OUTCOME OF NEONATES BORN TO MOTHERS WITH DIABETES MELLITUS IN A TERTIARY CARE CENTRE IN KOLAR-A PROSPECTIVE COHORT STUDY

By

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DISSERTATION SUBMITTED TO
SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR, KARNATAKA
In partial fulfillment of the requirement or the degree of

DOCTOR OF MEDICINE
IN
PAEDIATRICS

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JULY 2024

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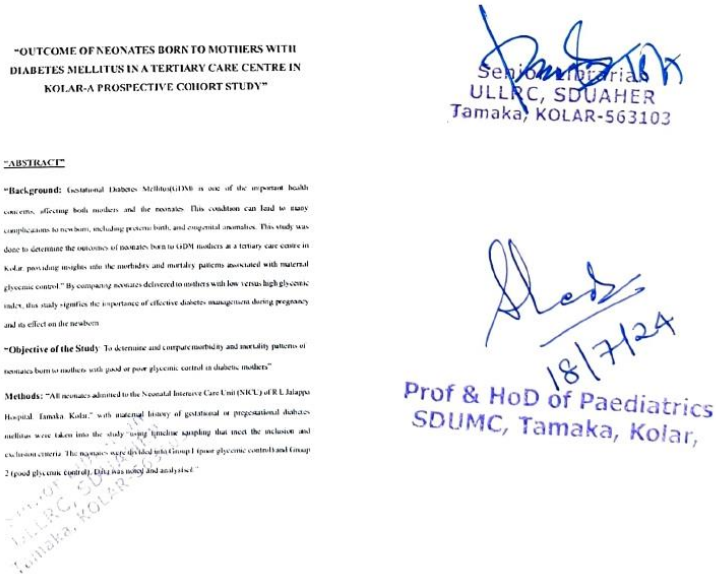

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"ABSTRACT"

"**Background:** Gestational Diabetes Mellitus(GDM) is one of the important health concerns, affecting both mothers and the neonates. This condition can lead to many complications to newborn, including preterm birth, and congenital anomalies. This study was done to determine the outcomes of neonates born to GDM mothers at a tertiary care centre in Kolar, providing insights into the morbidity and mortality patterns associated with maternal glycemic control." By comparing neonates delivered to mothers with low versus high glycemic index, this study signifies the importance of effective diabetes management during pregnancy and its effect on the newborn

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Methods: "All neonates admitted to the Neonatal Intensive Care Unit (NICU) of R L Jalappa Hospital Tamaka Kolar," with maternal history of gestational or pregestational diabetes mellitus were taken into the study using random sampling that meet the inclusion and exclusion criteria. The neonates were divided into Group I (poor glycemic control) and Group 2 (good glycemic control). Data was noted and analysed."

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ACKNOWLEDGEMENT

I would like God to give me the strength, opportunity, and courage throughout the post-graduation and complete my dissertation.

I express my heartfelt and humble gratitude to my beloved guide **Dr. Sudha Reddy V R**, HOD and Professor, Department of Paediatrics who hand-picked this topic for me and graced the study officially with her constant support and expert advice, her encouragement, wise constructive judgment the painstaking effort to weed out errors and her affection during the course of study leaves me permanently indebted to her.

I express my deep sense of gratitude and humble thanks to my senior Professors, **Dr.Krishnappa J** and **Dr. KNV Prasad, Dr.Narendra RR, Dr. James Daniel, Dr.Srikanth** for their advice and encouragement throughout the study.

My heartfelt thanks to Assistant Professors **Dr.Bhanuchand P, Dr.Karthik S, Dr. Shivaraj, Dr Chethan, Dr. Abhishek** for their practical tips, invaluable advice and constant encouragement. I would like to thank I would like to thank my senior Residents **Dr.Bindu, Dr Jaishree, Dr Parimala, Dr Shrishail, Dr Pravallika, Dr Manasa, Dr.Vidhya Shree B** for their guidance and support.

I extend my sincere thanks to my beloved seniors for supporting me throughout. I extend my sincere thanks to all seniors **Dr Jefrin, Dr Mathumitha, Dr Nikitha, Dr Trisali, Dr Prakash, Dr Praveen** for sharing their immense knowledge. I am thankful for their valuable guidance and helping me with my dissertation.

I would like to express my gratitude to my close friends **Dr.Kamal, Dr Mouna, Dr Ramswaroop, Dr Teja, Dr Janhavi** and **Dr Rana** for their

support and love. Heartfelt thanks to my juniors **Dr.Bhargavi, Dr Deepak, Dr Varsha, Dr Deepthi, Dr Teja, Dr Rohit** and **Dr Vishaka, Dr Ashwini, Dr Venkatesh, Dr Supreeth, Dr Indra, Dr Akhila, Dr Pravallika** and **Dr Debashish.**

I would express my deepest gratitude to my beloved parents Mr. **Shivram Chandra, Mrs. Snehalatha** and My sister **Mrs. Padmashree** and her husband **Mr.Raghul** and other family members for constantly believing in me and whose love, blessings made me the person what I am today .

I would like to thank **Mrs.Gayathri, Mr.Jagannath** who had helped me in the clerical work.

Lastly, I would like to express my gratitude to all my interns and nurses of paediatric ward and patients who were part of this study without whose support this study wouldn't have been possible.

Date **Dr. KARTHIK KANANGI**

Place : Kolar

LIST OF ABBREVIATIONS

Glossary	Abbreviations
NICU	Neonatal Intensive Care Unit
DM	Diabetes Mellitus
IADPSG	International Association of Diabetes in Pregnancy Study Group
GDM	Gestational Diabetes Mellitus
DIPAP	Diabetes in Pregnancy and Awareness Project conducted in India
OGTT	Oral Glucose Tolerance Test
IDM	Infant of Diabetes Mother
WHO	World Health Organization
BMI	Body Mass Index
IGF	Insulin-like Growth Factor
HAPO	Hyperglycemia and Adverse Pregnancy Outcome
BW	Birth Weight
GA	Gestational Age
TTNB	transient tachypnea of newborn
RDS	Respiratory Distress Syndrome
CHD	Congenital Heart Disease

IUGR	Intrauterine Growth Retardation
SGA	Small for Gestational Age
LGA	Large for Gestational Age
IUD	Intra Uterine Death
ASD	Atrial Septal Defect
VSD	Ventral Septal Defect
PDA	Patent Ductus Arteriosus
PFO	Patent Foramen Ovale
MSAF	Meconium Stained Amniotic Fluid
PROM	Premature Rupture Of Membranes
LSCS	Lower Segment Caesarean Section

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ABSTRACT

Background: Gestational Diabetes Mellitus (GDM) is one of the important health concerns, affecting both mothers and neonates. This condition can lead to many complications for newborns, including preterm birth and congenital anomalies. This study was done to determine the outcomes of neonates born to GDM mothers at a tertiary care center in Kolar, providing insights into the morbidity and mortality patterns associated with maternal glycemic control. By comparing neonates delivered to mothers with low versus high glycemic index, this study signifies the importance of effective diabetes management during pregnancy and its effect on the newborn.

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Results: Hypoglycemia and hypocalcemia were observed in neonates, but no significant association was found with maternal glycemic control. However, neonatal respiratory distress

and neonatal congenital heart disease were significantly higher in newborns delivered to mothers with poorly controlled glycemic index.

Conclusions: The study signifies the importance of maintaining good glycemic control during pregnancy to improve neonatal health outcomes, highlighting the need for targeted interventions and continuous monitoring of diabetic mothers and newborns delivered to them.

Keywords: *Gestational Diabetes Mellitus, Glycemic Control, Neonatal Respiratory Distress, Congenital Heart Disease*

INTRODUCTION

INTRODUCTION

One of the chronic metabolic diseases that affects mankind is Diabetes mellitus (DM), characterized by abnormalities in the β-cells and/or increased insulin resistance. It impacts people of different ages, including fetuses, newborns and adolescents.[1] DM is one of the most common medical conditions occurring during pregnancy, affecting between 0.5% to 5% of all pregnancies.[2] According to the International Association of Diabetes in Pregnancy Study Group (IADPSG), the global prevalence of gestational diabetes mellitus (GDM) in 2021 was estimated to be 14.0%.

The term GDM refers to a variable-severity high glucose values that first recognized in pregnancy. There is an increased chance of having GDM if the resistance to insulin is present along with inadequate pancreatic function.[4] GDM may be diagnosed for the first time during pregnancy or may occur before pregnancy (pre-gestational or overt diabetes). According to a study done by Anjum SK et al., most of these mothers are diagnosed with GDM (86%) after 20 weeks of gestation as compared to pre-gestational diabetes (14%).[5]

GDM is a complication seen in about 7% of pregnancies overall, accounting for about 200,000 cases every year.[6] Due to its increasing incidence, GDM has acquired significance on a global scale. The rising incidence of GDM is primarily attributed to the rising occurrence of type 2 diabetes and obesity type-2 diabetes and obesity, often referred to as diabesity, among younger women.[7,8]

A community-based research (DIPAP- Diabetes in Pregnancy and Awareness Project) conducted in India indicated that 13.9% of people had GDM. [3]

Many studies proved that pregnant women with GDM with controlled glycemic index gave birth to healthy babies. Numerous studies conducted in the West have shown that GDM is

linked to wide spectrum of negative consequeneces for the mother and the unborn baby. It Ethnicity variations have been noticed in both the incidence of GDM and the pregnancy's prognosis.[9]

The complications that can be included are impaired fetal growth, stillbirth, miscarriage, respiratory distress, cardiomyopathy, congenital malformations, shoulder dystocia, brachial plexus injury, clavicular fracture, metabolic issues like hypoglycemia and hypocalcemia asphyxia and increased perinatal mortality. Complications in mothers may involve preterm labor, early rupture of membranes, infections, hypertensive disorders , polyhydramnios, a higher rate of caesarean and surgical vaginal deliveries, and maternal trauma.[10] Complications associated in neonates born to diabetic mother include: 6% of serious congenital abnormalities, risk of respiratory distress syndrome constituted about 2 %, macrosomia comprised about 28%, hypoglycemia noticed in about 47%, hypocalcaemia comprised of about 2%, hyperbilirubinemia noticed in about 20%, and 34% had large for gestational age. [11] The above risks can be reduced with good glycemic control before and during pregnancy.[11]

One of the strongest predictive factors for diabetes in pregnancy is abnormal oral glucose tolerance test (OGTT); the factor with important attribution is BMI.[12,13] Neonates born to diabetic mothers had higher mortality rates compared to those born to non-diabetic mother. Congenital abnormalities are a major concern, three to four times more common in infants born to diabetic mother.[11]

Management of GDM remains a serious concern in underdeveloped nations. Better management of GDM in developed nations with good glycemic control before and during pregnancy results in better outcomes and better newborn care. According to studies, strict management of the maternal blood sugar levels during pregnancy has been linked to positive perinatal outcomes.[14]As per randomized controlled trials[15,16] -the incidence of macrosomia

decreases when blood glucose values in GDM are well controlled. In addition to providing the best possible obstetric care, coordination between the departments of obstetrics and neonatology is essential for planning newborn resuscitation, IDM assessment, and newborn care.[17] Hence the need for this study is to determine the different morbidity pattern occurring in neonats born to gestational diabetes mothers.

AIMS & OBJECTIVES

OBJECTIVES

Objectives of the study:

1) To determine the morbidity pattern in neonates born to diabetic mother
2) To determine the mortality of neonates born to diabetic mother
3) To compare the effects on morbidity and mortality patterns of neonates born to mothers with good or poor glycemic control in diabetic mothers

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Glucose intolerance, which is a hallmark of gestational diabetes, is more prevalent during pregnancy. It has been a challenge for medical professionals to diagnose gestational diabetes. Various societies worldwide have given a number of criteria to identify GDM. The World Health Organization (WHO) published guidelines in 1999, which was revised in 2006 and were mainly followed, to bring about uniformity. The WHO updated and developed new diagnostic standards for gestational diabetes in 2013. The observed new cut offs are considered according to risk for complications of pregnancy.[18]

A prospective study conducted in India found that prevalence of GDM among pregnant women was 13.9%, the rates differed significantly by location:17.8% in urban areas, 13.8% in semi-urban areas, and 9.9% in rural areas. Moreover, the body mass index (BMI) also affected the incidence. For maternal obesity (BMI $\geq$ 25 kg/m^2) frequency of incidence in rural, semi-urban, and urban settings was 16.1%, 23.8%, and 28.4% respectively.[19] Fetal complications in gestational diabetes mellitus (GDM) can arise due to fluctuating blood glucose levels during pregnancy.

The majority of significant abnormalities occur in the embryonic stage, particularly in the early gestation. As a result of maternal hyperglycemia, the developing fetus receives excess glucose for metabolism, which further affect various metabolites. These include: (1) altered cell lipid metabolism, specifically production of prostaglandin E2, which is important for maintaining the patency of the ductus arteriosus in utero; (2) high glucose levels can cause an excess of reactive oxygen species, which causes oxidative stress and subsequently increases the risk for fetal malformations, particularly neural tube defect; and (3) high glucose levels activate numerous proteins involved in apoptotic cell death, including caspase enzyme.[20]

NEONATAL OUTCOMES

Macrosomia

The most frequent consequence in GDM is macrosomia. A birth weight of 4000 gram or more is considered as macrosomia. However, gestational age is not considered in this definition. Large for gestational age (LGA) refers to a newborn whose birth weight is at or above the 90th percentile or exceeds +2 standard deviations for their gestational age (GA). This criteria helps to identify premature babies with increased fetal growth. When a newborn of a diabetic mother has macrosomia, there may be organomegaly, increased muscular mass, increased body fat, without increase in the size of the brain.[20]

"Sixty years ago, Pedersen-Freinkel proposed the notion that fetal enlargement is associated with greater transfer of maternal glucose across the placenta, which increases the secretion of insulin by pancreatic beta cells of fetus.[21] Upregulation of the Insulin-like Growth Factor (IGF) system causes fetal macrosomia and is crucial factor for growth of fetus. This theory is supported by the various researchers who have described the relationship between maternal glycemia and newborn fat mass or macrosomia.[22,23] More recently, the mother's metabolic environment and placental changes have been linked to other pathways that could potentially contribute to fetal overgrowth. Specifically, in the event of maternal diabetes, there may be an increase in the fetal access to and transportation of maternal lipids. Therefore, maternal diabetes of any kind increases the likelihood of macrosomia.[20]

Preterm birth

The association between GDM and preterm birth is a topic of ongoing debate. In a large cohort study, Hedderson et al. [24] demonstrated that GDM is a risk factor for spontaneous preterm birth. .However, Yogev et al.'s research [25] revealed that there was no statistical

association in the rate of spontaneous preterm delivery between GDM and non-GDM patients. However, both investigations discovered a link between preterm birth and higher mean blood glucose levels or higher glucose results on the OGTT. Thus, it is necessary to weigh the advantages of an early delivery to prevent shoulder dystocia or fetal death against the morbidity associated with preterm birth, particularly respiratory morbidity.

Metabolic disorders

Hypoglycemia

There is an relation between elevated cord C-peptide levels, macrosomia, and neonatal hypoglycemia. This association was verified by the Hyperglycemia and Adverse Pregnancy Outcome (HAPO) study, which revealed a robust association between higher cord serum C-peptide levels and newborn hypoglycemia.[26] A baby born to a diabetic mother is prone to transient hyperinsulinism, which increase glucose uptake by tissue at birth by inhibiting the normal activation of metabolic pathways responsible for producing glucose and ketone bodies.[27]

After birth, significant decrease in blood glucose levels is anticipated due to the disruption of placental flow, which decreases to the minimum value in 1-2 hours for healthy full-term neonate. Due to the activation of metabolic regulatory mechanisms, blood glucose then rises spontaneously after three hours of life, even in the absence of any oral intake. Regardless of the baby's birth weight (BW), if the baby tolerating feeds, the most effective way to prevent hypoglycemia is early and regular breastfeeding. Monitoring of postnatal glucose levels should be done. This enables the identification of infants who are unable to maintain appropriate early glucose homeostasis. [20]

Hypocalcemia[20,28]

Hypocalcemia is defined as a plasma calcium concentration of less than 2 mmol/L or an ionized calcium concentration of less than 1.1 mmol/L. Pregestational insulin-dependent diabetic women have been the primary cases of transient neonatal hypocalcemia, which may be partially linked to decrease in magnesium levels in mother and subsequently decrease in magnesium levels in fetus. Level of calcium in neonates correlate inversely with mother's HbA1c levels. Pregnancy likely disrupts calcium and phosphorus metabolism, leading to reduced levels of calcium and vitamin D, particularly in the third trimester. Treatment can include calcium gluconate (40-60 mg/kg/day) administered orally or intravenously, or magnesium, depending on plasma level.

Hyperbilirubinemia [20]

Neonates born to mothers with GDM have increased risk of developing icterus. They are prone to higher levels of oxidative stress, insulin, and IGF in utero. Additionally, elevated red blood cell mass associated with polycythemia in these infants further increases the likelihood of hyperbilirubinemia.

Hematologic disorders[20]

According to reports, neonates of GDM have higher chance of have polycythemia, or increased hematocrit (Hct) levels more than 65%. The mechanisms that are implicated are increased levels of insulin in fetus and decrease in transplacental oxygen delivery to the fetus, thereby resulting in fetal hypoxia and increased erythropoietin levels. Red blood cells synthesis can also be accelerated by elevated insulin and IGF levels. In the event of polycythemia, infants born to diabetic mothers have higher risk for hypoglycemia because of

the higher glucose consumption. In symptomatic newborns, partial exchange transfusion using saline solution should be administered.

Respiratory disorders [20,29]

Neonates born to GDM at higher risk of experiencing transient tachypnea of newborn (TTNB) or respiratory distress syndrome (RDS). This is more likely to occur in babies born by cesarian section (indication being macrosomia).[29]

Neonates born to mothers with GDM who have poor glycemic control may be at an increased risk for delayed lung maturation

Hypertrophic Cardiomyopathy

Neonates born to mothers with poor glycemic control are at a higher risk of developing hypertropic cardiomyopathy, it can also impact the heart in addition to its primary effect on the interventricular septum[20]. Myocardial hypertrophy has been observed in both pregestational diabetes and GDM, with wide distribution range, affecting 25% and 75% of newborns delivered to diabetic mothers.[30] Regardless of pregestational or gestational diabetes, recent research revealed that adequate maternal glycemic control does not completely avoid fetal cardiac function impairment and interventricular septum hypertrophy [31,32].

Cardiac malformations [20,33]

Some data suggests that neonates born to GDM have increased risk of congenital abnormalities (ORs between 1.1 and 1.3).[33] Most frequent described cardiac malformations are truncus arteriosus, hypoplastic left heart syndrome," transposition of the major arteries, double outlet right ventricle and ventricular septal abnormalities .Antenatal ultrasonography imaging may be an essential tool for screening for any structural or functional defect in fetal

heart. Prenatal screening of any cardiac abnormality is important for further cardiologist intervention if required. If a newborn exhibits clinical symptoms of cardiomyopathy (heart failure) or congenital cardiac abnormalities (cyanosis, murmur), proper follow up should be done with an 2-D echocardiography.

Neurological impairments

Perinatal asphyxia[20]

There is a higher incidence of perinatal asphyxia in newborns with macrosomia, especially if there is presentation with shoulder dystocia. Fetal hypoxia, a symptom of an impaired fetal environment, has also been suggested as a contributing factor.

