Date:

Signature of the investigator:

Study investigator’s name: Place: Date:

சுய ஒப்புதல் படிவம்

இடம் : அரசு கீழ்பாக்கம் மருத்துவக் கல்லூரி, சென்னை

எனக்கு சர்க்கரை நோய் உள்ளது. ஆகவே எனக்கும் எனது குழந்தைக்கும் ரத்தத்தில் ஏதேனும் வேறு பிரச்சனை உள்ளதா என்பதை ஆய்வு செய்து தெரிந்துக்கொள்ள எனக்கு சம்மதம் . இந்த இரத்த பரிசோதனைக்கு நான் முழுமனதோடு சம்மதிக்கிறேன்.

பங்கேற்பவரின் கையொப்பம்

MASTER CHART

Name	AGE	BMI	WEIGHT GAIN	TYPE OF DIABETES	DURATION OF DM	MODE OF TREATMENT	HBA1C @ DELIVERY	BIRTH ORDER	GESTATIONAL AGE	MODE OF DELIVERY	BIRTH WEIGHT
	yrs	NORMAL: 15-22		PGDM / GDM	YRS/MONTHS	MEAL PLAN/ INSULIN			WKS	NVD / LSCS	kg
		OVERWT.:22-25									
		OBESE: >25									
PRIYANKA	20	19	11	GDM	4 mon	meal plan	4	2	38	NVD	2.7
KALPANA	23	23	12	PGDM	2yrs	insulin	5.9	1	39	LSCS	4
MARY MURUGAN	22	26	14	PGDM	1.5yrs	insulin	6.2	1	38	LSCS	4.4
SURYA	21	25	13	GDM	3 mon	meal plan	4.9	2	38	NVD	3.1
BHAVANI	24	23	12	PGDM	1yr	insulin	4.8	1	40	NVD	2.8
PRIYA NICHOLAS	22	18	10	PGDM	12yrs	insulin	4.5	2	39	NVD	2.4
CHITRA	26	22	12	GDM	4 mon	meal plan	5.2	3	36	LSCS	3.2
POORNADHARSHINI	23	18	11	PGDM	5yrs	insulin	3.9	2	37	LSCS	2.9
MANJULA	24	15	9	GDM	3mon	meal plan	4	1	38	NVD	2.9
NANDHINI	21	22	13	PGDM	1yr	insulin	5	1	39	LSCS	3.2
DEEPA	25	25	13	PGDM	3yrs	insulin	6.1	2	38	LSCS	4.1
ALAMELU	27	20	11	PGDM	1yr	insulin	4.2	2	39	LSCS	2.9
IMALAR VENUGOPAL	30	22	12	PGDM	5yrs	insulin	5.6	1	40	LSCS	3.4
SATHIYA PRAKASH	32	26	14	GDM	4 mon	meal plan	5.9	1	38	LSCS	4.2
VIJAYALAKSHMI	22	25	14	PGDM	4yrs	insulin	5.3	2	38	NVD	3.5
CHINNAPONNU	26	24	11	PGDM	5yrs	insulin	5.1	3	37	LSCS	3.1
VANITHA	28	21	13	PGDM	3yrs	insulin	4.1	1	39	NVD	2.8