Brachial plexus injuries [20,34]

Birth trauma can cause symptoms due to injury of brachial plexus. Cervical roots C5 to C7 are affected in Erb's palsy. Presentation in the newborn is flexed wrist and an internally rotated arm on the affected side. When the phrenic nerve is affected, palsy of the hemi-diaphragm is also seen, resulting in respiratory insufficiency and the need for mechanical ventilation. Around 0.2% to 3% of babies born to diabetes mothers have brachial plexus palsy.[34]

Poor suckling

Neonatal activity may be affected by maternal GDM, which can result in hypotonia and lethargy due to delayed brain maturation. In a study by Bromiker et al.[35], it was noted that on the third day of life, poorer suckling patterns was observed in neonates born to mothers on insulin; as compared to neonates born to mothers on diet control. “This study highlighted that newborns show a certain level of neurologic immaturity during the early neonatal period.[3]

Digestive impairment

Newborns born to diabetic mothers may experience feeding difficulties due to poor nursing patterns and may also have a small colon, which is functional obstruction of the lower intestine that is similar to Hirschsprung disease. Although the pathogenesis is idiopathic, there is a strong correlation between neonatal small colon and maternal glycemic control. Treatment is conservative as long as there is no intestinal perforation.[36]

Salima et al. in 2018" conducted a cross-sectional observational study to evaluate the neonatal complications and mortality among neonates born to diabetic mothers. It was observed that twenty (22.2%) were preterm deliveries; forty (44.4%) had birth weight more than 4 kg; twelve (13.3%) had Apgar scores less than seven at one minute; thirty-five (38.1%) had blood sugar levels less than forty at one hour; and forty-seven (52.2%) required IV fluids to maintain blood sugar levels. Out of total deliveries 78.9% were done by caesarean section, 34.5% out of which were emergency section. Nine (10%) had hypocalcemia, seven(7.8%) had hyperbilirubinemia, and twenty(22.2%) had episodes of hypoglycemia. There were 23 cases of RDS (25.5%), 5 cases of neonatal sepsis (5.6%), 8 cases of TTNB (8.8%), 1 case of birth asphyxia (1.1%), 24 cases of congenital abnormalities (26.6%), 19 cases of congenital heart disease (21.1%), and 1 death (1.1%). It was concluded that macrosomia, premature birth, congenital abnormalities, CHD, RDS, TTNB, hypoglycemia, hypocalcaemia, and hyperbilirubinemia are some of the significant problems among these neonates. [37]

A hospital-based prospective study by **Anjum and Yashodha** in 2018, aimed to assess outcomes in newborn of diabetic mothers and the relationship between maternal glycemic status and various complications in newborn. In the study, 86% of the mothers had GDM, while 14% were pre-gestational diabetics. Babies of diabetic mothers were reported to have a

variety of issues, including respiratory distress syndrome, congenital cardiac disorders, polycythemia, hyperbilirubinemia, hypoglycemia, hypocalcemia, macrosomia, preterm, and TTNB. Of these, hypoglycemia accounted for 54% of observed complications, hypocalcemia accounted for 43%, polycythaemia for 35%, and macrosomia for 15%. Glycemic control in mothers was found to be significantly correlated with such outcomes. Therefore it was concluded that for the management of these high-risk neonates, babies should be delivered in hospitals with specialized neonatal care.[5]

A retrospective case-control study was done by Capobianco et al38 in 2020 on GDM and pregestational diabetes, to assess the maternal-fetal and neonatal clinical outcomes and to compare them with those without diabetes. It was observed that the incidence of premature delivery was 18.3% in the GDM patient group, 66.7% in type 1 or 2 diabetes mellitus patient group, and 23.2% in nondiabetic patient groupIn the control group, four out of 207 fetuses (1.93%) experienced fetal growth disorders like intrauterine growth retardation (IUGR) and small for gestational age (SGA), whereas 20 out of 207 fetuses (9.67%) in the case group were affected. Among the gestational diabetes mellitus (GDM) group, 16 out of 183 fetuses (8.74%) had these conditions, compared to 4 out of 24 fetuses (16.67%) in the type 1/type 2 diabetes group. Additionally, a strong correlation was found between preeclampsia and type 1 diabetes.[38]

In 2021, **Bayoumi et al**.[4] conducted a population-based cohort study in Qatari population to assess the effects of gestational diabetes mellitus (GDM) on various growth parameters in newborns, both before and after pregnancy." A total of 5195 infants were examined out of 17020 live births. Among the population of Qatar, 24.25% had GDM. Those women who had pre-pregnancy DM had higher HbA1c values before delivery than those with GDM. there

was no significant changes noted in mean length, head circumference and incidence of congenital anomalies.

A prospective observational study was done by **Satishkumar and colleagues**[39] in 2022, to determine the complications in infants born to mothers with DM. Among 70 neonates born to mothers with diabetes, 62 newborns were assessed. Two intrauterine deaths (IUDs) and 6 stillbirths were excluded from the research. It was observed that 29% were born preterm, while 70.96% were born term. Amongst total of 62 newborns, 64.5% neonates were born to women with GDM, 32.3% to mothers with type 2 DM, and 3.2% neonates were born to mother with type 1 DM. There were 12.9% of SGA cases and 16.2% of LGA cases. There were four neonates with hypocalcemia (6.7%). In 4 (13.3%) cases, hyperbilirubinemia was detected. Eight neonates had sepsis. 21.3% of cases had Atrial Septal Defect (ASD)/Patent Foramen Ovale (PFO), accounting for 42.9% of CHD cases. Ventral septal defect (VSD) was present in 10.7% of cases, 7.1% had patent ductus arteriosus (PDA), and 3.5% had septal hypertrophy. One patient had RDS, while the other had pyloric atresia. "There was no statistical association noted between HbA1c levels and outcomes like macrosomia, hypoglycemia, or congenital abnormalities in this study.[39]

In 2023, Muntean et al.17 conducted a prospective case-control study to examine how maternal glycemic control affects complications in newborns of diabetic mothers. Mothers with diabetes had higher body mass indices (BMIs), and cesarean section was the most prevalent mode of delivery. While comparing to control group, affected group had higher incidence of outcomes like congenital heart defects or myocardial hypertrophy, respiratory disease, NICU admission, and need for phototherapy. This study emphasized that even with optimal maternal glycemic control, poor newborn outcomes are

MATERIALS & METHODS

MATERIAL AND METHODS

Study place: Neonatal Intensive Care Unit (NICU) at R L Jalappa Hospital, Tamaka, Kolar"

Source of data: All neonates admitted to RL Jalappa Hospital (intramural and extramural) with maternal history of GDM and pregestational diabetes mellitus during the period of study.

Study population: Neonates born to diabetic mothers admitted to RL Jalappa Hospital, Tamaka, Kolar

Study design: A Prospective Cohort Study

Sampling technique:

Timeline sampling fitting our inclusion and exclusion criteria was considered in our study.

Babies born to mothers with poor glycemic control were considered as Group 1, and babies born to mothers with good glycemic control were considered as Group 2"

Sample size:

$$n = \frac{2(\bar{p})(1-\bar{p})(Z_{\beta} + Z_{\alpha/2})^2}{(p_1 - p_2)^2}$$

n - Sample size in each group (assuming equal-sized groups)

$(\bar{p})(1-\bar{p})$ - measure of variability (similar to standard deviation)

Z_{β} - represent the desired power

$Z_{\alpha/2}$ - represents the desired level of statistical significance (typically 1.96)

$(p_1 - p_2)^2$ - effective size (the difference in proportions)

Incidence of hypoglycemia among newborn delivered to GDM with HbA1c above 6.5 was 37% as reported by study Saha D et al.[40] Considering the power of 90%, 99% confidence

interval effective size of 76%, with P1 being 7 % and P2 being 30%, and according to unpublished institutional data (70 NICU admissions/year –neonates born to diabetic mothers), the estimated sample size of the study was 156.

Study period: September 2022 – March 2024

Method of collection of data:

- **Inclusion criteria:**

  Infants of diabetic mothers (Preterm, term, and post-term) admitted to R L Jalappa Hospital (Intramural and extramural) were included in the study.

- **Exclusion criteria:**

  a) Neonates with meconium stained amniotic fluid (MSAF), during the delivery.

  b) Maternal PROM (>18 hours).

Ethical considerations: The study was approved by the institutional ethical committee. Informed written consent was obtained from all the parents /guardians of the study participants and only those participants whose parents or guardians were willing to consent were include in the study. The voluntary nature of participation was explained to the parents/guardians of the participants before obtaining consent. Confidentiality of the study participants was maintained.

Methodology:

All the neonates with maternal GDM or pregestational diabetes mellitus admitted to RLJH during the study period were included in the study, after taking informed consent from the mother. The following data of all the pregnant women with diabetes were obtained:

a)Mother's age

b)Obstetric score

c)Antenatal scan

d)Type of diabetes– pre-gestational/ gestational

e)Glycemic control (HbA1c levels)_____at ____ weeks of gestation

The following neonatal data were noted: mode of delivery, gestational age, birth weight of the baby, and gender of the baby. The weight of the neonate was plotted on Fenton's chart (annexure 1) and was classified as small for gestational age (SGA), appropriate for gestational age (AGA), and large for gestational age (LGA).

At birth, the neonates were examined thoroughly for any visible congenital anomalies and birth trauma. APGAR score was observed at 1 minute and 5 minutes.

As per Departmental protocol, all neonates of diabetic mothers were admitted to NICU. For all neonates the following investigations were done on Day 1:

1)Blood glucose levels

2)Complete blood count

3)Serum calcium

The following investigations were done as and when needed:

a)Chest x-ray

b)Arterial blood gas analysis

c)Magnesium levels

d)Neurosonogram

e)USG Abdomen

f)2 D ECHO by cardiologist– for neonates suspected to have cardiac anomalies For all neonates, serial blood glucose levels were measured using a glucometer. It was done at birth 2, 6, 12, 24, 48 and 72 hours.

For the purpose of the study, the following definitions were used:

1. Gestational Diabetes- Gestational diabetes mellitus (GDM) is a state of hyperglycemia (fasting plasma glucose ≥91.8mg/dl, 1 h ≥ 180mg/dl, 2 h ≥153mg/dl during a 75 g oral glucose tolerance test according to IADPSG/WHO criteria) that is first diagnosed during pregnancy after 20 weeks of gestation. [41]

2. Pregestational Diabetes/Overt Diabetes- state of hyperglycemia before pregnancy or before 20 weeks of gestation (fasting plasma glucose ≥ 126 mg/dl, postprandial ≥ 200mg/dl, HbA1c ≥ 6.5%)[42]

3.Good glycemic control- maternal HbA1c < 6.5%[42]

4. Poor glycemic control - maternal HbA1c ≥ 6.5%[42]

5. AGA- defined as birth weight between the 10th percentile to 90thfor the gestational age, as per WHO growth chart[43]

6. SGA- defined as weight below the 10thpercentile for the gestational age, as per WHO growth chart[43]

7. LGA- defined as weight above the 90th percentile for the gestational, as per WHO growth charts[43]

8. Respiratory distress- one or more signs of increased work of breathing, such as tachypnoea, nasal flaring, chest retractions, or grunting.[44]

9. Birth injury - is defined as the structural destruction or functional deterioration of the neonate's body due to a traumatic event at birth- Injury to the brachial plexus, Fracture to the clavicle, Injury to the spine or spinal, Subdural, and cerebral haemorrhage.[45]

The babies were divided into two groups based on maternal glycemic control as follows:

A)Group 1: consists of neonates born to mothers with poor glycemic control (HbA1c≥ 6.5)

B)Group 2: consists of neonates born to mothers with good glycemic control (HbA1c< 6.5)

Statistical analysis:"

Descriptive statistical mean standard deviation and confidence interval were used for parameters like age Blood glucose levels, calcium, magnesium, etc

Frequency and percentage were used for outcomes amongst infants of diabetic mothers."

Data was entered into a Microsoft Excel data sheet and was analysed using SPSS 22 version software. Categorical data was represented in the form of Frequencies and proportions. The chi-square test was used as a test of significance for qualitative data. "

Graphical representation of data: MS Excel and MS Word were used to obtain various types of graphs such as bar diagrams and pie diagrams.

P value (Probability that the result is true) of <0.05 was considered statistically significant after assuming all the rules of statistical tests.

Statistical software: MS Excel, SPSS version 22 (IBM SPSS Statistics, Somers NY, USA) was used to analyse data.

RESULTS

RESULTS

A total of 156 samples were included in the present study.

Table 1: Distribution of cases according to Maternal Glycemic Control (n=156)

Maternal Glycemic Control	Number	Percentage
Group 1(HbA1c ≥ 6.5) Poor glycemic control	70	45
Group 2 (HbA1c < 6.5) Good glycemic control	86	55

Figure 1: Distribution according to maternal glycemic control

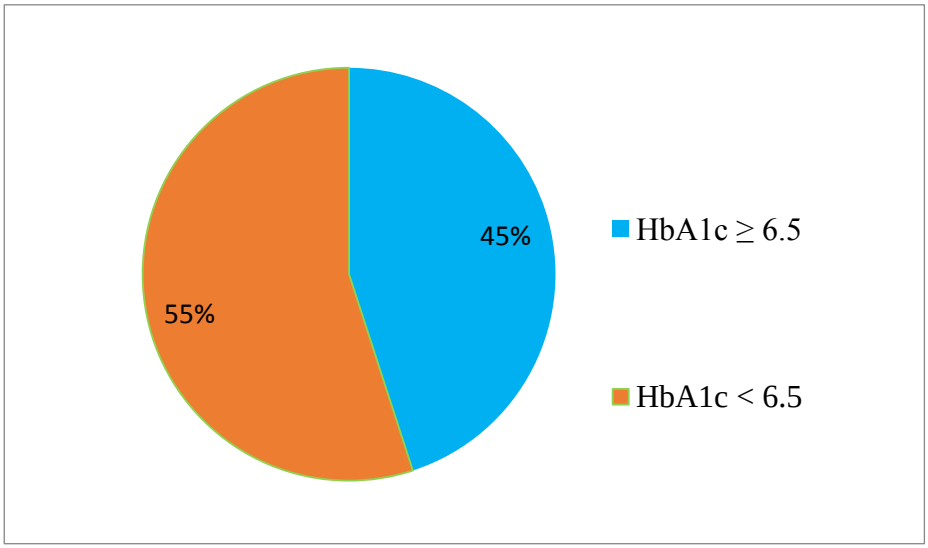


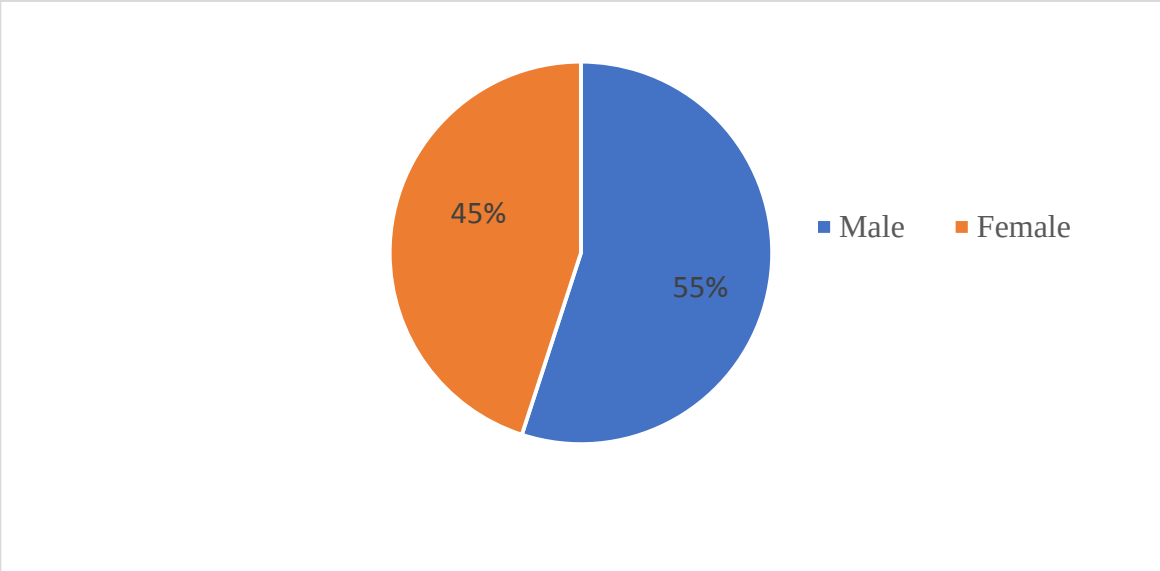
Table 1 and Figure 1 depict the distribution of cases according to maternal glycemic control.

Table 2: Distribution of cases according to gender (n=156)

Gender	Group 1 (n=70)	Group 2 (n=86)
Male	34 (48.6)	53 (61.6)
Female	36 (51.4)	33 (38.4)

There were 70 neonates (45%) born to mothers with poor glycemic control (Group 1) and 86 neonates (55%) were born to mothers with good glycemic control (Group 2).

Figure 2: Distribution of cases according to gender



Out of 70 infants born to mothers with poor glycemic control, 51.4% were female and 48.6 % were males. Distribution was almost similar.

Among infants born to mothers with good glycemic control majority, 61.6% were males while 38.4% were female. There was a male preponderance with a male-to-female ratio of 1.6:1

Table 3: Distribution of cases according to birth weight (n=156)

Birth weight (in kgs)	Group1 (n=70)	Group 2 (n=86)
<1.5	3 (4.3)	3 (3.5)
1.5-2.5	19 (27.1)	32 (37.2)
>2.5-3.5	35 (50.0)	43 (50.0)
>3.5	13 (18.6)	8 (9.3)

Figure 3: Distribution of cases according to birth weight (n=156)

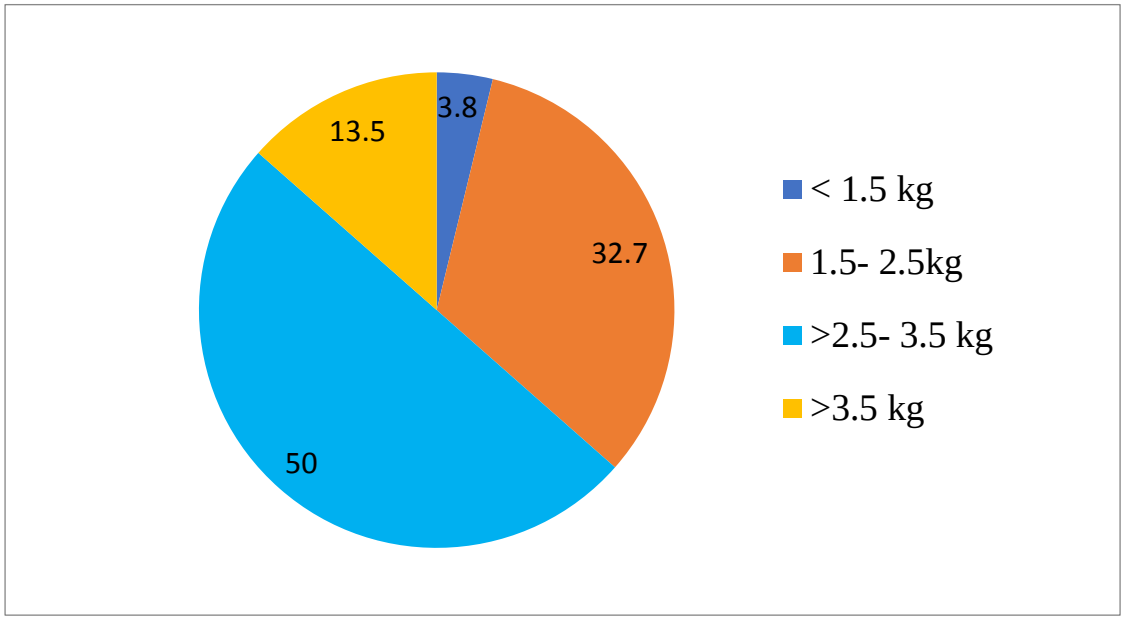


Table 3 and Figure 3 depict the distribution of cases according to birth weight. Out of 70 neonates in group 1,50% were in the birth weight category of >2.5 to 3.5kg. Similarly in group 2 out of 86 neonates,50% were in similar birth weight category of >2.5-3.5kg. Only 4.3% and 3.5% of neonates in Group 1 and Group 2 respectively had birth weight of <1.5 kg. In the birth weight category of >3.5 kg, 18.6% and 9.3% of neonates were in Group 1 and Group 2 respectively. In the birth weight category of 1.5-2.5 kg, 27.1% and 37.2% of neonates were in group 1 and group 2 respectively.

Table 4: Distribution of cases according to gestational age (n=156)

Gestational age	Group 1 (n=70)	Group 2(n=86)
Early Preterm (< 32 weeks)	4 (5.7)	2 (2.3)
Late Preterm(32weeks- <37 weeks)	23 (32.9)	22 (25.6)
Term (37-41 weeks)	43 (61.4)	60 (69.8)
Post-term (≥ 42 weeks)	0(0)	2 (2.3)

Figure 4: Distribution of cases according to gestational age (n=156)

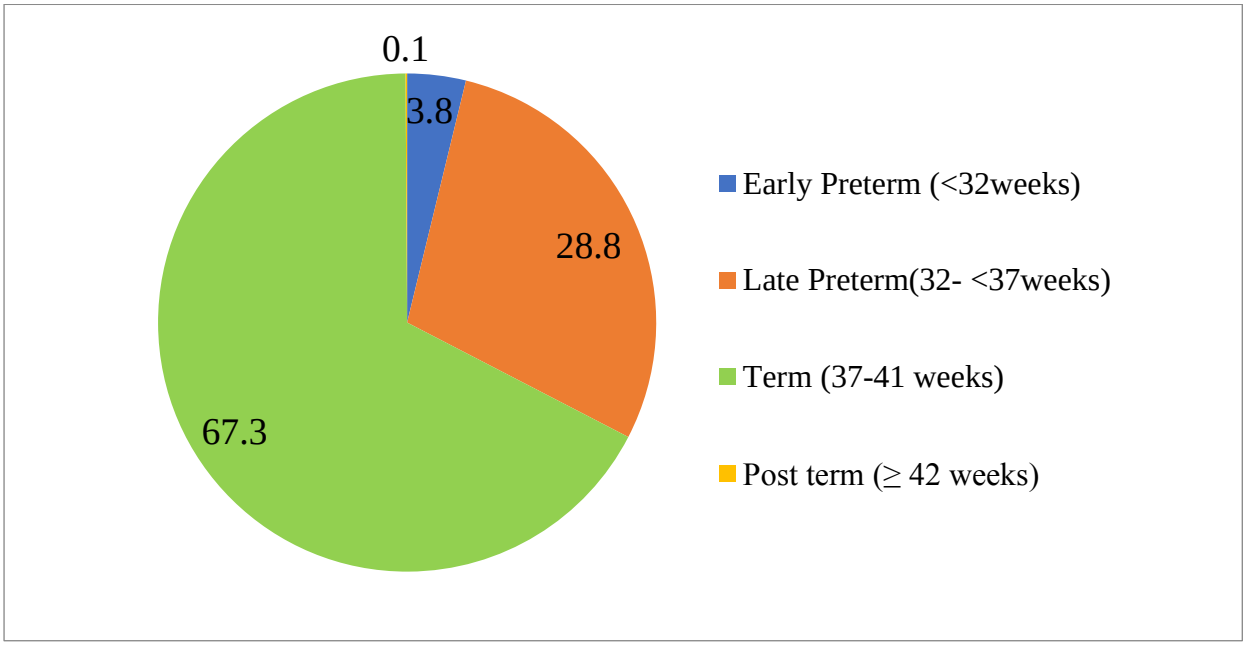


Table 4 and Figure 4 shows the distribution of cases according to gestational age.

Table 5: Distribution of cases according to maternal obstetric score (n=156)

Obstetric score	Group 1 (n=70)	Group 2 (n=86)
Primigravida	25 (35.7)	39 (45.3)
Multigravida	45 (64.3)	47 (54.7)

In Group 1, the majority (61.4%) were term neonates. Twenty-three (32.9%) of neonates were late preterm while 5.7% were early preterm neonates. None of the neonates were post-term in group 1.

In Group 2, the majority (69.8%) were term neonates. Late preterm neonates constituted 25.6%. Early preterm neonates and post-term neonates were 2.3% each.

Figure 5: Distribution of cases according to maternal obstetric score (n=156)

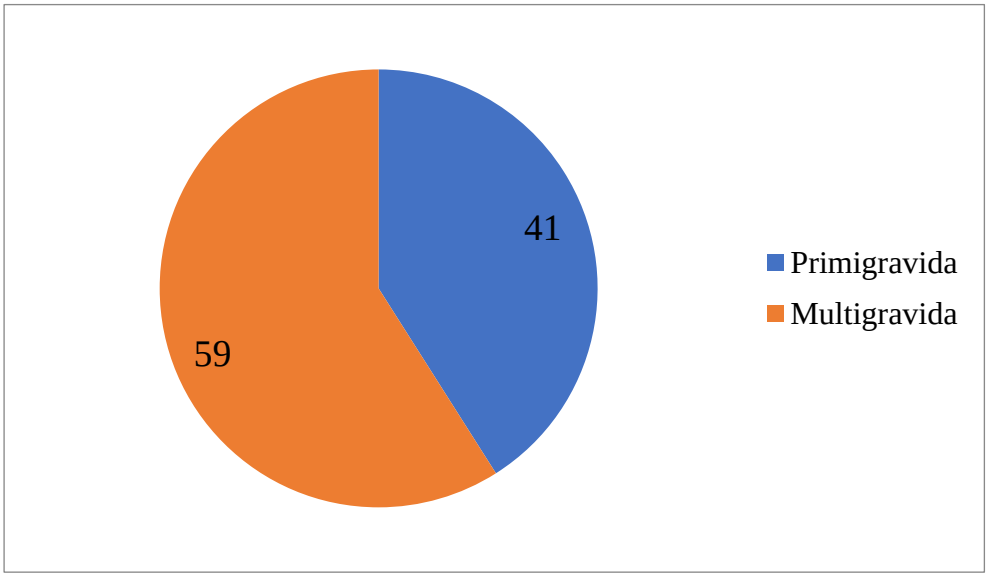


Table 5 and Figure 5 depict the distribution of cases according to maternal obstetric score. Majority (64.3%) of mothers in Group 1 were multigravida. In Group 2, 54.7% were multigravid while 45.3 % were primigravida

Table 6: Distribution of cases according to mode of delivery (n=156)

Mode of Delivery	Group 1 (n=70)	Group 2 (n=86)
LSCS	42 (60.0)	59 (68.6)
Forceps-assisted vaginal delivery	3 (4.3)	0 (0.0)
Vacuum-assisted vaginal delivery	4 (5.7)	2 (2.4)
Normal vaginal delivery	21 (30.0)	25 (29.0)

Figure 6: Distribution of cases according to mode of delivery

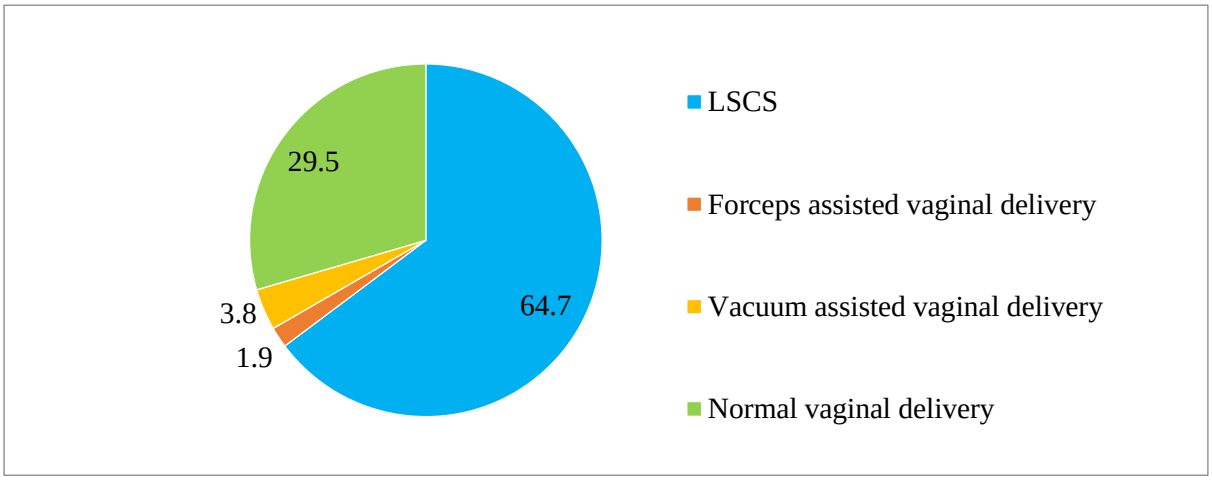
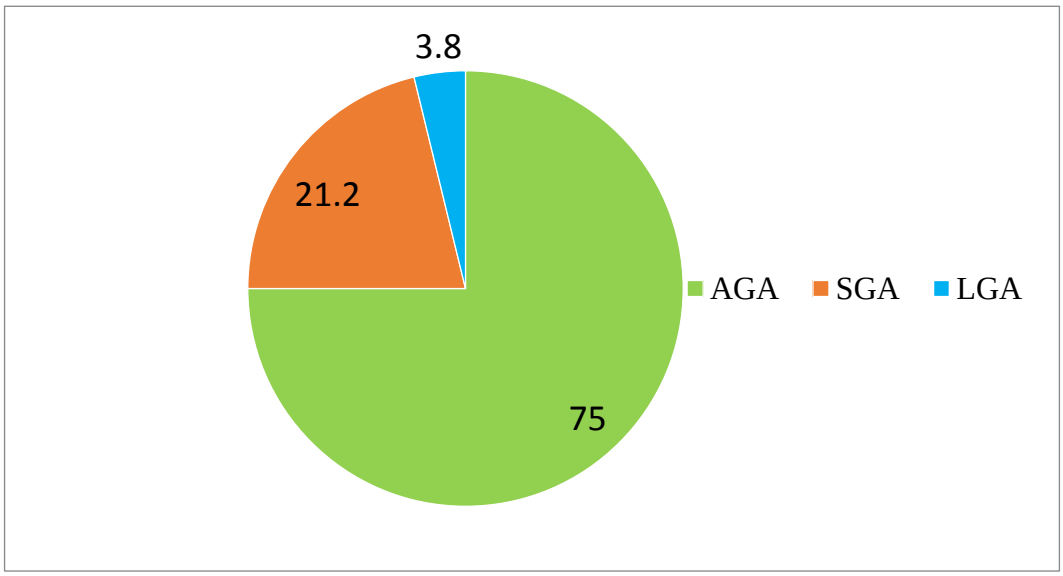


Table 6 and Figure 6 depict the distribution of cases according to the mode of delivery. Majority (60%) of neonates in Group 1 were delivered by LSCS and 45.7% were delivered through normal vaginal route. Forceps-assisted vaginal delivery + vacuum-assisted vaginal delivery was present in 4.3 % + 5.7% respectively. In Group 2, majority (68.6%) of neonates were delivered by LSCS, and 30% were delivered through normal vaginal route. Vacuum-assisted vaginal delivery in Group 2 was present in 2.4% whereas there were no forceps-assisted vaginal delivery present in Group 2.

Table 7: Distribution of cases according to weight for gestational age (n=156)

Weight for gestational age	Group 1 (n=70)	Group 2 (n=86)
AGA	56 (80.0)	61 (70.9)
SGA	10 (14.3)	23 (26.7)
LGA	4 (5.7)	2 (2.3)

Figure 7: Distribution of the cases according to weight for gestational age



- Table 7 and Figure 7 depict the distribution of cases according to weight for gestational age. Majority (80%) of neonates in Group 1 were appropriate for gestational age, 14.3% and 5.7% were small and large for gestational age respectively. In Group 2, majority of neonates (70.9%) were appropriate for gestational age, whereas 26.7% and 2.3% neonates were small and large for gestational age. Overall only 3.8% of neonates were large for gestational age.

Table 8: Distribution of cases according to type of maternal diabetes (n=156)

Type of diabetes	Group 1 (n=70)	Group 2 (n=86)
Gestational	51 (72.9)	73 (84.9)
Pregestational	19 (27.1)	13 (15.1)

Figure 8: Distribution of cases according to type of maternal diabetes

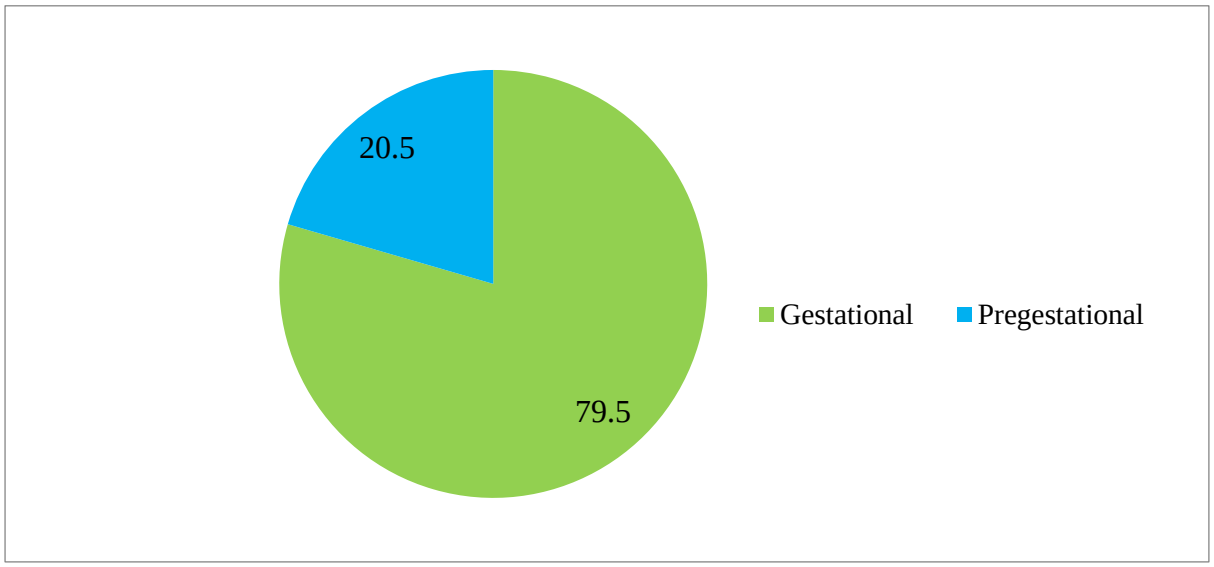


Table 8 and Figure 8 depict the distribution of cases according to type of maternal diabetes. In Group 1, majority (72.9%) of neonates were born to mothers with gestational diabetes, and 27.1% were born to mothers with pregestational diabetes.

Majority (84.9%) of neonates in Group 2 were born to mothers with gestational diabetes whereas 15.1% were born to mothers with pregestational diabetes.

Table 9: Distribution of cases according to Morbidity Pattern

MORBIDITY PATTERN	Number	Percentage
Neonatal hypoglycemia	23	14.7
Neonatal hypocalcemia	31	19.9
Neonatal respiratory distress	45	28.8
Congenital heart disease	47	30.1

Figure 9: Distribution of cases according to morbidity pattern

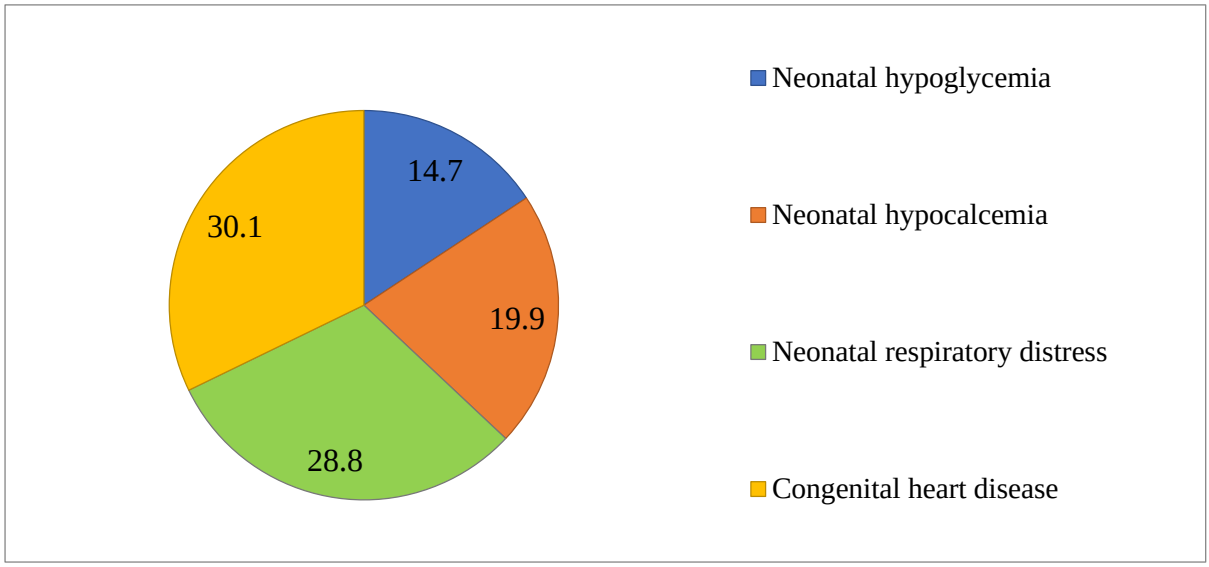


Table 9 and Figure 9 depict the overall morbidity pattern. Congenital heart disease was present in 30.1 % of cases, followed by respiratory distress in 28.8%, hypocalcemia in 19.9%, and hypoglycemia in 14.7%.

ANALYSIS

Table 10: Comparison of neonatal hypoglycemia between two groups

NEONATAL HYPOGLYCEMIA	Group 1	Group 2	TOTAL	P VALUE	ODDS RATIO
PRESENT	13 (56)	10 (44)	23(100)	1.48 (0.22)	1.73
ABSENT	57 (42.8)	76 (57.2)	133(100)		

- Table 10 depicts the comparison of neonatal hypoglycemia in two groups. In the present study, 23 neonates had episodes of hypoglycemia. Amongst these neonates, 56% were born to mothers with poor glycemic control, whereas 44% of neonates were born to mothers with good glycemic control. In Group 1, 18.6% of neonates had episode of hypoglycemia whereas in Group 2 only 11.6% of neonates had episode of hypoglycemia.
- The P value between the maternal glycemic control and neonatal hypoglycemia was 0.22(not significant). Hence the study found no statistically significant association between them.

Table 11: Comparison of neonatal hypocalcemia between the two groups

NEONATAL HYPOCALCEMIA	Group 1	Group 2	TOTAL	P VALUE	ODDS RATIO
PRESENT	17 (54.8)	14 (45.2)	31(100)	1.554 (0.2)	1.65
ABSENT	53 (42.4)	72 (57.6)	125(100)		

- Table 11 depicts the comparison of neonatal hypocalcemia according to maternal glycemic control. In the present study, neonatal hypocalcemia was present in 31 neonates. Amongst these neonates, 54.8% born to mothers with poor glycemic control had hypocalcemia. Whereas only 45.2% of neonates born to mothers with good glycemic control had hypocalcemia.

- The P value between the maternal glycemic control and neonatal hypocalcemia was 0.21 (not significant). Hence the study found no significant association between them.

Table 12: Comparison of neonatal respiratory distress between the two groups

RESPIRATORY DISTRESS	Group 1	Group 2	TOTAL	P VALUE	ODDS RATIO
PRESENT	27 (60)	18 (40)	45(100)	5.85 **(0.016*)**	2.37
ABSENT	43 (38.7)	68 (61.3)	111(100)		

- Table 12 depicts the comparison of neonatal respiratory distress between the two groups. In our study, neonatal respiratory distress was present in 45 neonates. Amongst these cases, 60% were born to mothers with poor glycemic control, while 40% were born to mothers with good glycemic control.

- The P value between maternal glycemic control and neonatal respiratory distress was **0.016 (significant).** Hence the study found a significant association between neonatal respiratory distress and poor maternal glycemic control.

- Outcomes of neonates born to mothers with poor glycemic control, having respiratory distress is **2.37 times** more than those born to mothers with good glycemic control.

Table 13: Comparison of congenital heart disease between the two groups

CONGENITAL HEART DISEASE	Group 1	Group 2	TOTAL	P VALUE	ODDS RATIO
PRESENT	27 (57.4)	20 (42.6)	47(100)	4.30 **(0.038*)**	2.072
ABSENT	43 (39.5)	66 (60.5)	109(100)		

- Table 13 depicts the comparison of congenital heart disease between the two groups. In our study, it was observed that congenital heart disease was present in 47 neonates. Amongst these neonates, 57.4% were born to mothers with poor glycemic control, while 42.6% of neonates with congenital heart disease were born to mothers with poor glycemic control.

- The P value between the maternal glycemic control and congenital heart disease was **0.038 (significant).** Hence our study found a significant association between congenital heart disease and poor maternal glycemic control.

- Odds of a neonate born to a mother with poor glycemic control, having congenital heart disease is **2.07 times** more than those born to mother with good glycemic control.

- Amongst 47 neonates with congenital heart disease, it was observed that some neonates presented with more than one 2D echo findings. There were 44 neonates with atrial septal defect (ASD), 8 neonates with ventral septal defect (VSD), and 8 neonates with significant patent ductus arteriosus (PDA).

Table 14: Comparison of congenital heart disease (ASD) between the two groups

ASD	Group 1	Group 2	Total	P value	Odds ratio
Present	25(56.8)	19(43.2)	44(100)	3.536 (0.06)	1.959
Absent	45(40.2)	67(59.8)	112(100)		

Table 14 compares the occurrence of atrial septal defect between the two groups. It was observed that out of 44 neonates with atrial septal defect, 56.8% were born to mothers with poor glycemic control, while 43.2% of neonates were born to mothers with good glycemic control.

The p-value between the maternal glycemic control and atrial septal defect was **0.06 (not significant).** Hence our study found no significant statistical association between atrial septal defect and maternal glycemic control.

Table 15: Comparison of congenital heart disease (VSD) between two groups.

VSD	Group 1	Group 2	Total	P value	Odds ratio
Present	4(50)	4(50)	8(100)	0.090	1.242
Absent	66(44.6)	82(55.4)	148(100)		

Table 15 compares the occurrence of ventral septal defect between the two groups. It was observed that out of 8 neonates with ventral septal defect, 50% of neonates were born to mothers with poor glycemic control, and 50% of neonates were born to mothers with good glycemic control.

P value between the maternal glycemic control and atrial septal defectwas **0.09 (not significant).** Hence our study found no significant statistical association between ventral septal defect and maternal glycemic control.

Table 16: Comparison of congenital heart disease (PDA) between the two groups

PDA	Group 1	Group 2	Total	P value	Odds ratio
Present	3(37.5)	5(62.5)	8(100)	0.185 (0.667)	0.725
Absent	67(45.2)	81(54.8)	148(100)		

Table 16 compares the occurrence of patent ductus arteriosus between the two groups. It was observed out of 8 neonates with patent ductus arteriosus, 37.5% of neonates were born to mother with poor glycemic control, and 62.5% of neonates were born to mothers with good glycemic control.

P value between the maternal glycemic control and patent ductus arteriosus was **0.185 (not significant).** Hence our study found no significant statistical association between patent ductus arteriosus and maternal glycemic control.

Table 17: Comparison of congenital anomalies between the two groups excluding congenital heart diseases

Congenital Anomaly	Group 1	Group 2	Total	P value	Odds ratio
Present	1(50)	1(50)	2 (100)	0.883	1.23
Absent	69(45)	85(55)	154 (100)		

Table 17 compares the occurrence of congenital anomalies between the two groups excluding congenital heart disease. In our study, out of 2 neonates with congenital anomalies, one neonate was born to mother with poor glycemic control and the other was born to mother with good glycemic control.

Mortality

- 1 out of 156 neonates had the outcome of death.

Table 18: Comparison of mortality between the two groups

Mortality	Group 1	Group 2	Total	P value	Odds ratio
Present	1	0	1 (100)	-	-
Absent	69(44.5)	86(55.5)	155 (100)		

Table 18 compares the occurrence of mortality between the two groups. In our study, only one neonatal death was noted, which was born to mother with poor glycemic control. There was no association between neonatal mortality and maternal glycemic control in our study.

DISCUSSION

DISCUSSION

The most prevalent endocrine condition during pregnancy is diabetes mellitus. The prognosis of the offspring is determined by the duration, severity, and glycemic control of the mother's diabetes throughout the pregnancy.[46] This current study is a prospective cohort study to determine the morbidity, mortality pattern and compare the various parameters among neonates born to mothers with GDM with poor (group 1) and good glycemic control (group 2). A total number of 156 subjects satisfying the inclusion criteria were included in the analysis. A slight female predominance (51.4%) was seen in Group 1 while in case of the Group 2, males (61.6%) were predominant. Study done by **Satishkumar and colleagues**[39] observed male predominance (54.5%) while female were 35.5%.