KAVITHA	29	15	10	GDM	2 mon	meal plan	4	2	38	NVD	2.5
TAMILSELVI	24	18	10	PGDM	2yrs	insulin	4.3	2	38	NVD	2.6
AMUDHA	23	26	15	PGDM	1yr	insulin	5.2	1	38	LSCS	3.1
DIVYA	21	22	11	GDM	4 mon	meal plan	5.1	1	38	NVD	3.2
SARASWATHY	20	28	15	PGDM	2yrs	insulin	6	1	39	LSCS	3.7
SATHYA SATHISH KUMAR	27	22	13	GDM	3mon	meal plan	4.7	2	38	NVD	2.9
JAITHUMNISHA	25	21	12	PGDM	2yrs	insulin	5.2	1	35	LSCS	3
DHIVYA BHAGIRAJ	28	25	13	GDM	4mon	meal plan	5.6	2	38	LSCS	3.8
STELLA MARY	24	24	12	PGDM	3yrs	insulin	5	3	39	NVD	2.8
MOHANA KUPPAN	24	22	12	PGDM	2yrs	insulin	5.2	2	38	LSCS	3.4
ARUL SELVI	22	27	14	PGDM	1yr	insulin	6.1	1	39	LSCS	4.4
AMUTHA	26	24	12	GDM	5mon	meal plan	5.1	1	36	NVD	3.2
THENMOZHI PRAKASH	27	25	13	GDM	4mon	meal plan	5.2	2	36	LSCS	3.5
JALAJA	28	23	13	PGDM	5yrs	insulin	5	1	36	LSCS	3.2
JACKLINE	26	22	12	PGDM	3yrs	insulin	4.6	2	38	NVD	2.9
DEEPA BHARATH	25	24	12	PGDM	2yrs	insulin	4.9	2	39	LSCS	3
UDAYARANI	27	25	13	PGDM	4yrs	insulin	5.4	2	38	NVD	3
MEENA POONGAVANAM	28	19	10	GDM	4mon	insulin	4.1	1	37	NVD	2.7
VALLARMATHI	21	18	10	GDM	2mon	meal plan	5.2	2	36	LSCS	3.5
NITHYA	22	19	9	GDM	3mon	insulin	4.8	1	39	NVD	2.8
MEENAKSHI A	22	16	8	GDM	2mon	meal plan	3.6	1	38	NVD	2.4
NIRMALATHA	28	19	10	GDM	1mon	meal plan	4	2	39	LSCS	3.4
GEETHA	26	20	10	PGDM	4yrs	insulin	4.7	1	37	LSCS	3.5
MARY PRINCY	33	22	12	PGDM	5yrs	insulin	4.9	3	36	NVD	3.2
PREETHA	19	24	12	PGDM	1yr	insulin	5.2	2	37	LSCS	3.3

VIJI	30	23	13	GDM	2mon	meal plan	5.4	1	37	LSCS	3.6
SUJATHA	33	21	12	GDM	1mon	meal plan	4.4	3	36	NVD	2.8
SAMUNDEESWARI	26	28	14	PGDM	4yrs	insulin	6.1	2	35	LSCS	4.3
PRIYA B	27	22	13	PGDM	3yrs	insulin	5.5	3	39	LSCS	3.7
PRIYA A	35	18	10	GDM	3mon	meal plan	4	2	36	NVD	2.5
DEVAPRIYA	36	24	14	PGDM	9yrs	insulin	5.5	3	39	LSCS	3.5
MALATHY	33	22	13	PGDM	5yrs	insulin	4.9	2	37	LSCS	2.8
NALINI	25	16	9	GDM	4mon	meal plan	3.9	2	38	NVD	2.7
EPSIBA	34	25	14	GDM	3mon	meal plan	6	3	37	LSCS	4.2
VIJAYAKUMARI	21	21	11	PGDM	1yr	insulin	5.3	2	39	NVD	3.8
PARIMALA	32	22	13	PGDM	6yrs	insulin	5.2	5	37	NVD	3.2
SUMATHI	23	22	12	PGDM	1yr	insulin	4.7	2	38	LSCS	3
DEEPA	21	21	11	PGDM	1yr	insulin	5	3	39	LSCS	3.1
NANDHINI	25	19	10	GDM	3mon	meal plan	4.8	3	37	LSCS	3.5
BUVANESWARI	26	17	10	GDM	3mon	meal plan	4.9	2	36	LSCS	3.6
SABINA BEGUM	23	19	11	GDM	4mon	meal plan	5.4	1	38	LSCS	3.9
SUMATHI B	27	21	11	GDM	2mon	insulin	5.5	2	39	LSCS	3.8
SHARMILA	22	18	10	PGDM	8yrs	insulin	5.4	3	40	LSCS	3.2
LOURDE MARY	27	17	10	GDM	2mon	insulin	4.4	3	38	LSCS	2.9
ANANDI	29	25	12	PGDM	4yrs	insulin	5.3	2	39	LSCS	3.7
ROSY P	23	26	15	PGDM	2yrs	insulin	6.4	2	39	LSCS	4.2
SARITHA	25	16	12	GDM	3mon	insulin	4.5	3	37	NVD	2.7
POORAVI	21	25	13	PGDM	1yr	insulin	5.3	1	39	LSCS	3.4
DEEPA BALA	28	14	10	GDM	2mon	meal plan	5.1	3	37	NVD	2.4
NISHI	29	23	12	PGDM	3yrs	insulin	4.7	3	38	LSCS	3.1

DIVYA	25	19	11	PGDM	7yrs	insulin	4.9	2	38	LSCS	3.2
DHANALAKSHMI	23	17	11	GDM	4mon	meal plan	3.7	2	38	NVD	2.7
RATHI	28	19	11	GDM	4mon	meal plan	4.1	2	37	LSCS	2.8
NOORNISHA	24	23	11	PGDM	2yrs	insulin	5	3	35	NVD	2.5
KALAUNDI	23	22	12	PGDM	2yrs	insulin	5.2	2	39	LSCS	3.7
SANGEETHA	26	24	13	GDM	3mon	meal plan	5.9	4	38	LSCS	4.1
LOGESWARI	27	21	12	PGDM	2yrs	insulin	5.4	2	39	LSCS	3.8
KAVITHA K	25	24	13	PGDM	1yr	insulin	5.3	1	38	LSCS	3.8
KAVITHA D	23	19	10	GDM	3mon	insulin	4.6	2	39	NVD	2.9
RADHA	25	16	9	PGDM	12yrs	insulin	4.2	2	38	LSCS	3.2
NIVEDHITA	25	22	12	GDM	4mon	insulin	5.1	2	37	LSCS	3.6
MEENAKUMARI	26	24	12	PGDM	5yrs	insulin	5.1	4	38	NVD	2.8
SANDHYA	23	17	10	GDM	2mon	meal plan	4.8	3	38	NVD	2.9
MALATHI	28	24	11	PGDM	4yrs	insulin	5	2	39	LSCS	3
VIJAYA	21	23	13	PGDM	1yr	insulin	4.8	2	38	NVD	2.8
KALAIYARASI B	28	24	13	PGDM	3yrs	insulin	5.9	3	37	LSCS	4
GEETHA	28	25	11	GDM	3mon	meal plan	4.9	2	36	NVD	2.7
MALAR	29	24	12	GDM	2mon	meal plan	5.1	2	38	LSCS	3.2
DEEPA SRINIVASAN	24	22	13	PGDM	2yrs	insulin	5.4	3	39	LSCS	3.3
KUMARI MURUGAN	25	24	12	PGDM	1yr	insulin	5.2	2	36	LSCS	3.1
HAJEERA	21	22	11	GDM	3mon	meal plan	5.1	3	38	LSCS	3.3
SANTHANALAKSHMI	27	25	13	PGDM	3yrs	insulin	5.3	4	37	LSCS	3.7
RAGINA MARY	28	23	11	PGDM	6yrs	insulin	4.5	5	36	NVD	2.9
SHARMILA	24	22	12	PGDM	2yrs	insulin	5.1	2	39	LSCS	3.4
SAMUNDESWARI	29	23	11	PGDM	4yrs	insulin	4.7	3	38	NVD	2.8

SASIKALA NAGARAJ	30	24	13	GDM	3mon	meal plan	5.4	3	39	LSCS	3.4
NANCY	24	25	12	GDM	2mon	meal plan	5.9	2	37	LSCS	4.2
RADHIKA	28	24	12	GDM	3mon	meal plan	5.1	3	35	LSCS	3.2
ROSLIN	24	25	13	GDM	3mon	meal plan	5.4	2	34	LSCS	3.6
RABIYA UMAR FAROOK	29	22	11	PGDM	3yrs	insulin	4.6	3	37	NVD	2.8
SANGEETHA	22	23	12	GDM	4mon	meal plan	4.7	2	39	LSCS	3.3
NADHIYA	25	24	15	PGDM	2yrs	insulin	5.2	1	37	LSCS	3.8
VIJAYALAKSHMI	23	23	11	GDM	2mon	meal plan	5.3	2	38	LSCS	3.9
KALAISELVI	28	22	11	GDM	3mon	meal plan	5.1	3	39	LSCS	3.7
MOHANALAKSHMI	25	23	12	PGDM	2yrs	insulin	5.2	2	37	LSCS	3.7
JEYABHARATHI	29	24	12	PGDM	5yrs	insulin	4.8	4	38	LSCS	3.2
PARVATHY	24	23	14	GDM	2mon	meal plan	5.3	1	38	LSCS	4
DEEPA	28	25	12	PGDM	4yrs	insulin	4.7	3	36	LSCS	3.1
ELLAMAL	26	24	11	PGDM	2yrs	insulin	4.9	3	39	LSCS	3.2
VIGNESWARI	29	22	13	PGDM	3yrs	insulin	4.7	3	38	LSCS	3.2
PHILOMEENA	22	23	11	GDM	1mon	meal plan	5	2	38	LSCS	3.7
GAYATHRI	29	24	10	PGDM	4yrs	insulin	4.8	2	39	LSCS	3.4
PRABHAVATI	26	23	13	PGDM	3yrs	insulin	5.1	2	39	LSCS	3.8
SARITHA	24	22	12	PGDM	1yr	insulin	5.4	2	40	LSCS	3.9
ANJALI M	26	24	13	GDM	2mon	meal plan	4.9	3	38	NVD	2.9
SELVI P	21	17	11	GDM	4mon	insulin	4.6	1	37	NVD	2.7
RAMANI	26	19	14	GDM	3mon	insulin	5.7	2	39	LSCS	4.2
JAYANTHI	31	24	13	PGDM	6yrs	insulin	5.2	2	37	LSCS	3.6
ANJALAI	26	25	10	PGDM	2yrs	insulin	4.7	2	38	NVD	2.8
MAHALAKSHMI	27	22	12	PGDM	4yrs	insulin	4.9	2	39	LSCS	3.4