Most of the individuals (50% each) in both groups belong to the birth weight category >2.5-3.5 kg followed by 1.5- 2.5 kg. **Satishkumar and colleagues**[39] observed 54.8% of the cases to have a birth weight of 2.5 - 4 kg. The data from the current study did not reveal any significant difference in the birth weight of the neonates born to either of the groups. No difference in birth anthropometric measurements seen between neonates born to GDM and those who were not.[47,48] Conversely, **Baptiste-Roberts K. et al**.[49] found that even after adjusting other factors like maternal BMI, and pregnancy weight gain, mothers with GDM gave birth to children with higher birth weights than their non-diabetic counterparts. Furthermore, compared to newborns of non-GDM mothers, **Sletner L et al.**[50] discovered that fetus of mothers with GDMd ha growth retardation in 2nd trimester of pregnancy but grew faster later on till delivery.

Most of the mothers belonging to Group 1 (64.3%) as well as Group 2 (54.7%) were multigravid. However, in a study by **Satishkumar and colleagues** [39], it was observed that most of the GDM mothers were primigravida. **Salima et al**[37] observed 90% of participants with GDM to be multigravida. It was observed rise in parity is a risk factor for GDM. This is consistent with research conducted in by **Qadir et al.**[51], who found that 76% of patients with GDM were multigravida, and **Randhawa and colleagues**[52] also reported that 80% of patients with GDM were multiparous. However, **Kheir et al**. [11] discovered that 40.7% of women with GDM were primiparous and 59.7% were multiparous.

Majority of the participants in Group 1(61.4%) as well as Group 2 (72.1%) were term babies (≥ 37 weeks). Similar results were also obtained in a study by **Mohanapriya and Srivastava.**[53] Majority of the participants (72.9%) Group 1 and 84.9% of Group 2) had gestational diabetes. Group 1 had diabetes for a median duration of 6.5 months while in case of the Group 2, it was 7 months.

Most of the babies in both the groups were delivered by LSCS (60% of Group 1 and 68.6% of Group 2) followed by vaginal delivery in 45.7% of the Group 1 cases and 54.3% of the Group 2 cases. This was in concordance with the results obtained in a few other studies[39,54,55] but was in contrast to a study by **Mohanapriya and Srivastava** [53] who observed majority of their participants having a vaginal delivery, however, more females with GDM delivered via LSCS. It is plausible that the evolving patterns in the handling of GDM pregnancies may be obscured by the increasing incidence of caesarean sections in emerging nations, which bear similarities to the group under study [56]. Pregnancies with diabetes are frequently considered to be at higher risk because of the possibility of problems such as fetal macrosomia or more difficulties achieving vaginal birth. As a result, medical professionals may decide to perform a caesarean section as a safety precaution for the mother and the child.

Majority of the neonates in both the groups were AGA (80.0% of Group 1 and 70.9% of Group 2) followed by SGA (14.3% of Group 1 and 26.7% of Group 2). Similar results were seen in a study by **Satishkumar and colleagues**[39] A Swedish study examined the features of 1547 newborns born to mothers with GDM between 1998 and 2007, comparing them to over 83,000 infants delivered to mothers without GDM. The above study found that the incidence of LGA was 26% among neonates born to GDM mothers. [57]

Hypoglycemia was noted in 54% while hypocalcemia was observed in 43% of newborns in a study by **Anjum and Yashodha**.[5] According to data from the literature, neonates born to women with GDM (25.1%) and type 1 DM (58.3%) are more prone to develop hypoglycemia compared to normal newborn. [58] **Mahmood and Kayes**[59], **Ranade et al**[60], and **Mountain**[61] reported the incidence of hypoglycemia to be 23%, 50%, and 55.2% in their respective studies. According to **Mannan et al**.[62], only 8% of newborns had hypoglycemia. Consequently, it is understood that if a newborn has hypoglycemia, it is often suggestive that the mother's blood sugar levels might not have been properly regulated. [51] However, in our study, there was no statistically significant association noted between neonatal hypoglycemia and neonatal hypocalcemia with maternal glycemic control.

All newborn with blood glucose levels lesser than 40 mg/dl, irrespective of gestational age or symptoms, is considered to have hypoglycemia. In the fetal period there is hyperplasia of the islets of Langerhans leading to hyperinsulinism. After delivery when the maternal supply of glucose is cut off by clamping the cord, the extra insulin in the baby causes neonatal hypoglycemia .[5]

Overall, 19.9% of the participants in the current study had hypocalcemia (24.3% in Group 1 and 16.3% in Group 2). [63] The incidence of hypocalcemia was reported by **Merchant et al.**[64], **Opara et al**. [65], **Ahmed et al.**[66], **Ranade et al**[60], and **Mountain**[61] to be 60%, 23.4%, 22.8%, 14%, and 25-50%, respectively.

Neonatal RDS and CHD were significantly higher in Group 1 as compared to Group 2 in the current study. A significant association was noticed in the current study between Neonatal RDS and CHD with poor maternal glycemic control. The odds of a baby delivered to a mothers with poor glycemic control, having RDS is 2.37 times more than those delivered to mothers with good glycemic control. Similarly, the odds of a baby born to a mother with poor glycemic control, having CHD is 2.07 times more than those born to a mother with good glycemic control." Respiratory distress was seen in 19.3% of cases in a study by **Satishkumar and colleagues**[39]. In research by **Prakash et al.,**[67] respiratory distress syndrome was the most common consequence (11%) that was identified. In **Crowther et al**.'s [68] experiment, respiratory distress syndrome was noted more frequently in the intervention arm.

An extensive analysis conducted by Piper in 2002 underscored the importance of managing glucose levels, demonstrating that infants born to diabetic mothers who maintained adequate glycemic control showed lung maturation comparable to that of the general population.[69] Saturated phosphatidylcholine shortage in the lungs and amniotic fluid is the result of elevated insulin levels, which hinder the absorption of choline into lecithin and cause RDS. [5]

Despite good maternal glycemic control, prenatal hyperglycemia and hyperinsulinism results in an increased risk of cardiac hypertrophy in this newborn. The interventricular septum is caused due to fluctuations in these values, and in extreme situations, a varying degree of left ventricular outflow blockage has been reported.The finding from various studies were inconsistent, but significant changes were noted with poor glycemic control, indicated by HbA1c levels exceeding 6.5%. [31]

Overall incidence of CHD in the current study was 30.1%. **Øyen et al**.[70] discovered that mothers with pre-gestational diabetes mellitus had a four-fold increase in the offspring

developing CHDs over a cohort study involving two million births spanning 34 year. The risk of gestational diabetes mellitus was only marginally elevated. Improper diabetes management and insufficient prenatal care were significant confounding factors that raised the risk. Pregnancies with inadequate glycaemic management in the first trimester were reported to be more likely to have fetal cardiac disease by **Todorova et al**.[71]. Research has shown that women with adequate glycemic control at the time of conception and during the early stages of pregnancy, have a significantly lower chances of having a newborn with cardiac malformations than women with inadequate glycemic control.[72]

Two cases out of 156 had a congenital anomaly. One case (neonate of a mother with good glycemic control) had an abnormal swelling over the lumbar region which was later diagnosed as left lumbar hernia while another case (neonate of a mother with poor glycemic control) was diagnosed with bilateral paramedian cleft lip. One neonate from the cases group expired due to cardiorespiratory failure with pneumopericardium /severe RDS/ extreme preterm/ probable sepsis. **Satishkumar and colleagues**[39] in their research observed two neonatal deaths. They did not report any neonates with congenital anomalies in their study. Other research has revealed the percentage of congenital malformations to be 2%, 3.8%, and 5.7% .[51,59,73]

The relatively high rates of unfavourable outcomes for mothers and newborns make GDM a serious issue even with the advancements in diagnosis, monitoring, and treatment that have occurred recently. The results seen in our study highlight the value of a collaborative approach between neonatology and feto-maternal medicine. This balance is crucial in determining the optimal timing for delivery, especially in complicated pregnancies where the risks of preterm birth must be considered alongside the risks of continuing the pregnancy. Such coordination can help in making informed decisions that aim to maximize the better health outcomes for both the mother and the child.

Universal screening for GDM is indeed a critical step in ensuring early diagnosis and management of the condition. The survey by **Mahalakshmi et al**. reflects a positive shift towards widespread adoption of universal screening practices. With **85%** of healthcare professionals such as physicians, obstetricians, and diabetologists already implementing this approach, it indicates a significant move toward better maternal and fetal health outcomes.[74] Early diagnosis through universal screening can lead to timely interventions, which may include medical nutrition therapy and insulin therapy if needed, thereby potentially reducing the complications associated with GDM among the mother and the child.

LIMITATIONS

- Furthermore, our study lacks data on the dietary habits and lifestyle of the mother during the various stages of pregnancy making it difficult to investigate the underlying mechanisms. Convenience sample and the comparatively small size of the impacted group in comparison to the previously described studies represent two further significant limitations.
- Larger prospective studies with long-term outcome evaluations are crucial for gaining a better understanding of the causes of newborn morbidities linked to GDM. Longitudinal research that follows infants over an extended period can reveal patterns and risk factors that may not be apparent in shorter studies.
- The categorization of the neonates according to good and poor maternal glycemic control was based on the levels of Hb1Ac recorded in the third trimester. Further research involving multiple values of Hb1Ac recorded throughout the pregnancy from conception till delivery and their average is recommended as it will reduce the chances of error.

CONCLUSION

CONCLUSION

In this hospital-based prospective cohort study of 156 neonates born to diabetic mothers at RLJH, Tamaka, revealed that neonates born to diabetic mothers are prone to morbidities like hypoglycemia, hypocalcemia, respiratory distress, and congenital heart disease including ASD, VSD, and PDA. Maternal glycemic control significantly impacts these neonatal outcomes. While there was no statistically significant association found between maternal glycemic control and neonatal hypoglycemia or hypocalcemia, there was a notable correlation with neonatal respiratory distress and congenital heart disease, both of which were more prevalent in neonates born to mothers with poor glycemic control.

Hence this study emphasizes the importance of maintaining good glycemic control during pregnancy to improve neonatal health outcomes.

SUMMARY

SUMMARY

A hospital-based prospective cohort study was done on 156 neonates born to diabetic mothers during one and half year period at RLJH, Tamaka. Among the 156 neonates,70 were born to mothers with poor glycemic control (Group 1) and 86 to mothers with good glycemic control (Group 2). This study was conducted to determine and compare morbidity and mortality patterns of neonates born to mothers with good or poor glycemic control in diabetic mothers.

- A slight female predominance (51.4%) was seen in Group 1 while in Group 2, males (61.6%) were predominant.
- Most of the neonates (50% of Group 1 and 50% of Group 2) belonged to the birth weight category 2.5-3.5 kg.
- Majority of the neonates (64.3% Group 1 and 54.7% Group 2) were born to multigravida mothers.
- Term babies (≥ 37 weeks) comprised about 61.4% among Group 1 and 72.1% among the Group 2.
- Babies delivered by LSCS predominated by 60% in Group 1 and 68.6% in Group 2 followed by vaginal delivery in 45.7% of Group 1 and 54.3% of Group 2.
- Most of the neonates in both groups were AGA (80.0% of Group 1 and 70.9% of Group 2) followed by SGA (14.3% Group 1 and 26.7% Group 2)
- Neonatal hypoglycemia was noted in 14.7% neonates and neonatal hypocalcemia in 19.9% neonates.
- Out of 23 neonates with hypoglycemia, majority 56% belonged to Group 1 and 44% belonged to Group 2. While among 31 neonates with hypocalcemia, majority 54.8%

belong to Group 1 and 45.2% belong to Group 2. But there was no statistical significance noted between maternal glycemic control and these neonatal outcomes.

- There were 28.8% neonates with respiratory distress and 30.1% neonates with congenital heart disease observed in this study.
- Majority of the neonates about 60% with respiratory distress were from Group 1, while majority of neonates about 57.4% with CHD belonged to Group 1. Significant association is present between neonatal respiratory distress and congenital heart disease with maternal glycemic control.
- Among the neonates with congenital heart disease, some neonates presented with more than one 2D echo findings- 44 neonates had ASD, 8 neonates had VSD and 8 neonates had PDA.
- In this study, only 2 neonates (1.2%) had congenital anomalies, one with bilateral complete cleft lip and cleft palate belonging to group 1 and one with left lumbar hernia belonging to group 2.
- Mortality among the neonates in this study was 0.7% due to pneumopericardium belonging to Group 1.

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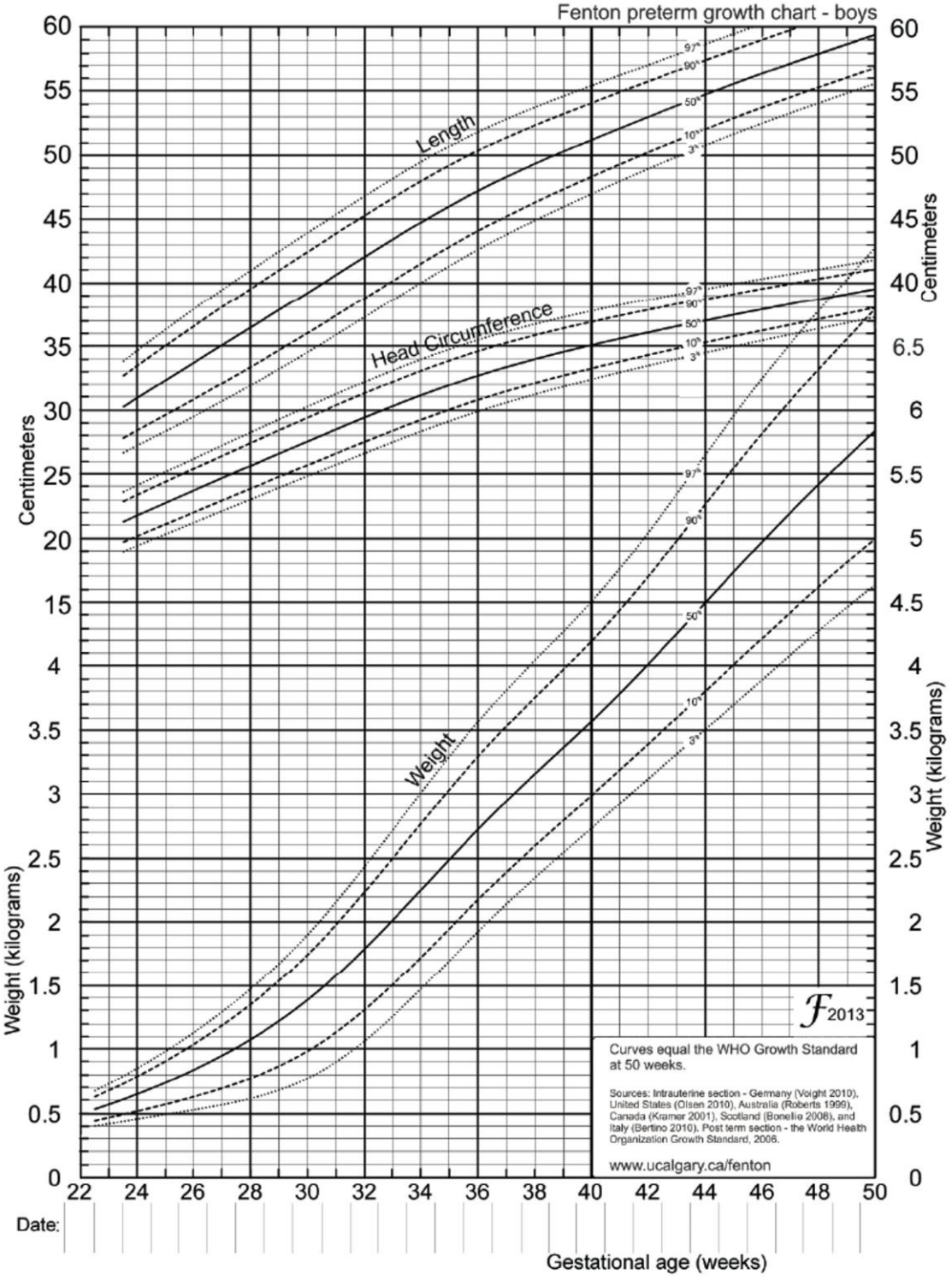
ANNEXURE

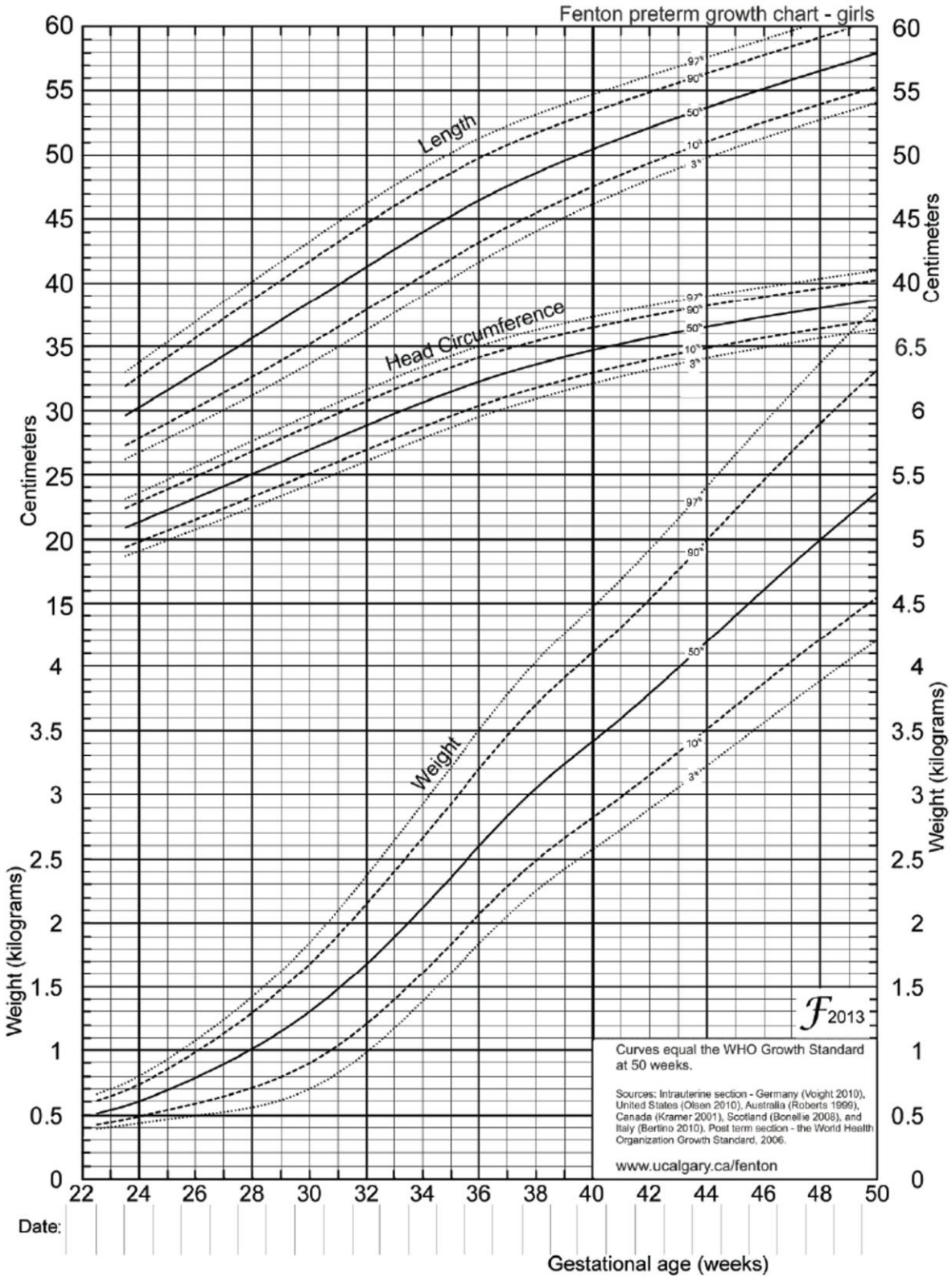
ANNEXURE 1

FENTON CHART-BOYS



ANNEXURE 1

FENTON CHART-GIRLS



PATIENT INFORMATION SHEET

"Outcome of Neonates Born To Mothers With Diabetes Mellitus In A Tertiary Care Centre In Kolar-A Prospective Cohort Study"

Principal investigator: DR.KARTHIK KANANGI /DR. SUDHA REDDY V.R.

I Dr KARTHIK KANANGI, Post graduate student in Department at Sri Devraj Urs Medical College, will be conducting a study titled

"Study of Outcome Of Neonates Born To Mothers With Diabetes Mellitus In A Tertiary Care Centre In Kolar-A Prospective Cohort Study, for my dissertation under the guidance of DR. SUDHA REDDY V.R., Professor of Department of Paediatrics. The participants of this study include all neonates born to diabetic mother ,who are admitting to NICU .The participants of this study i.e. neonates will be undergoing relevant investigations such as CBC, serum electrolytes, serum urea creatinine, serum total and direct bilirubin, serial blood glucose levels as and when required .

All the data will be kept confidential and will be used only for research purpose by this institution. You are free to provide consent for the participation of your child in this study. You can also withdraw your child from the study at any point of time without giving any reasons whatsoever. Your refusal to participate will not prejudice you to any present or future care at this institution.

Name and Signature of the Investigator

Date :

ರೋಗಿಯಮಾಹಿತಿಹಾಳೆ

"ಕೋಲಾರದ ತೃತೀಯ ಆರೈಕೆ ಕೇಂದ್ರದಲ್ಲಿ ಮಧುಮೇಹ ಮೆಲ್ಲಿಟಸ್ ಹೊಂದಿರುವ ತಾಯಂದಿರಿಗೆ ಜನಿಸಿದ ನವಜಾತ ಶಿಶುಗಳ ಫಲಿತಾಂಶ-ಒಂದು ನಿರೀಕ್ಷಿತ ಸಮಂಜಸ ಅಧ್ಯಯನ"

ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿ: ಡಾ.ಕಾರ್ತಿಕ್ ಕಾನಂಗಿ / ಡಾ. ಸುಧಾ ರೆಡ್ಡಿ ವಿ.ಆರ್.

ಶ್ರೀ ದೇವರಾಜ್ ಅರ್ಸ್ ಮೆಡಿಕಲ್ ಕಾಲೇಜಿನಲ್ಲಿ ವಿಭಾಗದಲ್ಲಿ ಸ್ನಾತಕೋತ್ತರ ವಿದ್ಯಾರ್ಥಿಯಾಗಿರುವ ಡಾ ಕಾರ್ತಿಕ್ ಕಾನಂಗಿ ಅವರು ಅಧ್ಯಯನವನ್ನು ನಡೆಸಲಿದ್ದಾರೆ.

"ಡಯಾಬಿಟಿಸ್ ಮೆಲ್ಲಿಟಸ್ ಹೊಂದಿರುವ ತಾಯಂದಿರಿಗೆ ಜನಿಸಿದ ನವಜಾತ ಶಿಶುಗಳ ಫಲಿತಾಂಶದ ಅಧ್ಯಯನ ಕೋಲಾರದ ತೃತೀಯ ಆರೈಕೆ ಕೇಂದ್ರದಲ್ಲಿ-ಒಂದು ನಿರೀಕ್ಷಿತ ಸಮಂಜಸ ಅಧ್ಯಯನ, ಡಿಆರ್ ಅವರ ಮಾರ್ಗದರ್ಶನದಲ್ಲಿ ನನ್ನ ಪ್ರಬಂಧಕ್ಕಾಗಿ. ಸುಧಾ ರೆಡ್ಡಿ ವಿ.ಆರ್., ಮಕ್ಕಳ ವಿಭಾಗದ ಪ್ರಾಧ್ಯಾಪಕರು. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಿದವರಲ್ಲಿ ಮಧುಮೇಹಿ ತಾಯಿಗೆ ಜನಿಸಿದ ಎಲ್ಲಾ ನವಜಾತ ಶಿಶುಗಳು ಸೇರಿದ್ದಾರೆ, ಅವರು NICU ಗೆ ಸೇರಿದ್ದಾರೆ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವವರು ಅಂದರೆ ನವಜಾತ ಶಿಶುಗಳು CBC, ಸೀರಮ್ ಎಲೆಕ್ಟ್ರೋಲೈಟ್‌ಗಳು, ಸೀರಮ್ ಯೂರಿಯಾ ಕ್ರಿಯೇಟಿನೈನ್, ಸೀರಮ್ ಒಟ್ಟು ಮತ್ತು ನೇರ ಬೈಲಿರುಬಿನ್, ಸರಣಿ ರಕ್ತ ಮುಂತಾದ ಸಂಬಂಧಿತ ತನಿಖೆಗಳಿಗೆ ಒಳಗಾಗುತ್ತಾರೆ. ಗ್ಲೂಕೋಸ್ ಮಟ್ಟಗಳು ಮತ್ತು ಅಗತ್ಯವಿದ್ದಾಗ.

ಎಲ್ಲಾ ಡೇಟಾವನ್ನು ಗೌಪ್ಯವಾಗಿ ಇರಿಸಲಾಗುತ್ತದೆ ಮತ್ತು ಈ ಸಂಸ್ಥೆಯಿಂದ ಸಂಶೋಧನಾ ಉದ್ದೇಶಕ್ಕಾಗಿ ಮಾತ್ರ ಬಳಸಲಾಗುತ್ತದೆ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನಿಮ್ಮ ಮಗುವಿನ ಭಾಗವಹಿಸುವಿಕೆಗೆ ಒಪ್ಪಿಗೆ ನೀಡಲು ನೀವು ಸ್ವತಂತ್ರರಾಗಿದ್ದೀರಿ. ಯಾವುದೇ ಕಾರಣಗಳನ್ನು ನೀಡದೆ ನೀವು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ನಿಮ್ಮ ಮಗುವನ್ನು ಅಧ್ಯಯನದಿಂದ ಹಿಂಪಡೆಯಬಹುದು. ಭಾಗವಹಿಸಲು ನಿಮ್ಮ ನಿರಾಕರಣೆಯು ಈ ಸಂಸ್ಥೆಯಲ್ಲಿ ಯಾವುದೇ ಪ್ರಸ್ತುತ ಅಥವಾ ಭವಿಷ್ಯದ ಕಾಳಜಿಗೆ ನಿಮ್ಮನ್ನು ಪೂರ್ವಾಗ್ರಹ ಮಾಡುವುದಿಲ್ಲ.

ತನಿಖಾಧಿಕಾರಿಯ ಹೆಸರು ಮತ್ತು ಸಹಿ

ದಿನಾಂಕ:

INFORMED CONSENT FORM

Date:

I, Mr/Mrs _____ ,have been explained in my own vernacular language that my child will be included in A Prospective Observational Study-**Study of Outcome Of Neonates Born To Mothers With Diabetes Mellitus In A Tertiary Care Centre In Kolar-A Prospective Cohort Study**hereby I give my valid written informed consent without any force or prejudice for recording the observations of haematological and clinical parameters . The nature and risks involved have been explained to me, to my satisfaction. I have been explained in detail about the study being conducted. I have read the patient information sheet and I have had the opportunity to ask any question. Any question that I have asked, have been answered to my satisfaction. I provide consent voluntarily to allow my child as a participant in this research. I hereby give consent to provide history, undergo physical examination, undergo investigations and provide its results and documents etc to the doctor / institute etc. All the data may be published or used for any academic purpose.

_____ _____

(Name of Pt. Attendant) (Signature/Thumb impression)

_____ _____

(Witness)

(Signature & Name of Reseacher/Doctor)

ಮಾಹಿತಿನೀಡಿದಒಪ್ಪಿಗೆನಮೂನೆ

ದಿನಾಂಕ

ನಾನು, ಶ್ರೀ/ಶ್ರೀಮತಿ _____, ಕೋಲಾರದ ತೃತೀಯ ಆರೈಕೆ ಕೇಂದ್ರದಲ್ಲಿ ಡಯಾಬಿಟಿಸ್ ಮೆಲ್ಲಿಟಸ್ ಹೊಂದಿರುವ ತಾಯಂದಿರಿಗೆ ಜನಿಸಿದ ನವಜಾತ ಶಿಶುಗಳ ನಿರೀಕ್ಷಿತ ವೀಕ್ಷಣಾ ಅಧ್ಯಯನ-ಅಧ್ಯಯನದಲ್ಲಿ ನನ್ನ ಮಗುವನ್ನು ಸೇರಿಸಲಾಗುವುದು ಎಂದು ನನ್ನದೇ ಆದ ಸ್ಥಳೀಯ ಭಾಷೆಯಲ್ಲಿ ವಿವರಿಸಲಾಗಿದೆ-ಒಂದು ನಿರೀಕ್ಷಿತ ಸಹಕಾರಿ ಅಧ್ಯಯನ ಹೆಮಟೊಲಾಜಿಕಲ್ ಮತ್ತು ಕ್ಲಿನಿಕಲ್ ನಿಯತಾಂಕಗಳ ಅವಲೋಕನಗಳನ್ನು ದಾಖಲಿಸಲು ನಾನು ಯಾವುದೇ ಬಲ ಅಥವಾ ಪೂರ್ವಾಗ್ರಹವಿಲ್ಲದೆ ನನ್ನ ಮಾನ್ಯವಾದ ಲಿಖಿತ ತಿಳುವಳಿಕೆಯನ್ನು ನೀಡುತ್ತೇನೆ. ಒಳಗೊಂಡಿರುವ ಸ್ವಭಾವ ಮತ್ತು ಅಪಾಯಗಳನ್ನು ನನಗೆ ವಿವರಿಸಲಾಗಿದೆ, ನನ್ನ ತೃಪ್ತಿ. ನಡೆಸುತ್ತಿರುವ ಅಧ್ಯಯನದ ಬಗ್ಗೆ ನನಗೆ ವಿವರವಾಗಿ ವಿವರಿಸಲಾಗಿದೆ. ನಾನು ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆಯನ್ನು ಓದಿದ್ದೇನೆ ಮತ್ತು ಯಾವುದೇ ಪ್ರಶ್ನೆಯನ್ನು ಕೇಳಲು ನನಗೆ ಅವಕಾಶವಿದೆ. ನಾನು ಕೇಳಿದ ಯಾವುದೇ ಪ್ರಶ್ನೆಗೆ ನನ್ನ ತೃಪ್ತಿಗೆ ಉತ್ತರಿಸಲಾಗಿದೆ. ನನ್ನ ಮಗುವನ್ನು ಈ ಸಂಶೋಧನೆಯಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವಂತೆ ಅನುಮತಿಸಲು ನಾನು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಒಪ್ಪಿಗೆಯನ್ನು ನೀಡುತ್ತೇನೆ. ನಾನು ಇತಿಹಾಸವನ್ನು ಒದಗಿಸಲು, ದೈಹಿಕ ಪರೀಕ್ಷೆಗೆ ಒಳಗಾಗಲು, ತನಿಖೆಗೆ ಒಳಗಾಗಲು ಮತ್ತು ಅದರ ಫಲಿತಾಂಶಗಳು ಮತ್ತು ದಾಖಲೆಗಳನ್ನು ಇತ್ಯಾದಿಗಳನ್ನು ವೈದ್ಯರು / ಸಂಸ್ಥೆ ಇತ್ಯಾದಿಗಳಿಗೆ ಒದಗಿಸಲು ನಾನು ಈ ಮೂಲಕ ಒಪ್ಪಿಗೆ ನೀಡುತ್ತೇನೆ. ಎಲ್ಲಾ ಡೇಟಾವನ್ನು ಪ್ರಕಟಿಸಬಹುದು ಅಥವಾ ಯಾವುದೇ ಶೈಕ್ಷಣಿಕ ಉದ್ದೇಶಕ್ಕಾಗಿ ಬಳಸಬಹುದು.

_____ _____

(ಪಂ.ನ ಸಹಿ ಮತ್ತು ಹೆಸರು. ಪರಿಚಾರಕ) (ಸಹಿ/ಹೆಬ್ಬೆರಳಿನ ಗುರುತು)

_____ _____

(ಸಾಕ್ಷಿ) (ಸಹಿ ಮತ್ತು ಸಂಶೋಧಕ/ವೈದ್ಯರ ಹೆಸರು)

PROFORMA

MATERNAL DETAILS

Name of Mother :

Age :

UHID no :

Obstetric score:

Antenatal Anomaly scan:

Type of diabetes: Gestational / Pregestational

Onset /duration of diabetes:

Maternal HbA1C levels _____ at _____ gestation (weeks)

FETAL DETAILS

Date and time of delivery:

UHID no:

Gestational age :

Mode of delivery:

Weight:

Gender of baby:

APGAR SCORE:

Vitals- PR: RR: Spo2:

If respiratory distress;

Downes/ Silvermann Anderson score:

Provisional diagnosis:

Condition of Baby at admission:

Blood glucose level

At birth-

2hr- 24hr-

6hr- 48hr-

12hr- 72hr-

Biochemical parameters

CBC

Hb- PCV- WBC- Platelets: ABG:

Serum calcium -

Serum magnesium-

2D ECHO –

Chest Xray

Neurosongram

Congenital anomalies (if any):