MEENA K	28	24	11	GDM	4mon	insulin	4.4	3	36	NVD	3
BADRUNISHA	29	21	11	GDM	4mon	insulin	4.7	2	39	LSCS	3.2
KALAIVANI	22	22	13	PGDM	1yr	insulin	5.4	1	38	LSCS	3.9
DEVI PASUPATHY	24	27	15	GDM	2mon	meal plan	6.1	2	37	LSCS	4.3
VANITHA	23	23	12	PGDM	2yrs	insulin	5.3	1	39	LSCS	3.7
REVATHI	29	23	11	PGDM	5yrs	insulin	5.2	3	37	LSCS	3.8
GRACY DAVID	27	18	12	GDM	1mon	meal plan	4.8	2	36	NVD	2.8
MALATHI	25	22	14	GDM	3mon	meal plan	5	1	38	LSCS	3.2
KANNIMOZHI	26	23	11	PGDM	2yrs	insulin	4.1	2	39	LSCS	2.5
SAVITHRI S	24	19	11	PGDM	5yrs	insulin	4.2	2	38	LSCS	2.6
GAYATHRI RAJASEKHAR	23	24	12	GDM	2mon	insulin	4.1	2	37	NVD	2.6
KANIYAMMAL	27	16	12	GDM	2mon	meal plan	5.2	2	38	LSCS	3.2
SUGANTHI	28	17	13	PGDM	10yrs	insulin	5.5	2	39	LSCS	3.7
PRABHA	24	15	11	GDM	3mon	meal plan	5.2	1	37	LSCS	3.4
PARVATHY PRAKASH	25	19	12	PGDM	6yrs	insulin	4.4	2	38	LSCS	3.1
JHANSI KAMARAJ	22	25	11	PGDM	1yr	insulin	4.3	1	37	NVD	2.9
ANUPAMA	25	26	14	PGDM	2yrs	insulin	5.8	1	37	LSCS	4.3
BHUVANESWARI MANIKANDAN	24	23	12	GDM	1mon	meal plan	5.2	2	37	LSCS	3.5
PREETHIKA	27	17	14	GDM	4mon	insulin	5.9	3	38	LSCS	3.8
VINSIKA	28	19	13	GDM	4mon	insulin	4.8	3	37	LSCS	3.2
KARPAGALLI	25	24	11	PGDM	1yr	insulin	4.4	2	36	LSCS	3.1
HARINI	23	22	12	PGDM	1yr	insulin	4.5	1	38	LSCS	3.2
MENAGA	24	19	11	GDM	2mon	meal plan	4.9	1	38	LSCS	3.4
NIRMALA	27	23	13	PGDM	4yrs	insulin	5.4	3	38	LSCS	3.9
ANGELINE	29	21	12	GDM	2mon	meal plan	5.2	3	39	LSCS	3.7