Remark

MASTER CHART

Sr no	Age of mother	UHID (mother)	Obstretic score	Antenatal Scan	Type of Diabetes	Duration of maternal Diabetes	Maternal HbA1c value	Group	UHID(Baby)	Gestational AGE	Mode of delivery	Birth Weight (kg)	Gender	PR	RR	SPO2	Respiratory Distress Score (D/SA)	Diagnosis	GRBS -at birth	2hours	6 HOURS	12 hours	24 hours	48 hours	72 hours	Hb	PCV	WBC	Platelet	Calcium	Magnesium	Chest xray (if required)	2D ECHO	NEUROSONOGRAM	Congenital anomalies
1	29	145545	Primigravida	normal	gestational	7 months of ammenorrhea	< 6.5	control	145692	31wk+3day	vaginal delivery	2.14	male	160	64	94 at RA	NIL	PT(31WK+3 DAYS)/AGA/MALE/RDS 2 HMD/NNHB/IDM/ACHD-3MM ASD	58	78	74	82	90	78	68	16.2	51.1	9.92	287	9.2	3		ACHD- 3MM ASD	-	NIL
2	27	145667	G2P1L1	normal	gestational	8 months of ammenorrhea	<6.5	control	145866	34wk + 1 day	forceps assisted vaginal delivery	2.54	female	158	64	98% on CPAP support	Silvermann Anderson score-4	LPT(34 WK+ 1 DAY)/ AGA/ FEMALE/ RDS SEC HMD/ IDM/PROBABLE SEPSIS//ACHD- 5MM ASD 2MM PDA	52	72	90	102	97	74	92	16.8	50	25	265	8.9	1.8	-	ACHD- 5MM ASD, 2MM PDA	-	NIL
3	26	146231	Primigravida	normal	gestational	6 months of ammenorrhea	>6.5	case	145360	34wk	forceps assisted vaginal delivery	2.4	female	156	64	98% on CPAP support	Silvermann Anderson score-3	LPT(34wk)/AGA/FEMALE/RDS SEC HMD/IDM/PROBABLE SEPSIS/ACHD- 4MM ASD 2MM PDA	46	60	74	84	85	102	74	17.4	52	25	291	8.6	2.1	-	ACHD- 4MM ASD 2MM PDA	-	NIL
4	28	145180	G2P1L1	normal	overt	2 years (on insulin)	<6.5	control	145233	36wk	vaginal delivery	2.48	male	158	66	96% on CPAP support	Silvermann Anderson score-5	LPT(36wk)/AGA/MALE/IDM/HYPOCALCEMIA/ACHD-4MM ASD PDA,PAH (40 MM HG)	48	64	78	88	103	98	92	18.4	54.6	11	278	7.6	1.8	-	ACHD- 4MM ASD 3MM PDA,PAH (40 MM HG)	-	NIL
5	25	146465	G2P1L1	normal	gestational	8 months of ammenorrhea	<6.5	case	146465	36wk+ 3 day	LSCS	2.42	female	154	58	94% on RA	NIL	LPT(36 WK+ 3 DAY/ AGA/FEMALE/IDM/ACHD- 2MM ASD 3MM VSD	56	70	73	74	84	78	90	18.3	51.7	9.18	162	7.7	1.8	-	ACHD- 2MM ASD 3 MM MUSCULAR VSD , DILATED RA RV ,MILD TR MILD PAH (40 MM HG)	-	NIL
6	34	145611	G3P2L1D1	normal	overt	1 year (on oral metformin)	>6.5	case	147689	37wk +4 days	LSCS	2.82	female	142	58	94% on RA	NIL	T/AGA/FEMALE/IDM/NNHB/ACHD- 5MM ASD 3MM PDA	52	53	68	84	89	90	92	14.9	51.7	11.2	265	9.4	1.2	-	ACHD- 5MM ASD, 3MM PDA, MILD TR,MILD PAH (30 MM HG)	-	NIL
7	26	148762	Primigravida	normal	gestational	7 months of ammenorrhea	>6.5	case	148765	39wk+ 5 days	LSCS	2.96	female	148	68	96% on 2litre NP	Downe score -2	T/AGA/FEMALE/IDM/RDS SEC TO TTNB /HYPOTHYROID MOTHER/ACHD- 5MM ASD ,DILATED RA RV ,MODERATE PAH(50 MM HG)	46	66	65	72	96	97	80	18	53.7	52.8	140	7.3	1.8	Perihilar Interstitial marking	5MM ASD DILATED RA RV, MODERATE TR, MODERATE PAH(PASP- 50 MM HG)	-	NIL
8	29	177005	G2A1	normal	overt	3 months of ammenorhea	>6.5	case	177005	38 wks + 3 days	LSCS	3.28	male	150	52	94% on RA	NIL	TERM/AGA/MALE/IDM/SMALL PFO	58	90	85	96	81	97	92	19	53	22.4	217	9.6	1.9	-	ACHD-2MM SMALL PFO	-	NIL
9	23	197391	Primigravida	normal	gestational	7 months of ammenorrhea	<6.5	control	197391	38 wks + 6 days	LSCS	3.34	male	140	66	96% on 2litre NP	Downe score -3	TERM/AGA/MALE/IDM/RD SEC TO MAS/HYPOTHYROID MOTHER/ SMALL PFO	62	50	97	102	59	91	90	18.8	57.6	18.5	205	9	2.1	B/L Homogenous opacities	SMALL PFO, MILD PR, MILD TR, MILD PAH (PASP=30 MM HG)	B/L PERIVENTRICULAR FLARING	NIL
11	28	202905	G2P1L1	normal	overt	2 years (on oral metformin)	>6.5	case	143278	34 wks + 6 days	LSCS	2.36	male	158	64	96% on 2litre NP	Silvermann Anderson score-2	LPT(34wk + 6)/AGA/MALE/IDM/DCDA TWIN 2/RD SEC HMD	68	80	46	92	86	84	98	18.8	54.7	12	241	4.7	1.6	B/L Homogenous opacities	SMALL PFO , TRIVIAL TR ,PAH (20 MM HG)	-	NIL
10	28	202905	G2P1L1	normal	overt	2 years (on oral metformin)	>6.5	case	143280	34 wks + 6 days	LSCS	2.54	male	160	64	96% on 2litre NP	Silvermann Anderson score-3	LPT(34wk + 6)/AGA/MALE/IDM/DCDA TWIN 1/RD SEC HMD	48	78	81	80	89	90	67	19.3	57.2	11.5	176	5.8	1.8	B/L Homogenous opacities	4MM ASD-NORMAL	-	NIL
12	29	197397	G5P1L1A3	normal	gestational	8 months of ammenorrhea	>6.5	case	206861	36 wk +5 days	LSCS	2.56	male	142	52	94% on RA	NIL	LPT(36wk +5 days)/AGA/MALE/HYPOTHYROID MOTHER/IDM	52	42	68	76	88	89	78	22.2	63.3	10.3	157	6.2	1.8	-	SMALL PFO ,PAH (35 MM HG)	-	NIL
13	28	205991	G6A5	normal	gestational	8 months of ammenorrhea	> 6.5	case	211185	36 wk +4 days	LSCS	1.46	female	148	48	96% on RA	NIL	LPT(36wk +5 days)/SGA/FEMALE/HYPOTHYROID MOTHER/UTEROPLACENTAL INSUFFICIENCY/IDM	86	80	92	86	82	80	108	19.9	52.9	10.2	213	213	3	-	3MM ASD, 2MM PDA	-	NIL
14	28	145479	G3P1L1A1	normal	gestational	8 months of ammenorrhea	<6.5	control	145479	38 wks	vaginal delivery	2.6	male	158	62	96% on RA	NIL	TERM/SGA/MALE/RH NEGATIVE MOTHER/IDM	52	78	82	86	99	102	88	16.3	49.1	11.4	207	8.7	2.7	-	2MM ASD, MILD TR	-	NIL
15	27	216556	Primigravida	normal	overt	2 months of amenorhhea	<6.5	control	216768	38 wk+4 days	LSCS	3.04	male	146	42	94 % on RA	NIL	TERM/AGA/MALE/IDM/HYPOTHYROID MOTHER	78	55	75	72	84	80	71		58	14		8.4	1.8		3MM ASD	-	NIL
16	26	156760	G3P2L2	normal	gestational	8 months of ammenorrhea	<6.5	control	160367	36wk+ 5 days	LSCS	3.46	female	146	54	96 % on RA	NIL	LPT(36wk+ 5 day)/AGA/FEMALE/IDM	72	68	78	45	86	84	70	15.7	45.3	15.7	239	9.7	1.2	-	4MM ASD, TINY PDA,MILD TR,MODERATE PAH(45 MM HG)	-	NIL
17	26	163166	G3P1L1A1	NA	gestational	6 months of ammenorrhea	<6.5	control	163189	36wk+6 days	forceps assisted vaginal delivery	2	male	150	64	94% on 2litre NP	Silvermann Anderson score-4	LPT(36wk+ 6 day)/SGA/MALE/IDM/PERINATAL DEPRESSION	48	94	170	122	103	80	96	17.9	56	10.2	119	9.2	2	B/L Paracardiac Infiltrates	3MM ASD, 2MM VSD ,MILD TR,MODERATE PAH(40MM HG)	fluid collection in b/l parietal region,crossing cranial suturs-hematoma. Another subperiosteal collection-cephalohematoma	NIL
18	27	176424	G2P1L1	normal	gestational	6 months of ammenorrhea	>6.5	case	176575	38 wk	LSCS	3.46	male	152	52	94% on RA	NIL	TERM/AGA/MALE/IDM	52	79	83	101	70	98	90	20.3	57.7	17	200	10.1	1.2	-	4 MM ASD, MILD TR, MILD PAH (30 MM HG)	-	NIL
19	26	212736	G2P1L1	normal	gestational	8 months of ammenorrhea	>6.5	case	213161	41 wk+2 days	vaccuum assisted vaginal delivery	3.12	male	142	52	94% on RA	NIL	TERM/SGA/MALE/IDM	78	80	82	97	90	88	79	16.3	53.6	14.4	188	6.4	1.7	-	3MM ASD, 3MM PDA, MODERATE TR, MODERATE PAH (48MM HG)	-	NIL
20	28	157074	Primigravida	normal	overt	3 months of ammenorhea	>6.5	case	158227	36 wk +2 days	vaginal delivery	2.55	male	146	56	98% on RA	NIL	LPT(36 wk+2 days)/AGA/MALE/IDM/PPROM/PROBABLE SEPSIS	61	68	88	72	90	102	92	17.2	58	14	297	9.9	2	-	4MM ASD, TINY PDA,MILD TR,MODERATE PAH(45 MM HG)	-	NIL
21	22	139947	G4P1L1A2	normal	gestational	8 months of ammenorrhea	<6.5	control	163448	38 wk +1 day	vaginal delivery	2.16	female	148	56	92% on RA	NIL	TERM/SGA/FEMALE/IDM	54	83	80	78	66	72	90	21	46	9.9	112	9.9	1.6	-	SMALL PFO- NORMAL	-	NIL
22	20	158912	Primigravida	normal	overt	3 years on inj H.ACTRAPID	<6.5	control	167829	37 wk +6 days	LSCS	2.4	female	144	44	94% on RA	NIL	TERM/SGA/FEMALE/IDM	100	89	84	76	92	63	89	12.1	48.8	15.3	322	10.8	2.1	-	3MM ASD, TRIVIAL TR (PASP - 25 MM HG)	-	NIL
23	25	237223	G4P2L1A1D1	NA	gestational	6 months of ammenorrhea	>6.5	case	237253	39 wk + 6 days	LSCS	4.32	female	146	48	96% on RA	NIL	TERM/LGA/FEMALE/IDM	38	58	57	58	65	72	89	17.2	54.2	14.6	256	9.1	1.8	-	SMALL PFO, TINY PDA- NORMAL	-	NIL
24	29	236778	G2P1L1	normal	gestational	8 months of ammenorrhea	<6.5	control	236778	38 wk + 4 days	LSCS	4.12	male	146	56	95% on RA	NIL	TERM/AGA/MALE/IDM	33	65	113	83	84	72	92	16.3	48.3	12.9	220	10.6	-	-	SMALL PFO, TINY PDA, MODERATE PAH (48 MM HG)	-	NIL
25	31	236769	G3P1L1A1	normal	gestational	7 months of ammenorrhea	>6.5	case	236807	37 wk +5 days	vaginal delivery	3.06	female	134	54	95% on RA	NIL	TERM/AGA/FEMALE/IDM	68	72	96	75	79	92	111	17.8	51	15.9	211	8.2	-	-	SMALL PFO, TINY PDA, MILD TR, MILD PAH (35MM HG)	-	NIL
26	21	170079	Primigravida	NA	gestational	7 months of ammenorrhea	<6.5	control	170082	38 wk + 5 days	LSCS	2.7	male	142	52	96% on RA	NIL	TERM/AGA/MALE/IDM/LUMBAR HERNIA	86	80	97	81	74	87	89	17	47.7	17.2	277	9.8	2.2	-	SMALL PFO - NORMAL	-	Abnormal swelling over lumbar region-LEFT LUMBAR HERNIA
27	23	171272	G2P1L1	normal	gestational	8 months of ammenorrhea	>6.5	case	177018	39 wk + 5 days	vaccuum assisted vaginal delivery	3.16	female	150	64	94% on 2litre NP	Downe score -3	TERM/AGA/FEMALE/IDM/PERINATAL DEPRESSION/RD SEC TO TTNB	46	52	84	90	92	88	90	20.4	51.8	15.6	173	9.8	1.8	-	4MM ASD	-	NIL
28	30	129470	G3P2LO	normal	gestational	7 months of ammenorrhea	<6.5	control	156976	39 wk + 5 days	vaginal delivery	2.9	female	152	52	94% on RA	NIL	TERM/AGA/FEMALE/IDM	62	54	74	68	104	94	84	19.7	51.6	13.4	301	9.2	1.7	-	SMALL PFO- NORMAL	-	NIL
29	23	197229	Primigravida	normal	gestational	7 months of ammenorrhea	>6.5	case	197306	38 wk + 4 days	LSCS	2.94	male	150	52	92% on RA	NIL	TERM/AGA/MALE/IDM	56	60	84	71	102	94	88	23.1	62.6	15.6	215	8.2	1.7	-	SMALL PFO- NORMAL	-	NIL
30	32	124899	G3P1L1A1	normal	gestational	8 months of ammenorrhea	<6.5	control	140481	38 wk + 2 days	LSCS	2.92	male	146	50	94% on RA	NIL	TERM/AGA/MALE/IDM	59	50	65	84	72	98	104	21.7	62.6	10.63	263	7.2	1.6	-	SMALL PFO- NORMAL	-	NIL
31	25	155141	G4P2L2A1	normal	gestational	7 months of ammenorrhea	<6.5	control	155154	37 wk	vaginal delivery	2.86	male	148	48	94% on RA	NIL	TERM/AGA/MALE/IDM	49	52	78	92	86	88	102	16.2	39.2	2.8	256	9.6	2.1	-	SMALL PFO- NORMAL	-	NIL
32	30	212641	G2P1L1	normal	gestational	7 months of ammenorrhea	<6.5	control	202913	38 wk +1 day	LSCS	3.1	male	148	46	94% on RA	NIL	TERM/AGA/MALE/IDM	54	58	68	92	89	76	90	16.3	45.6	15.8	306	9.1	1.6	-	-	-	NIL
33	27	212401	G2P1L1	normal	gestational	7 months of ammenorrhea	<6.5	control	181464	34 wks + 4 days	LSCS	1.3	female	146	56	96% on RA	NIL	LPT(34wk + 4day)/SGA/FEMALE/ASYMMETRICAL IUGR/IDM	48	58	66	81	78	89	66	21.7	63.4	10.5	223	9.6	2.1	-	SMALL PFO- NORMAL	-	NIL
34	24	180115	Primigravida	normal	gestational	8 months of ammenorrhea	<6.5	control	180325	36 wk +6 days	LSCS	2.06	male	142	50	94% on RA	NIL	LPT(36wk+ 6 day)/SGA/MALE/HYPOTHYROID MOTHER/IDM	68	84	58	62	102	92	89	20.5	57.3	12.6	152	8.6	1.6	-	SMALL PFO, 2MM PDA	-	NIL
35	24	163646	G2P1L1	normal	gestational	7 months of ammenorrhea	<6.5	control	163861	33wk + 6 days	LSCS	1.9	female	154	68	98% on 2litre NP	Silvermann Anderson score-3	PT(33WK+6 DAYS)/AGA/FEMALE/RDS 2 HMD/IDM	48	74	96	89	128	88	89	16.2	46.6	4.99	215	9.3	1.2	B/L Homogenous opacities	TRIVIAL TR (PASP-18 MM HG)	-	NIL
36	28	195397	G3P1L1A1	normal	gestational	8 months of ammenorrhea	<6.5	control	195479	38 wk	vaginal delivery	2.6	male	158	62	96% on RA	NIL	TERM/SGA/MALE/RH NEGATIVE MOTHER/IDM	86	78	82	86	99	72	80	16.3	49.1	11.4	207	8.9	1.6	-	-	-	NIL
37	22	222687	G2A1	normal	overt	4 years on inj. H. ACTRAPID	<6.5	control	238550	43 wk+1 days	LSCS	3.44	female	148	52	94% on RA	NIL	POST TERM/AGA/FEMALE/IDM	68	54	61	80	78	102	88	18.6	55.3	25.2	165	9.1	2.1	-	SMALL PFO, TINY PDA- NORMAL	-	NIL
38	29	118708	G2P1L1	normal	gestational	6 months of ammenorrhea	>6.5	case	139313	38 wk + 3 days	vaginal delivery	3.27	male	144	68	96% on CPAP support	Downe score -5	TERM/AGA/MALE/IDM/RD SEC TO TTNB/ IDM/SMALL PFO	46	52	64	80	72	94	98	15.5	44.5	33	229	7.9	2	Perihilar Interstitial marking	SMALL PFO,MILD TR,MILD PAH (PASP= 35MM HG)	-	NIL
39	38	225588	Primigravida	normal	overt	2 months of ammenorhea	<6.5	control	238146	39 wk + 3 days	LSCS	2.82	male	150	54	95% on RA	NIL	TERM/AGA/MALE/IDM	56	49	76	67	86	89	101	21.6	50.3	11.9	176	7.4	2.6	-	SMALL PFO- NORMAL	-	NIL
40	34	210864	Primigravida	normal	gestational	6 months of ammenorrhea	<6.5	control	163345	39 wk + 2 days	LSCS	2.84	male	140	52	96% on RA	NIL	TERM/AGA/MALE/IDM	58	81	88	82	94	88	78	16.7	48.4	20.7	396	9.9	1.2	-	3MM ASD, MILD TR, MILD PAH (PASP= 25 MM HG)	-	NIL
41	26	233526	G4P2L2A1	normal	gestational	6 months of ammenorrhea	<6.5	control	233926	34 wk+ 2 days	LSCS	1.9	male	149	48	95% on RA	NIL	LPT(34wk + 2 day)/AGA/MALE/IDM	49	61	72	68	89	72	98	17.1	48.3	9.1	268	10.1	1.9	-	SMALL PFO- NORMAL	-	NIL
42	26	233526	G4P2L2A1	normal	gestational	6 months of ammenorrhea	<6.5	control	233927	34 wk+ 2 days	LSCS	1.74	male	148	52	95% on RA	NIL	LPT(34wk + 2 day)/AGA/MALE/IDM	64	52	68	101	86	78	88	18.1	50	9.8	230	9.7	2	-	SMALL PFO- NORMAL	-	NIL
43	24	179784	G3P2L1D1	normal	gestational	8 months of ammenorrhea	<6.5	control	238146	39 wk+ 2 days	LSCS	2.8	male	148	50	94% on RA	NIL	TERM/AGA/MALE/IDM	48	58	72	66	81	96	90	18.1	58	10.2	216	9.4	2.1	-	SMALL PFO, TINY PDA- NORMAL	-	NIL
44	27	241252	G2P1L1	normal	gestational	7 months of ammenorrhea	<6.5	control	238550	43 wk + 2 days	LSCS	3.44	female	142	48	94% on RA	NIL	POST TERM/AGA/FEMALE/IDM	54	51	69	94	74	68	101	18.6	55.3	25.3	165	10.4	2.7	-	5MM ASD,2 MM PDA, MILD TR,MILD PAH	-	NIL
45	24	242186	Primigravida	normal	gestational	6 months of ammenorrhea	<6.5	control	237252	38 wk + 5 days	LSCS	4.32	female	150	48	94% on RA	NIL	TERM/AGA/FEMALE/IDM	56	76	81	99	71	68	84	18.3	47.6	16.5	311	6.8	2.2	-	SMALL PFO, TINY PDA- NORMAL	-	NIL
46	26	242158	G2P1L1	normal	gestational	8 months of ammenorrhea	<6.5	control	238468	39 wk + 2 days	LSCS	2.44	male	148	50	94% on RA	NIL	TERM/SGA/MALE/IDM	42	60	58	48	69	76	82	19.8	46.5	16.6	138	8.6	2.1	-	SMALL PFO- NORMAL	-	NIL
47	23	245261	Primigravida	normal	gestational	8 months of ammenorrhea	<6.5	control	245443	39 wk + 4 days	LSCS	4.18	male	150	56	94% on RA	NIL	TERM/LGA/MALE/IDM	74	56	68	72	96	102	86	17.6	38.5	10.6	362	8.6	3.1	-	SMALL PFO, 2MM PDA	-	NIL
48	24	267899	Primigravida	normal	gestational	7 months of ammenorrhea	<6.5	control	268122	38 wk + 2 days	LSCS	3.6	female	150	50	94% on RA	NIL	TERM/AGA/FEMALE/IDM	79	68	62	98	106	86	88	16.8	39.2	11.8	386	8.8	2.8	-	SMALL PFO- NORMAL	-	NIL
49	29	241783	G2P1L1	normal	gestational	7 months of ammenorrhea	<6.5	control	236807	37 wk + 5 days	LSCS	3.06	female	152	48	92% on RA	NIL	TERM/AGA/FEMALE/IDM	68	54	78	106	96	88	77	18.2	48.2	9.8	256	9.1	1.8	-	SMALL PFO- NORMAL	-	NIL
50	28	240888	Primigravida	normal	gestational	6 months of ammenorrhea	<6.5	control	240908	39 wk + 5 days	LSCS	3.14	male	154	48	92% on RA	NIL	TERM/AGA/MALE/IDM/PROM	78	62	98	111	102	85	88	18.2	48.2	7.8	281	8.9	2.2	-	SMALL PFO- NORMAL	-	NIL
51	32	242856	G2P1L1	normal	gestational	7 months of ammenorrhea	<6.5	control	241115	37 wk	LSCS	3.54	male	156	66	94% on HFNC	Downe score -3	TERM/AGA/MALE/RDS/IDM	61	46	79	84	92	108	98	17.8	44.6	12.8	315	8.6	1.8	-	SMALL PFO, 3MM ASD	-	NIL

Sr no	Age of mother	UHID (mother)	Obstetric score	Antenatal Scan	Type of Diabetes	Duration of maternal Dabetes	Maternal HbA1c value	Group	UHID(Baby)	Gestational AGE	Mode of delivery	Birth Weight (kg)	Gender	PR	RR	SPO2	Respiratory Distress Score (D/SA)	Diagnosis	GRBS -at birth	2hours	6 HOURS	12 hours	24 hours	48 hours	72 hours	Hb	PCV	WBC	Platelet	Calcium	Magnesium	Chest xray (if required)	2D ECHO	NEUROSONOGRAM	Congenital anomalies
52	26	258108	G2P1L1	normal	gestational	7 months of ammenorrhea	<6.5	control	258360	39 wk	vaginal delivery	2.14	female	148	52	92% on RA	NIL	TERM/SGA/FEMALE/IDM/ RH NEGATIVE MOTHER	67	82	72	68	111	90	88	17.8	45.8	8.9	291	6.9	2.1	-	SMALL PFO- NORMAL	-	NIL
53	**29**	**290589**	**G3A2**	**B/L PARAMEDIAN CLEFT LIP**	**overt**	**2 years (on oral metformin)**	**>6.5**	**case**	**290720**	**37 wk + 5 days**	**vaginal delivery**	**3**	**female**	**146**	**48**	**94% on RA**	**NIL**	**TERM/AGA/FEMALE/IDM/BILATERAL COMPLETE CLEFT LIP WITH COMPLETE CELFT PALATE**	**68**	**84**	**90**	**74**	**108**	**111**	**89**	**20.3**	**58.5**	**14.5**	**259**	**8.3**	**2.1**	**-**	**SMALL PFO, MILD TR (PASP 25 MM HG)**	**-**	**NIL**
54	26	276231	Primigravida	normal	gestational	6 months of ammenorrhea	<6.5	control	276866	35 wk + 4 days	LSCS	2.74	female	148	50	94% on RA	NIL	LPT/AGA/FEMALE/DCDA TWIN 1/IDM	62	82	70	106	94	101	81	18.2	43.3	9.1	285	8.8	1.9	-	2MM ASD, 2MM PDA	-	NIL
55	26	276231	Primigravida	normal	gestational	6 months of ammenorrhea	<6.5	control	276867	35 wk + 4 days	LSCS	1.74	female	150	54	94% on HFNC	Silvermann Anderson score-4	LPT/AGA/FEMALE/DCDA TWIN 2/IDM	82	56	71	66	59	81	92	12.9	39	6.9	251	6.7	1.2	B/L GROUND GLASS OPACITIES	2MM ASD 2MM PDA	-	NIL
56	28	186432	G2P1L1	normal	gestational	6 months of ammenorrhea	<6.5	control	186806	39 wk + 5 days	vaccuum assisted vaginal delivery	2.8	male	150	48	94% on 2litre NP	Silvermann Anderson score-2	TERM/AGA/MALE/IDM/TTNB	56	76	90	78	101	92	88	16.2	38.2	5.8	236	9.1	1.6	-	2MM ASD , 2 MM VSD	-	NIL
57	29	279960	G2P1L1	normal	gestational	6 months of ammenorrhea	>6.5	case	280080	40 wk + 3 days	LSCS	3.92	male	160	62	94% on HFNC	Downe score -5	TERM/AGA/MALE/IDM/RD SEC TO MAS	56	72	68	66	84	102	76	16.2	40.1	6.4	231	6.8	1.8	B/L Paracardiac Infiltrates	4MM ASD, 2MM VSD	-	NIL
58	29	288799	G2P1L1	normal	gestational	6 months of ammenorrhea	<6.5	control	289066	35 wk +2 days	vaginal delivery	2.5	female	152	50	94% on 2litre NP	Silvermann Anderson score-4	LPT(35WK+2 DAYS)/AGA/FEMALE/RDS 2 HMD/IDM	49	62	78	90	77	69	102	14.2	38.6	8.2	265	8.1	2.1	B/L Paracardiac Infiltrates	-	-	NIL
59	32	186304	G2P1L1	normal	gestational	7 months of ammenorrhea	<6.5	control	186616	38 wk + 4 days	LSCS	2.92	male	148	50	94% on RA	NIL	TERM/AGA/MALE/IDM	82	47	62	78	102	94	112	18.2	42	5.5	290	8.9	1.8	-	2MM ASD, SMALL PFO	-	NIL
60	26	252465	G2P1L1	normal	overt	3 year (on oral metformin)	<6.5	control	252730	36 wk + 5 days	vaccuum assisted vaginal delivery	1.4	male	156	50	94% on RA	NIL	LPT(36 WK+6 DAYS)/SGA/MALE/IDM	46	52	68	80	101	88	92	18.1	40.2	10.6	301	6.9	1.6	-	3MM ASD, 2MM PDA	-	NIL
61	29	256410	G2P1L1	normal	gestational	7 months of ammenorrhea	<6.5	control	256682	38wk + 4 days	LSCS	2.82	male	150	48	94% on RA	NIL	TERM/AGA/MALE/IDM	51	89	56	81	102	89	111	16.4	50.2	8.1	292	8.9	1.9	-	-	-	NIL
62	26	294101	Primigravida	normal	overt	1 year (on oral metformin)	>6.5	case	294182	36 wk	vaginal delivery	2.02	male	152	58	94% on RA	NIL	LPT(36 WK)/AGA/MALE/IDM	47	66	109	136	111	101	81	19.2	52.4	6.2	228	6.7	1.8	-	3MM ASD, 2MM PDA	-	NIL
63	29	267825	G2P1L1	normal	overt	3 months of ammenorrhea	<6.5	control	267824	38wk + 6 days	LSCS	2.16	female	150	48	94% on RA	NIL	TERM/SGA/MALE/IDM	70	59	84	92	102	111	89	16.2	45.1	8.9	229	8.9	0.9	-	3MM ASD, 2MM VSD	-	NIL
64	24	193896	Primigravida	normal	gestational	7 months of ammenorrhea	<6.5	control	193992	39 wk + 2 days	LSCS	2.86	female	148	48	94% on RA	NIL	TERM/AGA/FEMALE/IDM	79	68	96	126	118	90	111	14.8	42	7.9	219	7.1	1.1	-	-	-	NIL
65	32	197125	G2P1L1	normal	gestational	6 months of ammenorrhea	>6.5	case	197229	38wk + 6 days	LSCS	2.64	male	150	48	93% on RA	NIL	TERM/AGA/MALE/IDM	81	72	89	106	96	111	126	15.2	42.6	7.2	218	8.6	1.1	-	-	-	NIL
66	24	224042	Primigravida	normal	gestational	7 months of ammenorrhea	<6.5	control	224121	37 wk + 5 days	LSCS	2.4	male	148	48	92 % on RA	NIL	LPT(37 WK +5 DAYS)/AGA/MALE/IDM	54	44	78	94	108	111	98	16.1	45	8.9	289	8.9	1.3	-	mild TFO , mild TR	-	NIL
67	26	227492	G2P1L1	normal	gestational	7 months of ammenorrhea	<6.5	control	227680	38 wk + 3 days	LSCS	2.98	male	159	68	94% on HFNC	Downe score -4	TERM/AGA/MALE/RDS SEC TO MAS/IDM	62	89	102	90	109	92	89	17.2	40.1	9.1	218	8.9	1.1	-	2MM ASD, 3MM PDA	-	NIL
68	24	280618	Primigravida	normal	gestational	6 months of ammenorrhea	<6.5	control	280757	37 wk	LSCS	2.76	male	148	52	94% on RA	NIL	TERM/AGA/MALE/IDM	60	52	69	96	81	102	118	18.1	46.4	4.9	228	5.9	2.1	-	SMALL PFO, MILD TR	-	NIL
69	28	284625	G2P1L1	normal	overt	1 year (on oral metformin)	<6.5	control	284793	36 wk + 2 days	vaginal delivery	1.74	female	152	52	94% on RA	NIL	LPT(36 WK+2 DAYS)/SGA/FEMALE/IDM	71	69	84	90	120	101	98	14.8	42.1	8.6	285	8.8	1.1	-	SMALL PFO	-	NIL
70	29	262891	G2P1L1	normal	overt	4 months of ammenorrhea	>6.5	case	262934	31 wk + 6 days	vaginal delivery	1.62	female	155	64	94% on CPAP	Silvermann Anderson score-4	PT(31 WK+6 DAYS)/AGA/FEMALE/RDS 2 HMD/IDM	66	78	59	82	109	112	122	16.8	45.4	10.8	220	7.8	1.1	B/L GROUND GLASS OPACITIES	3MM ASD, 2MM PDA	-	NIL
71	26	260018	Primigravida	normal	gestational	7 months of ammenorrhea	>6.5	case	260154	38 wk + 4 days	LSCS	2.9	male	158	66	94% on HFNC	Downe score -5	TERM/AGA/MALE/RD SEC TO TTNB/IDM	59	78	90	81	120	101	92	18.4	46.2	7.8	219	8.6	0.9	B/L Paracardiac Infiltrates	3MM ASD, SMALL PFO	-	NIL
72	26	256026	Primigravida	normal	gestational	7 months of ammenorrhea	<6.5	control	256174	37 wk + 4 days	vaginal delivery	2.08	female	148	50	94% on RA	NIL	TERM/SGA/FEMALE/IDM	72	82	101	90	86	106	91	14.3	40.8	9.6	215	8.2	1.3	-	SMALL PFO, MILD TR	-	NIL
73	29	289002	G2P1L1	normal	gestational	6 months of ammenorrhea	<6.5	control	289066	35 wk +2 days	LSCS	2.5	female	150	50	94% on RA	NIL	LPT(35 WK+2 DAYS)/AGA/FEMALE/IDM	84	102	98	98	84	104	92	14.8	43.2	8.7	281	8.6	1.2	-	2MM ASD, 2MM VSD	-	NIL
74	29	272467	G2P1L1	normal	gestational	7 months of ammenorrhea	>6.5	case	272603	39 wk + 4 days	LSCS	2.68	male	156	54	94% on RA	NIL	TERM/AGA/MALE/IDM	69	78	80	102	102	98	89	15.8	44.8	9.6	285	8.2	1.8	-	2MM ASD, SMALL PFO	-	NIL
75	29	227592	Primigravida	normal	overt	2 year (on oral metformin)	>6.5	case	227631	40 wk	LSCS	3.6	female	150	46	94% on RA	NIL	TERM/AGA/FEMALE/IDM	56	66	49	55	76	87	91	14.8	44.9	10.1	299	11.4	-	-	4MM ASD, TINY PDA,MILD TR,MODERATE PAH(45 MM HG)	-	NIL
76	26	256723	G2P1L1	normal	gestational	6 months of ammenorrhea	<6.5	control	256783	38wk + 3 days	vaginal delivery	2.36	female	148	48	94% on RA	NIL	TERM/SGA/FEMALE/IDM	58	69	81	99	102	97	110	16.2	46.1	8.8	324	10.8	1.6	-	SMALL PFO	-	NIL
77	28	288601	G2P1L1	normal	gestational	6 months of ammenorrhea	<6.5	control	288638	38wk + 5 days	LSCS	3.1	male	152	50	94% on RA	NIL	TERM/AGA/MALE/IDM	71	62	84	96	104	98	111	17.2	46.9	6.3	265	8.9	-	-	-	-	NIL
78	29	285682	G2P1L1	normal	gestational	7 months of ammenorrhea	<6.5	control	285703	39 wk + 2 days	LSCS	2.76	male	146	48	94% on RA	NIL	TERM/SGA/MALE/IDM	72	68	79	102	94	112	108	16.1	44.6	9.6	289	8.4	-	-	NORMAL	-	NIL
79	28	284186	G2P1L1	normal	gestational	7 months of ammenorrhea	<6.5	control	284227	39 wk	LSCS	3.14	male	158	64	94% on RA	Downe score - 2	TERM/AGA/MALE/RD SEC TO TTNB/IDM	56	47	69	88	96	111	98	16.8	45.2	9.1	291	8.1	-	B/L Paracardiac Infiltrates	NORMAL	-	NIL
80	25	278597	Primigravida	normal	overt	3 months of ammenorrhea	<6.5	control	278674	34 WK	vaginal delivery	2.02	female	152	56	94% on RA	NIL	LPT(36 WK)/AGA/FEMALE/IDM	64	52	79	92	110	102	96	15.1	44.6	8.2	297	8.6	-	-	SMALL PFO	-	NIL
81	23	277169	Primigravida	normal	gestational	7 months of ammenorrhea	>6.5	case	277273	39 wk + 2 days	vaccuum assisted vaginal delivery	2.9	male	158	66	94% on 2litre NP	Downe score - 2	TERM/AGA/MALE/RD SEC TO TTNB/IDM	79	63	84	102	116	108	121	14.2	42.8	6.5	315	6.7	1.8	B/L Paracardiac Infiltrates	SMALL PFO	-	NIL
82	30	298198	G4P1L1A2	normal	overt	8 months on inj H.ACTRAPID	<6.5	control	298369	38wk + 5 days	LSCS	3.12	male	152	52	94% on RA	NIL	TERM/AGA/MALE/IDM	68	79	94	121	111	98	114	16.2	46.4	8.9	199	8.9	1.2	-	2MM ASD, SMALL PFO	-	NIL
83	32	297249	G6P1L1A4	normal	gestational	7 months of ammenorrhea	<6.5	control	297315	38wk + 4 days	LSCS	2.52	male	146	48	94% on RA	NIL	TERM/AGA/MALE/IDM	64	52	78	91	112	108	126	18.1	52.3	9.2	256	9.1	1.8	-	-	-	NIL
84	26	289084	Primigravida	normal	gestational	6 months of ammenorrhea	<6.5	control	289146	39 wk	vaginal delivery	2.03	male	150	50	94% on RA	NIL	TERM/SGA/MALE/IDM	46	60	78	101	112	94	102	17.4	47.1	6.8	281	8.2	-	-	-	-	NIL
85	25	296246	Primigravida	normal	gestational	6 months of ammenorrhea	>6.5	case	296572	36 wk	vaginal delivery	2.66	female	156	50	94% on RA	NIL	LPT(36 WK)/AGA/FEMALE/IDM	48	68	89	72	98	86	101	16.9	48.4	11.2	298	6.7	1.9	-	SMALL PFO	-	NIL
86	29	299065	G2P1L1	normal	gestational	7 months of ammenorrhea	>6.5	case	299113	40 wk	LSCS	3.6	female	150	64	94% on 2litre NP	Downe score -4	TERM/AGA/FEMALE/RDS SEC TO MAS/IDM	68	62	74	86	92	84	78	16.8	44.8	9.8	245	9.1	2.2	-	MILD PAH (30 MM HG)	-	NIL
87	24	289078	Primigravida	normal	overt	1 year (on oral metformin)	<6.5	control	289151	39 wk + 2 days	vaginal delivery	2.88	female	148	52	94% on RA	NIL	TERM/AGA/FEMALE/IDM	62	78	86	92	80	77	96	18.4	53.1	8.9	256	6.7	2.1	-	-	-	NIL
88	28	298863	G2P1L1	normal	gestational	6 months of ammenorrhea	<6.5	control	298919	36 wk	LSCS	3.18	male	152	50	94% on RA	NIL	TERM/AGA/MALE/IDM	61	72	58	84	89	98	101	16.8	44.2	5.9	281	9.1	2.2	-	NORMAL	-	NIL
89	23	290586	Primigravida	normal	gestational	7 months of ammenorrhea	<6.5	control	290720	37 wk + 1 day	LSCS	3	female	148	48	94% on RA	NIL	TERM/AGA/FEMALE/IDM	81	70	68	84	79	88	92	16.2	45.8	8.2	224	8.9	1.9	-	NORMAL	-	NIL
90	19	291192	Primigravida	normal	gestational	4 months of ammenorrhea	<6.5	control	291273	31 wk	vaginal delivery	1.3	male	152	62	94% on CPAP	Silvermann Anderson score-4	PT(31 WK)/AGA/MALE/RDS 2 HMD/IDM	79	63	42	86	98	90	88	18.2	55.1	10.2	214	6.9	1.8	B/L GROUND GLASS OPACITIES	SMALL PFO	B/L PERIVENTRICULAR FLARING, ventriculomegaly	NIL
91	18	294883	Primigravida	normal	overt	6 months on inj H.ACTRAPID	>6.5	case	294995	31 wk + 6 days	LSCS	1.64	male	148	64	94% on CPAP	Silvermann Anderson score-4	PT(31 WK + 6 DAYS)/AGA/MALE/RDS 2 HMD/IDM	52	64	78	82	101	90	108	19	52.5	10.2	178	8.2	2.8	B/L GROUND GLASS OPACITIES	2MM SUBAORTIC VSD, SMALL PFO, SEVERE SAH (60 MM HG)	-	NIL
92	22	307081	G3P2L2	normal	gestational	6 months on inj H.ACTRAPID	<6.5	control	307132	35 wk +3 days	LSCS	2.58	male	158	62	94% on 2litre NP	Silvermann Anderson score-2	LPT(35 WK + 3 DAYS)/AGA/MALE/IDM/ RDS SEC TTNB	85	84	97	78	96	98	78	23.3	66.5	7.8	239	6.8	2.1	B/L Paracardiac Infiltrates	4 MM ASD, MILD PAH (30 MM HG)	-	NIL
93	32	308396	G4P3L3	normal	gestational	7 months of ammenorrhea	<6.5	control	308439	37 wk + 6 day	LSCS	3.76	male	148	52	94% on RA	NIL	TERM/AGA/MALE/IDM	25	65	74	90	100	81	111	21	58.3	5.9	278	8.9	2.8	-	3MM PDA,3MM PFO, MILD TR (PASP 25 MM HG)	-	NIL
94	32	309086	G3P2L1D1	normal	gestational	3 months of ammenorhea (INJ MIXTARD)	>6.5	case	309148	37 wk + 6 day	LSCS	3.26	male	150	50	94% on RA	NIL	TERM/AGA/MALE/IDM	45	51	55	82	88	102	92	20	57.3	9.8	218	9.6	2.1	-	3MM ASD, 2MM PDA	-	NIL
95	31	299489	G2P1L1	normal	overt	2 year (on oral metformin)	<6.5	control	299593	38 wk	vaginal delivery	2.5	female	148	56	94% on RA	NIL	TERM/AGA/FEMALE/IDM	60	72	79	91	88	102	111	12.9	40.6	6.9	265	9.2	1.9	-	NORMAL	-	NIL
96	24	300179	Primigravida	normal	gestational	6 months of ammenorrhea	<6.5	control	300179	35 wk	vaginal delivery	1.92	male	152	60	94% on 2litre NP	Silvermann Anderson score-2	LPT(35 WK)/AGA/MALE/ RDS SEC HMD/ IDM	54	62	74	88	101	96	112	17.4	45.9	9.6	228	8.4	2.2	-	NORMAL	-	NIL
97	28	300385	G2P1L1	normal	gestational	6 months of ammenorrhea	<6.5	control	300401	38 wk+2 days	LSCS	2.94	female	156	64	96% on HFNC	Downe score -4	TERM/SGA/FEMALE/RDS SEC TO MAS/IDM	72	76	84	96	94	86	102	16.2	45.8	9.8	225	9.1	2.2	B/L Paracardiac Infiltrates	3MM ASD, SMALL PFO	-	NIL
98	26	302189	Primigravida	normal	overt	1 year (on oral metformin)	<6.5	control	302227	35 wk	vaginal delivery	1.88	male	148	58	94% on 2litre NP	Silvermann Anderson score-3	LPT(35 WK)/SGA/MALE/ RDS SEC HMD/ IDM	54	60	77	79	100	96	106	17.1	49.2	8.7	315	6.7	1.9	B/L GROUND GLASS OPACITIES	SMALL PFO	-	NIL
99	26	304986	Primigravida	normal	gestational	7 months of ammenorrhea	<6.5	control	305007	38 wk	vaginal delivery	2.7	male	148	48	94% on RA	NIL	TERM/SGA/FEMALE/IDM	62	69	78	84	101	98	111	16.2	46.1	9.2	263	8.8	1.9	-	NORMAL	-	NIL
100	29	304866	G2P1L1	normal	gestational	6 months of ammenorrhea	>6.5	case	304989	38 wk + 2 days	LSCS	3.68	female	158	66	94% on HFNC	Downe score -5	TERM/SGA/FEMALE/RDS SEC TO MAS/IDM	52	62	68	79	92	102	98	14.2	45.6	9.1	205	8.1	1.2	B/L GROUND GLASS OPACITIES	3MM ASD, SMALL PFO	-	NIL
101	30	305186	G2P1L1	normal	gestational	6 months of ammenorrhea	>6.5	case	305239	39 wk + 2 days	vaccuum assisted vaginal delivery	3.32	male	148	62	94% on 2litre NP	Downe score -3	TERM/AGA/MALE/RD SEC TO TTNB/IDM	72	80	96	88	101	96	108	17.1	49.6	6.8	212	7.2	1.8	B/L Paracardiac Infiltrates	NORMAL	-	NIL
102	25	307192	Primigravida	normal	gestational	1 year (on oral metformin)	<6.5	control	307276	37 wk	vaginal delivery	1.82	male	150	48	94% on RA	NIL	TERM/SGA/MALE/IDM	72	68	79	87	110	121	98	17.2	46.8	7.8	251	9.1	2.2	-	SMALL PFO	-	NIL
103	24	307872	Primigravida	normal	gestational	6 months of ammenorrhea	<6.5	control	307985	37 wk	LSCS	1.84	male	152	60	94% on 2litre NP	Downe score - 2	TERM/SGA/MALE/RD SEC TO TTNB/IDM	62	74	92	111	88	79	91	15.2	44.9	8.7	289	8.9	1.9	-	NORMAL	-	NIL
104	30	317663	G2P1L1	normal	overt	1 year (on oral metformin)	<6.5	control	317719	41 wk	vaginal delivery	2.14	male	152	48	94% on RA	NIL	POST TERM/SGA/MALE/IDM	59	79	82	102	110	118	98	16.3	44.5	10.3	286	8.9	1.1	-	NORMAL	-	NIL
105	24	311682	Primigravida	normal	gestational	6 months of ammenorrhea	<6.5	control	311725	39 wk + 5 days	LSCS	3.44	male	152	54	94% on RA	NIL	TERM/AGA/MALE/IDM	62	78	80	96	111	118	16102	17.2	46.2	8.9	218	9.2	1.2	-	MILD TR,MILD PAH	-	NIL
106	29	315872	G2P1L1	normal	gestational	7 months of ammenorrhea	<6.5	control	315943	38 wk + 4 days	LSCS	3.16	male	154	52	94% on RA	NIL	TERM/AGA/MALE/IDM	71	61	78	92	101	109	111	17.4	48.6	11.4	241	8.9	1.6	-	NORMAL	-	NIL
107	25	313969	Primigravida	normal	gestational	7 months of ammenorrhea	<6.5	control	314090	41 wk + 3 days	LSCS	3.36	male	150	48	94% on RA	NIL	TERM/AGA/MALE/IDM	62	54	68	72	96	90	104	18.6	49.1	10.2	281	9.2	1.8	-	NORMAL	-	NIL
108	28	324447	G2P1L1	normal	overt	1 year (on oral metformin)	>6.5	case	324499	39 wk	LSCS	2.74	female	150	50	96% on RA	NIL	TERM/AGA/FEMALE/IDM	58	62	84	90	96	111	101	16.2	48.4	8.8	282	8.9	2.1	-	5MM ASD, MILD TR,MILD PAH (PASP 40MM HG)	-	NIL