MARY JACOB	27	22	10	PGDM	3yrs	insulin	4.6	2	37	NVD	2.9
MARIAMMAL	23	21	13	GDM	1mon	meal plan	4.8	2	39	LSCS	3
MUNNESWARI	21	25	13	PGDM	1yr	insulin	4.7	1	37	NVD	3
POONGAVALI	25	24	12	PGDM	2yrs	insulin	4.6	2	38	LSCS	3
ASHWITHA	31	23	11	PGDM	7yrs	insulin	4.2	3	37	NVD	2.9
ANGEL G	27	24	12	PGDM	3yrs	insulin	4.1	2	38	LSCS	2.7
KAVISHRI	19	25	11	GDM	2mon	meal plan	4.5	1	39	LSCS	3.2
DHARSHINI	22	22	11	PGDM	2yrs	insulin	4.2	2	37	NVD	2.9
DHANALAKSHMI	27	25	11	PGDM	1yr	insulin	4.6	2	39	LSCS	3.3
PRIYA	26	17	12	GDM	4mon	meal plan	3.9	1	37	NVD	2.6
KEERTHIGA	28	18	10	GDM	1mon	meal plan	4.3	3	38	LSCS	3.1
PRIYADHARSHINI	27	21	13	PGDM	4yrs	insulin	5	3	37	LSCS	3.6
BHARANI	25	22	12	GDM	3mon	meal plan	5.3	2	38	LSCS	3.8
CHITHRA K	23	22	12	PGDM	1yr	insulin	4.8	2	37	LSCS	3.4
DHARANI	25	23	14	PGDM	1yr	insulin	5.6	2	39	LSCS	3.9
HEMALATHA	22	19	11	GDM	3mon	meal plan	4.5	2	36	LSCS	3.1
VINOTHINI	21	19	15	GDM	1mon	meal plan	6	1	38	LSCS	4.1
JEBAMALAR	23	23	10	GDM	2mon	meal plan	4.7	2	39	LSCS	3.4
MEENAKSHI A	24	22	11	GDM	2mon	meal plan	4.5	2	37	LSCS	3
RAMYA	25	21	11	PGDM	2yrs	insulin	4.7	2	39	LSCS	3.4
BUSHRA BEGUM	22	16	11	GDM	2mon	meal plan	5.8	2	37	LSCS	3.8
ILLAKKIYA	21	15	12	PGDM	5yrs	insulin	5.2	1	37	LSCS	3.6
SHWETHA	22	25	13	PGDM	1yr	insulin	5.3	2	36	LSCS	3.6
SRUTHI	26	19	11	GDM	1mon	meal plan	4.4	2	37	LSCS	3.1
OVIYA B	25	25	12	PGDM	3yrs	insulin	4.8	3	38	LSCS	3.3

AARTHI	22	18	11	GDM	2mon	meal plan	4.9	2	36	LSCS	3.4
MALLISWARAN	26	27	10	PGDM	2yrs	insulin	5.9	2	37	LSCS	3.8
THANISHA	28	22	13	GDM	2mon	meal plan	5.4	3	35	LSCS	3.6
NIVETHA	25	24	12	GDM	2mon	meal plan	4.8	1	37	LSCS	3.1
DIVYADHARSHINI	28	21	12	GDM	4mon	insulin	4.7	1	38	NVD	3
SOWMIYA	22	23	10	PGDM	1yr	insulin	4.3	2	38	LSCS	2.8
JANANI	29	21	12	GDM	1mon	meal plan	4.2	2	38	NVD	2.9
VARALAKSHMI	24	20	13	PGDM	3yrs	insulin	4.8	2	37	LSCS	3.2
MEGHALA T	23	25	11	PGDM	3yrs	insulin	3.8	1	38	NVD	2.7
VARSHA	27	17	13	GDM	2mon	meal plan	5.1	2	38	LSCS	3.6
JAISREE	25	20	13	PGDM	3yrs	insulin	5.5	2	38	LSCS	3.9
KEERTHANA	28	19	12	GDM	1mon	meal plan	4.9	3	38	LSCS	3.4
FIZA PARVEEN	22	22	12	PGDM	2yrs	insulin	4.8	2	36	LSCS	3.2
VIDHYA SREE	29	19	11	GDM	1mon	meal plan	4.6	3	37	LSCS	3.2
ABINAYA	26	16	13	GDM	2mon	meal plan	5.3	1	39	LSCS	3.7
THANUSHIYA	25	15	11	GDM	3mon	meal plan	5.2	3	37	LSCS	3.7
SANTHANA LAKSHMI B	24	24	14	GDM	4mon	meal plan	5.8	2	37	LSCS	3.8
YUVASHREE	27	25	12	PGDM	1yr	insulin	4.7	3	38	LSCS	3.2
PAVITHRA	26	21	14	GDM	2mon	meal plan	5.7	3	39	LSCS	3.8
JEROSHA	26	24	10	PGDM	2yrs	insulin	4.4	3	39	LSCS	2.9
DHANSHIKA SREE	27	25	12	PGDM	3yrs	insulin	4.5	2	39	LSCS	3
SUMATHI	24	17	12	GDM	2mon	meal plan	4.8	1	39	LSCS	3.2
MALARVIZHI	23	23	12	PGDM	1yr	insulin	3.8	2	37	NVD	2.8
JENNIFER	28	19	11	GDM	1mon	meal plan	5.4	2	36	LSCS	3.8
PRASANNA	28	18	11	PGDM	1yr	insulin	5.1	1	37	LSCS	3.5