Sr no	Age of mother	UHID (mother)	Obstetric score	Antenatal Scan	Type of Diabetes	Duration of maternal Dabetes	Maternal HbA1c value	Group	UHID(Baby)	Gestational AGE	Mode of delivery	Birth Weight (kg)	Gender	PR	RR	SPO2	Respiratory Distress Score (D/SA)	Diagnosis	GRBS -at birth	2hours	6 HOURS	12 hours	24 hours	48 hours	72 hours	Hb	PCV	WBC	Platelet	Calcium	Magnesium	Chest xray (if required)	2D ECHO	NEUROSONOGRAM	Congenital anomalies
109	23	320943	Primigravida	normal	gestational	6 months of ammenorrhea	>6.5	case	320985	39 wk + 3 days	vaginal delivery	3.48	male	147	48	94% on RA	NIL	TERM/AGA/MALE/IDM	59	63	79	91	101	121	98	16.1	49.2	8.5	265	9.8	1.9	-	TINY PDA, SMALL PFO	-	NIL
110	29	325396	G2P1L1	normal	overt	2 year (on oral metformin)	<6.5	control	325440	38wk + 4 day	vaginal delivery	2.74	male	150	52	94% on RA	NIL	TERM/AGA/MALE/IDM	52	60	73	86	101	95	99	19.3	55.1	9.6	229	9.2	1.1	-	NORMAL	-	NIL
111	22	326587	Primigravida	normal	overt	1 year (on oral metformin)	>6.5	case	326642	38wk +2 day	vaginal delivery	1.96	male	152	54	94% on RA	NIL	TERM/SGA/MALE/IDM	60	47	56	71	88	91	97	14.8	45.2	9.7	281	6.8	1.8	-	NORMAL	-	NIL
112	22	318651	G3D1A2L0	normal	overt	3 year (on oral metformin + H.ACTRAPID)	>6.5	case	318698	36wk +3 days	LSCS	3.2	female	148	50	94% on RA	NIL	TERM/AGA/FEMALE/IDM	57	60	63	67	62	76	90	19.5	58.3	15.5	269	9.1	2.3	-	5MM ASD, 2MM MID MUSCULAR VSD, 2MM PDA	-	NIL
113	26	395491	G2P1L1	normal	gestational	6 months of ammenorrhea	>6.5	case	395540	37wk + 2 days	vaginal delivery	2.04	male	156	54	94% on RA	NIL	TERM/SGA/MALE/IDM	54	67	81	90	102	98	103	14.8	45.6	8.9	252	9	1.4	-	NORMAL	-	NIL
114	24	323062	Primigravida	normal	gestational	6 months of ammenorrhea	<6.5	control	323115	35wk + 4 day	vaginal delivery	1.86	female	160	64	94% on CPAP	Silvermann Anderson score-4	LPT(35 WK + 4 DAYS)/AGA/FEMALE/IDM/ RDS 2 HMD	53	60	66	78	96	102	97	18.6	50.2	9.8	291	6.8	2	B/L Homogenous opacities	SMALL PFO	-	NIL
115	24	334014	Primigravida	normal	gestational	5 months of ammenorrhea	<6.5	control	334039	36 wk	LSCS	3.12	female	152	54	94% on RA	NIL	LPT (36WK)/LGA/FEMALE/IDM	56	78	80	82	94	102	96	17.8	50.1	8.9	281	8.6	1.2	-	NORMAL	-	NIL
116	33	349052	G4P3L2D1	normal	gestational	6 months of ammenorrhea	<6.5	control	349098	39 wk + 2 days	vaginal delivery	2.64	male	162	64	94% on 2litre NP	Silvermann Anderson score-4	TERM/AGA/MALE/RD SEC TO TTNB/IDM	57	62	64	68	82	86	99	19.8	56.8	12.5	194	6.7	2.2	B/L Paracardiac Infiltrates	2MM PFO,TINY PDA, MILD PAH (PASP 28MM HG)	-	NIL
117	32	349024	G3P1L1A1	normal	overt	5 years on H.ACTRAPID	>6.5	case	350287	37 wk + 4 days	LSCS	3.48	female	154	56	94% on RA	NIL	TERM/AGA/FEMALE/IDM	58	61	74	80	89	82	99	16.9	51.7	24.9	338	7.9	1.8	-	5MM ASD ,TINY PDA MILD PAH (PASP 40 MM HG)	-	NIL
118	30	335596	G2P1L1	normal	gestational	5 months of ammenorrhea	>6.5	case	335626	39 wk	LSCS	4.64	female	158	54	94% on RA	NIL	TERM/LGA/FEMALE/IDM	58	52	68	74	96	84	102	17.9	50.4	8.01	197	8.6	1.8	-	2MM SMALLPFO, MILD TR, MILD PAH (PASP 30 MM HG)	-	NIL
119	30	351035	Primigravida	normal	gestational	5 months of ammenorrhea	<6.5	control	351064	36 wk + 2 days	LSCS	3.36	female	148	64	94% on 2litre NP	Silvermann Anderson score-3	LPT(36 WK + 2 DAYS)/AGA/FEMALE/IDM/ RDS 2 TTNB	49	51	68	99	81	65	101	16.9	51	14.7	251	5.6	1.9	B/L Paracardiac Infiltrates	3-4 MM PDA, 2MM PFO, MILD TR, MILD PAH (PASP 30 MM HG)	-	NIL
120	27	332319	Primigravida	normal	gestational	6 months of ammenorrhea	<6.5	control	332397	39 wk + 6 days	vaginal delivery	2.28	female	152	58	94% on RA	NIL	TERM/SGA/FEMALE/IDM	60	52	79	91	98	110	94	17.7	52.1	26.8	182	10.4	2	-	NORMAL	-	NIL
121	40	358947	G5P4L3D1	normal	overt	5 years on H.ACTRAPID	>6.5	case	358958	36 wk + 2 days	LSCS	2.2	female	152	64	95% on 2 litre NP	Silvermann Anderson score- 2	LPT(35 WK + 4 DAYS)/AGA/FEMALE/IDM/ RDS 2 TTNB/ PROBABLE SEPSIS	113	45	67	79	66	101	89	17.9	54	8.96	235	7.9	1.9	B/L Paracardiac Infiltrates	TINY PDA, SMALL PFO, MILD TR (PASP = 25 MM HG), LVEF = 60 %	-	NIL
122	26	361873	G3P1L1A1	normal	gestational	3 months of ammenorrhea (oral metformin)	>6.5	case	361882	38 wk + 4 days	vaginal delivery	3.22	female	152	MV support	95% on MV support	Downes score- 6	TERM/AGA/FEMALE/IDM/RESPIRATORY FAILURE SECONDARY TO BIRTH ASPHYXIA	68	98	130	79	53	80	86	16.9	52.3	18.8	141	8.3	1.8		2-3MM PFO,2MM PDA, MILD PAH (PASP 40MM HG), GRADE I TR	-	NIL
123	29	358638	G5P2L1A2	normal	gestational	6 months of ammenorrhea	<6.5	control	358670	34 wk + 6 days	vaginal delivery	1.64	female	152	50	94% on RA	NIL	LPT(34 WK + 6 DAYS)/AGA/FEMALE/IDM	54	66	72	80	79	92	88	17.2	49.7	10.3	282	10.2	-	-	NORMAL	-	NIL
124	20	355253	Primigravida	normal	gestational	5 months of ammenorrhea	<6.5	control	355257	38 wk + 3 days	vaginal delivery	2.78	female	149	48	94% on RA	NIL	TERM/AGA/FEMALE/IDM	53	68	55	72	91	88	102	17.2	50.1	9.8	263	9.6	-	-	NORMAL	-	NIL
125	18	356716	Primigravida	normal	gestational	4 months of ammenorrhea	<6.5	control	356724	36 wk + 3 days	LSCS	1.96	female	148	48	94% on RA	NIL	LPT(36 WK + 3 DAYS)/AGA/FEMALE/IDM	58	62	72	85	102	95	90	18.6	49.2	10.6	281	7.9	2.1	-	NORMAL	-	NIL
126	27	359843	G2P1L1	normal	gestational	3 months of ammenorrhea	<6.5	control	360400	39 wk + 4 days	LSCS	3.8	female	146	52	94% on RA	NIL	TERM/AGA/FEMALE/IDM	30	51	59	63	71	72	78	18.2	56.1	8.39	254	9.6	-	-	3MM ASD,2MM PDA, SEVERE TR,SEVERE PAH (PASP 60MM HG)	-	NIL
127	31	361419	Primigravida	normal	gestational	5 months of ammenorrhea	>6.5	case	361889	36 wk + 4 days	LSCS	3.88	female	146	46	94% on RA	NIL	LPT(36 WK + 3 DAYS)/LGA/FEMALE/IDM	79	34	66	70	68	70	80	19.7	55.8	10.4	251	8.9	-	-	3MM ASD, DILATED RA RV (PASP 38 MM HG)	-	NIL
128	26	3558389	G3P1L1A1	normal	gestational	5 months of ammenorrhea	>6.5	case	366098	36 wk + 6 days	LSCS	3.42	female	142	52	96% on RA	NIL	LPT(36 WK + 6 DAYS)/AGA/FEMALE/IDM	37	48	136	78	101	96	95	20.7	59.4	8.14	143	6.6	2.1	-	4 MM ASD ,TINY PDA, MILD TR,MILD PAH (PASP 30MM HG)	-	NIL
129	30	366781	G3P1A1	normal	gestational	8 months of ammenorrhea	<6.5	control	366813	37 wk + 5 days	LSCS	3.56	male	146	48	94% on RA	NIL	TERM/AGA/MALE/IDM	44	59	74	86	79	97	99	17.3	51.4	17.6	202	9.2	1.6	-		-	NIL
130	33	311272	G3P2L2	normal	overt	3 years on H.ACTRAPID	>6.5	case	371064	35 wk +1 day	vaginal delivery	2.26	male	152	48	94% on RA	NIL	LPT (35 wk + 1 DAY)/AGA/MALE/IDM	55	35	85	76	70	61	68	18.6	46.8	7.9	281	9.2	1.2	-	NORMAL	-	NIL
131	20	370129	Primigravida	normal	gestational	7 months of ammenorrhea	<6.5	control	370133	39 wk + 6 days	vaginal delivery	2.04	female	146	48	94% on RA	NIL	TERM/SGA/FEMALE/IDM	56	61	72	85	84	101	96	20.2	59.3	17.8	221	9.4	1.6	-	NORMAL	-	NIL
132	23	368778	Primigravida	normal	gestational	7 months of ammenorrhea	<6.5	control	369479	38 wk + 2 days	LSCS	2.86	male	146	48	94% on RA	NIL	TERM/SGA/MALE/IDM	54	60	69	82	101	96	84	19.2	47.2	10.1	291	8.9	1.1	-	NORMAL	-	NIL
133	28	367285	G2P1L1	normal	gestational	6 months of ammenorrhea	<6.5	control	368156	38 wk + 4 days	LSCS	2.66	male	168	64	95% on 2 litre NP	NIL	TERM/AGA/MALE/IDM/ RDS 2 TTNB	61	52	68	74	78	80	92	18.2	52.1	17.4	219	6.8	1.8	B/L Paracardiac Infiltrates	NORMAL	-	NIL
134	26	366787	Primigravida	normal	gestational	6 months of ammenorrhea	<6.5	control	373244	39 wk + 2 days	LSCS	2.52	female	142	46	94% on RA	NIL	TERM/SGA/FEMALE/IDM/ RDS 2 TTNB	61	55	74	71	89	98	72	17.1	51.9	8.5	243	8.1	-	-	NORMAL	-	NIL
135	22	372510	Primigravida	normal	gestational	7 months of ammenorrhea	<6.5	control	372510	39 wk + 1 day	LSCS	2.5	male	158	62	95% on 2 litre NP	Downes score- 2	TERM/AGA/MALE/IDM/ RDS 2 TTNB	77	98	82	96	102	101	106	15.3	44.5	8.9	291	9.5	1.1	B/L Paracardiac Infiltrates	NORMAL	-	NIL
136	25	372322	Primigravida	normal	gestational	6 months of ammenorrhea	<6.5	control	379569	38 wk + 4 days	vaginal delivery	2.28	male	150	48	94% on RA	NIL	TERM/SGA/MALE/IDM	59	51	68	75	89	103	92	17.8	50.1	8.6	282	9.2	1.4	-	NORMAL	-	NIL
137	20	375703	Primigravida	normal	overt	1 year (on oral metformin)	<6.5	control	379551	37 wk	LSCS	2.32	female	148	48	94% on RA	NIL	TERM/AGA/FEMALE/IDM	56	61	67	74	82	90	96	18.1	46.2	12.3	256	9.6	1.2	-	NORMAL	-	NIL
138	24	380744	Primigravida	normal	gestational	5 months of ammenorrhea	<6.5	control	381112	39 wk	LSCS	2.08	male	152	48	94% on RA	NIL	TERM/AGA/MALE/IDM	61	72	58	94	88	106	97	19.2	52.8	11.4	265	9.7	1.8	-	NORMAL	-	NIL
139	33	379616	Primigravida	normal	gestational	6 months of ammenorrhea	<6.5	control	379621	39 wk + 2 days	LSCS	2.46	male	146	48	94% on RA	NIL	TERM/AGA/MALE/IDM	62	71	66	72	84	101	92	15.4	44.6	9.6	281	10.1	-	-	NORMAL	-	NIL
140	33	380744	Primigravida	normal	gestational	5 months of ammenorrhea	<6.5	control	381883	41 wk	LSCS	3.54	female	148	46	94% on RA	NIL	TERM/AGA/FEMALE/IDM	53	64	71	80	82	96	99	16.8	46.1	11.2	252	10.2	1.8	-	NORMAL	-	NIL
141	42	382508	G2P1D1	normal	gestational	5 months of ammenorrhea	<6.5	control	383961	38 wk + 4 days	LSCS	2.88	female	146	66	96% on 2 litre NP	Downes score- 2	TERM/AGA/FEMALE/IDM/ RDS 2 TTNB	46	51	60	74	82	96	80	16.7	51.9	10.4	240	10.1	-	-	3MM PDA , 3MM OS ASD, DILATED RA RV MILD TR, MILD PAH (PASP 30 MM HG)	-	NIL
142	25	384181	Primigravida	normal	gestational	5 months of ammenorrhea	<6.5	control	384185	39 wk + 2 days	vaginal delivery	3.1	female	158	64	94 % on HFNC	Downes score- 4	TERM/AGA/FEMALE/RDS SEC TO MAS/IDM	59	62	87	91	81	104	99	16.1	47.2	12.1	252	7.6	1.2	B/L FLUFFLY INFILTRATES	3MM PDA , 4MM ASD, DILATED RA RV MILD TR, MILD PAH (PASP 35 MM HG)	MILD PERIVENTRICULAR FLARING, CAPUT SUCCEDANEUM	NIL
143	**23**	**362291**	**G2P1L1**	**normal**	**gestational**	**2 months of ammenorrhea (oral metformin)**	**>6.5**	**case**	**386724**	**31 wk**	**LSCS**	**1.46**	**female**	**156**	**MV support**	**94% on MV support**	**Silvermann Anderson score-7**	**PT(31 WK)/AGA/FEMALE/RDS 2 HMD/PROBABLE SEPSIS/ PPROM/IDM**	**92**	**51**	**62**	**-**	**-**	**-**	**-**	**14.4**	**42.3**	**19.2**	**212**	**10.1**	**-**	**B/L RETICULOGRANULAR PATTERN, PNEUMOPERICARDIUM**		**-**	**NIL**
144	38	301557	G3P1L2A1	normal	gestational	5 months of ammenorrhea	<6.5	control	390220	37 wk + 2 days	LSCS	3.92	male	146	48	94% on RA	NIL	TERM/LGA/MALE/IDM	56	45	61	77	68	82	96	18.2	46.2	8.9	289	9.2	1.8	-	NORMAL	-	NIL
145	25	390848	G2P1L1	normal	gestational	7 months of ammenorrhea	<6.5	control	391118	39 wk + 1 day	LSCS	3.7	male	160	62	94% on 2 litre NP	Downes score- 2	TERM/AGA/MALE/IDM/ RDS 2 TTNB	62	51	77	81	79	97	104	16.8	45.1	10.4	261	10.2	-	-	NORMAL	-	NIL
146	22	385366	Primigravida	normal	gestational	6 months of ammenorrhea	<6.5	control	370351	40 wk + 2 day	LSCS	3.66	male	152	48	94% on RA	NIL	TERM/AGA/MALE/IDM	71	52	68	79	101	96	117	15.4	45.2	8.9	255	11.1	-	-	NORMAL	-	NIL
147	37	325911	Primigravida	normal	gestational	5 months of ammenorrhea	>6.5	case	394979	38 wk + 5 days	vaginal delivery	2.9	female	152	46	95% on RA	NIL	TERM/AGA/FEMALE/IDM	48	52	61	74	99	86	102	17.8	51.2	6.1	281	10.2	-	-	SMALL PFO	-	NIL
148	19	396808	Primigravida	normal	gestational	7 months of ammenorrhea	<6.5	control	397310	38 wk + 1 day	LSCS	1.82	male	136	56	94% on RA	NIL	TERM/SGA/MALE/IDM	53	60	74	79	84	99	112	18.8	53.3	12.6	165	9.6	-	-	NORMAL	-	NIL
149	22	398592	G2P1L1	normal	gestational	6 months of ammenorrhea	<6.5	control	399667	36 wk	LSCS	2.34	male	140	68	94% on 2 litre NP	Silvermann Anderson score-3	LPT(36 WK)/AGA/MALE/IDM/ RDS 2 TTNB	61	53	59	66	78	102	95	18.7	52	21.7	297	9.2	-	B/L Paracardiac Infiltrates	NORMAL	-	NIL
150	36	398631	G3P1L1A1	normal	gestational	5 months of ammenorrhea	<6.5	control	398636	39 wk + 3 days	LSCS	2.94	female	150	48	94% on RA	NIL	TERM/AGA/FEMALE/IDM	52	59	68	61	79	88	102	18.4	52.1	14.1	197	9.2	-	-	NORMAL	-	NIL
151	27	373726	G4P3L3	normal	gestational	8 months of ammenorrhea	>6.5	case	399498	37wk + 3 days	LSCS	3.64	male	142	48	94% on RA	NIL	TERM/AGA/MALE/IDM	86	54	60	60	59	78	92	16.2	50.1	11.8	281	9.8	-	-	-	-	NIL
152	24	298134	G2A1	normal	gestational	6 months of ammenorrhea	>6.5	case	397237	34 wk + 3 days	LSCS	2.44	female	136	70	97 % on HFNC	Silvermann Anderson score-2	PT(34 WK + 3 DAYS)/AGA/FEMALE/RDS 2 HMD/DCDA TWIN 1/IDM	50	62	92	82	88	106	91	14.7	42.3	13.8	229	9.9	-	B/L Paracardiac Infiltrates	SMALL PFO, PASP (50 MMHG)	NORMAL	NIL
153	24	298134	G2A1	normal	gestational	6 months of ammenorrhea	>6.5	case	397238	34 wk + 3 days	LSCS	1.86	female	140	72	94 % on HFNC	Silvermann Anderson score-2	PT(34 WK + 3 DAYS)/AGA/FEMALE/RDS 2 HMD/DCDA TWIN 2/IDM	62	77	95	90	91	86	96	17	48.5	11.2	137	11.4	-	B/L Paracardiac Infiltrates	SMALL PFO, PASP (30 MMHG)	NORMAL	NIL
154	27	400783	Primigravida	normal	gestational	3 years on H.ACTRAPID	>6.5	case	400795	31 wk	vaginal delivery	1.1	male	140	68	96% on CPAP support	Silvermann Anderson score-3	PT(31 WK)/AGA/MALE/RDS 2 HMD/IDM	86	79	70	79	101	117	88	17	48	8.4	254	7.4	-	B/L Paracardiac Infiltrates	SMALL PFO	NORMAL	SINGLE UMBILICAL ARTERY- USG KUB NORMAL
155	23	400783	Primigravida	normal	gestational	6 months of ammenorrhea	>6.5	case	400285	39 wk + 4 days	LSCS	3.72	male	142	46	94% on RA	NIL	TERM/AGA/MALE/IDM	79	84	64	81	94	80	101	18.1	46.2	16.4	289	9.8	-	-	NORMAL	-	NIL
156	28	398873	G2P1L1	normal	gestational	7 months of ammenorrhea	<6.5	control	398873	39 wk + 4 days	vaginal delivery	2.86	male	148	50	94% on RA	NIL	TERM/AGA/MALE/IDM	61	54	84	79	102	94	78	16.2	45.2	10.1	256	10.2	-	-	-	-	NIL

Sr no	case- one/ control- two	Respiratory Distress	Neonatal Hypoglycemia	Neonatal Hypocalcemia	Congenital Heart Disease	ASD	VSD	PDA
1	2	2	2	2	1	1	2	
2	1	1	2	2	1	1	2	
3	1	1	1	2	1	1	2	
4	1	1	2	2	1	1	2	
5	2	2	2	2	1	1	1	
6	1	2	2	2	1	1	2	
7	1	1	2	1	1	1	2	
8	1	2	2	2	2	2	2	
9	2	1	2	2	2	2	2	
11	1	1	1	1	2	2	2	
10	1	1	2	1	2	2	2	
12	1	2	1	1	2	2	2	
13	1	2	2	2	1	1	2	
14	2	2	2	2	1	1	2	
15	2	2	2	2	1	1	2	
16	2	2	1	2	1	1	2	
17	1	1	2	2	1	1	1	
18	1	2	2	2	1	1	2	
19	1	2	2	1	1	1	2	
20	1	2	2	2	1	2	2	
21	2	2	2	2	2	2	2	
22	2	2	2	2	1	1	2	
23	1	2	2	2	2	2	2	
24	2	2	1	2	2	2	2	
25	1	2	2	2	2	2	2	
26	2	2	2	2	2	2	2	
27	1	1	1	2	1	1	2	
28	2	2	2	2	2	2	2	
29	1	2	2	2	2	2	2	
30	2	2	2	1	2	2	2	
31	2	2	2	2	2	2	2	
32	2	2	2	2	2	2	2	
33	2	2	2	2	2	2	2	
34	2	2	2	2	2	2	2	
35	1	1	1	2	2	2	2	
36	2	2	2	2	2	2	2	
37	2	2	2	2	2	2	2	
38	1	1	2	1	2	2	2	

Sr no	case- one/ control- two	Respiratory Distress	Neonatal Hypoglycemia	Neonatal Hypocalcemia	Congenital Heart Disease	ASD	VSD	PDA
39	2	2	2	1	2	2	2	
40	2	2	2	2	1	1	2	
41	2	2	2	2	2	2	2	
42	2	2	2	2	2	2	2	
43	2	2	2	2	2	2	2	
44	2	2	2	2	1	1	2	
45	2	2	2	1	2	2	2	
46	2	2	1	2	2	2	2	
47	2	2	2	2	2	2	2	
48	2	2	2	2	2	2	2	
49	2	2	2	2	2	2	2	
50	2	2	2	2	2	2	2	
51	1	1	2	2	2	2	2	
52	2	2	2	1	2	2	2	
53	1	2	2	2	2	2	2	
54	1	2	2	2	1	1	2	
55	2	1	2	1	1	1	2	
56	2	1	2	2	1	1	1	
57	1	1	2	1	1	1	1	
58	2	1	2	2	2	2	2	
59	2	2	2	2	1	1	2	
60	2	2	1	1	1	2	2	
61	2	2	2	2	2	2	2	
62	1	2	2	1	1	1	2	
63	2	2	2	2	1	1	1	
64	2	2	2	1	2	2	2	
65	1	2	2	2	2	2	2	
66	2	2	1	2	2	2	2	
67	2	1	2	2	1	1	2	
68	2	2	2	1	2	2	2	
69	2	2	2	2	2	2	2	
70	1	1	2	2	1	1	2	
71	1	1	2	2	1	1	2	
72	2	2	2	2	2	2	2	
73	2	2	2	2	1	1	1	
74	1	2	2	2	1	1	2	
75	1	2	2	2	1	1	2	
76	2	2	2	2	2	2	2	

Sr no	case- one/ control- two	Respiratory Distress	Neonatal Hypoglycemia	Neonatal Hypocalcemia	Congenital Heart Disease	ASD	VSD	PDA
77	2	2	2	2	2	2	2	
78	2	2	2	2	2	2	2	
79	1	1	1	2	2	2	2	
80	2	2	2	2	2	2	2	
81	1	1	2	1	2	2	2	
82	2	2	2	2	1	1	2	
83	2	2	2	2	2	2	2	
84	2	2	1	2	2	2	2	
85	1	2	2	1	2	2	2	
86	1	1	2	2	2	2	2	
87	1	2	2	1	2	2	2	
88	1	2	2	2	2	2	2	
89	2	2	2	2	2	2	2	
90	2	1	1	1	2	2	2	
91	1	1	2	2	1	2	1	
92	2	1	2	1	1	1	2	
93	2	2	1	2	2	2	2	
94	1	2	1	2	1	1	2	
95	1	2	2	2	2	2	2	
96	2	1	2	2	2	2	2	
97	2	1	2	2	1	1	2	
98	2	1	2	1	2	2	2	
99	2	2	2	2	2	2	2	
100	1	1	2	2	1	1	2	
101	1	1	2	1	2	2	2	
102	1	2	2	2	2	2	2	
103	2	1	2	2	2	2	2	
104	2	2	2	2	2	2	2	
105	2	2	2	2	2	2	2	
106	2	2	2	2	2	2	2	
107	2	2	2	2	2	2	2	
108	1	2	2	2	1	1	2	
109	1	2	2	2	2	2	2	
110	2	2	2	2	2	2	2	
111	1	2	1	1	2	2	2	
112	1	2	2	2	1	1	1	
113	1	2	2	2	2	2	2	
114	1	1	2	1	2	2	2	

Sr no	case- one/ control- two	Respiratory Distress	Neonatal Hypoglycemia	Neonatal Hypocalcemia	Congenital Heart Disease	ASD	VSD	PDA
115	2	2	2	2	2	2	2	
116	2	1	2	1	2	2	2	
117	1	2	2	1	1	1	2	
118	1	2	2	2	2	2	2	
119	2	1	2	1	2	2	2	
120	2	2	2	2	2	2	2	
121	1	1	1	2	2	2	2	
122	1	1	2	2	2	2	2	
123	2	2	2	2	2	2	2	
124	1	2	2	2	2	2	2	
125	2	2	1	2	2	2	2	
126	1	2	1	2	1	1	2	
127	1	2	1	2	1	1	2	
128	1	2	2	1	1	1	2	
129	2	2	1	2	2	2	2	
130	1	2	1	2	2	2	2	
131	1	2	2	2	2	2	2	
132	2	2	2	2	2	2	2	
133	1	2	2	1	2	2	2	
134	1	2	2	2	2	2	2	
135	2	1	2	2	2	2	2	
136	2	2	2	2	2	2	2	
137	2	2	2	2	2	2	2	
138	2	2	2	2	2	2	2	
139	2	2	2	2	2	2	2	
140	1	2	2	2	2	2	2	
141	2	1	2	2	1	1	2	
142	2	1	2	1	1	1	2	
143	1	1	2	2	2	2	2	
144	1	2	2	2	2	2	2	
145	2	1	2	2	2	2	2	
146	2	2	2	2	2	2	2	
147	1	2	1	2	2	2	2	
148	1	2	2	2	2	2	2	
149	2	1	2	2	2	2	2	
150	2	2	2	2	2	2	2	
151	1	2	2	2	2	2	2	
152	1	1	2	2	2	2	2	

Sr no	case- one/ control- two	Respiratory Distress	Neonatal Hypoglycemia	Neonatal Hypocalcemia	Congenital Heart Disease	ASD	VSD	PDA
153	1	1	2	2	2	2	2	
154	1	1	2	2	2	2	2	
155	1	2	2	2	2	2	2	
156	2	2	2	2	2	2	2	
		case=26						