RANJANI	29	25	12	PGDM	3yrs	insulin	4.7	2	37	LSCS	3.3
DEEPIKA SREE	24	19	11	GDM	2mon	meal plan	5.2	3	39	LSCS	3.6
ALAMELU	27	26	14	PGDM	2yrs	insulin	6.2	3	37	LSCS	4.2
DEVADHARSHINI	22	22	13	PGDM	1yr	insulin	5.4	2	37	LSCS	3.8
SATHYA MARY	24	25	11	PGDM	2yrs	insulin	4.6	2	38	LSCS	3
SUDHARSHINI	27	23	12	PGDM	5yrs	insulin	4.9	2	38	LSCS	3.4
YAMUNA	23	24	10	PGDM	1yr	insulin	4.2	1	37	LSCS	3.1
ALMA	28	19	11	GDM	2mon	meal plan	4.3	2	39	LSCS	3.1
PARVEEN BANO	28	25	14	GDM	3mon	meal plan	5.4	2	37	LSCS	3.7
POOVARASAI	26	23	12	PGDM	2yrs	insulin	4.9	2	37	LSCS	3.4
SURIYA	27	24	11	PGDM	2yrs	insulin	4.8	3	37	LSCS	3.3
KIRUTHIKA	25	25	13	PGDM	3yrs	insulin	5.1	2	38	LSCS	3.5
NITHYA	27	17	13	GDM	3mon	meal plan	5.2	2	38	LSCS	3.5
DIVYA MALINI	23	24	12	PGDM	2yrs	insulin	4.9	1	37	LSCS	3.3
YASHIKA	24	24	10	PGDM	3yrs	insulin	4.5	1	38	NVD	3.1
ABIRAMI	23	25	10	GDM	2mon	insulin	4.2	2	37	NVD	3
RAMYA S	23	23	14	PGDM	1yr	insulin	5.7	1	39	LSCS	3.9
SAIRA PARVEEN	21	25	12	GDM	3mon	meal plan	5.2	1	36	LSCS	3.8
AGHALYA	23	24	11	PGDM	2yrs	insulin	4.3	1	39	NVD	3
ZEBA KHATOON	26	23	13	PGDM	3yrs	insulin	5.1	2	36	LSCS	3.6
HASINI	27	18	11	GDM	3mon	meal plan	3.8	3	35	NVD	2.9
INDUMATHI	28	25	11	PGDM	3yrs	insulin	4.5	3	36	NVD	3
HAJIRA SURFURI	29	23	13	PGDM	4yrs	insulin	4.9	3	38	LSCS	3.3
LAKSHITHA	23	24	12	PGDM	1yr	insulin	4.8	2	38	LSCS	3.3
LAKSHMI P	24	23	12	PGDM	3yrs	insulin	4.4	1	38	LSCS	3.2

ROOBIKA M	27	25	10	GDM	4mon	insulin	4.1	2	39	LSCS	2.9
SUMAIYA PARVEEN	30	24	11	PGDM	5yrs	insulin	3.9	3	37	LSCS	2.8
DHARINI	31	19	12	GDM	2mon	meal plan	4.8	2	38	LSCS	3.5
KAYALVIZHI	24	24	13	PGDM	2yrs	insulin	4.9	2	39	LSCS	3.5
MOSHITA D	28	20	11	PGDM	4yrs	insulin	4.7	2	36	LSCS	3.2
LOSHINI	24	23	12	PGDM	1yr	insulin	4.5	1	39	LSCS	3.1
JEEVADHARSHINI	26	24	12	GDM	2mon	meal plan	5.2	2	37	LSCS	3.7
SHANKARI M	24	25	14	PGDM	2y	insulin	5.5	2	35	LSCS	3.7
YASIN	28	26	13	GDM	3mon	insulin	6.1	2	37	LSCS	4.2
KAVIYA P	25	24	11	PGDM	3yrs	insulin	4.8	2	39	LSCS	3.2
MUNDESWARI	29	25	13	GDM	2mon	meal plan	5.3	3	37	LSCS	3.5