"MATERNAL AND PERINATAL OUTCOME IN WOMEN WITH GESTATIONAL DIABETES MELLITUS – A ONE YEAR CROSS SECTIONAL STUDY"

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LIST OF ABBREVIATIONS

ACHOIS	-	Australian Carbohydrate Intolerance Study in Pregnant womentrail group
ACOG	-	American College of Obstetricians and Gynecologists
ADA	-	American Diabetes Association
AFI	-	Amniotic Fluid Index
BMI	-	Body Mass Index
BPP	-	Biophysical Profile
CC	-	Carpenter Coston
CDA	-	Canadian Diabetic Association
CHARGE	-	The Childhood Autism Risks from Genetics and the Environment Study
DFMC	-	Daily Foetal Movement Count
DGGT	-	Decreased Gestational Glucose Tolerance
DIPSI	-	The Diabetes in Pregnancy Study Group in India
DM	-	Diabetes Mellitus
EASD	-	European Association of Study of Diabetics
FBG	-	Fasting Blood Glucose
FBS	-	Fasting Blood Sugar
FPG	-	Fasting Plasma Glucose
GADA	-	Glutamic Acid Decarboxylase Antibodies
GDM	-	Gestational Diabetes Mellitus
HAPO	-	The Hyperglycemia and Adverse Pregnancy Outcome Study
HbA1c	-	Glycated Haemoglobin
HLA	-	Human Leukocyte Antigen
IA-2A	-	Islet Antigen 2 Antibodies

IAA	-	Insulin Auto-Antibodies
IADPSG	-	International Association of Diabetes and Pregnancy Study Groups
ICA	-	Islet Cell Antibodies
IGGT	-	Impaired Gestational Glucose Tolerance
IGT	-	Impaired Glucose Tolerance
IWC	-	International Work Shop Conference
LGA	-	Large For Gestational Age
MNT	-	Medical Nutrition Therapy
MODY	-	Maturity-Onset Diabetes of the Young
MPG	-	Mean Plasma Glucose
NDDG	-	The National Diabetic Data Group
NICE	-	The National Institute of Health and Care Excellence
NIH	-	National Institute of Health
OCP	-	Oral Contraceptive Pill
OGCT	-	Oral Glucose Challenge Test
OGTT	-	Oral Glucose Tolerance Test
PCOD	-	Poly Cystic Ovarian Disease
PG	-	Plasma Glucose
PPBS	-	Post Prandial Blood Sugar
RBG	-	Random Blood Glucose
RDS	-	Respiratory Distress Syndrome
RPG	-	Random Plasma Glucose
SMBG	-	Self Monitoring of Blood Glucose
USPSTF	-	US Preventive Services Task Force
WHO	-	World Health Organization

ABSTRACT

"Maternal and perinatal outcome in women with gestational diabetes mellitus – A one year cross sectional study"

Background*:* Gestational diabetes mellitus is a rising global public health problem that can have short- and long-term sequelae for both mother and offspring.As such GDM has implications beyond the index pregnancy, identifying two generations (mother and her offspring) at risk of future diabetes. Therefore, this study aimed to assess the effects of gestational diabetes mellitus on both maternal and perinatal outcome.

Methods: It is a prospective observational study conducted in KAHER'S Dr Prabhakar Kore charitable hospital over a period of 15months in which women attending the obstetric unit were screened and diagnosed for gestational diabetes according to the IADPSG criteria. Maternal and perinatal outcome were studied.

Results: In this study, prevalence of GDM being 14% and incidence was more in multigravida(60%).Most of the pregnancies were complicated with PIH(18.8%),PROM(9%),and UTI(9%).76%of women were managed alone on diet therapy. In this study78%patients underwent LSCS of which previous LSCS was the main indication. 40% of babies needed NICU care.18% babies had hypoglycaemic episodes at birth 14% babies were macrocosmic at birth. 2% had congenital anomalies while 2% patients had fresh still birth.

Conclusions: Gestational diabetes complicating pregnancy has adverse maternal and fetal outcome. Considering high prevalence in Indian population, it is recommended and suggested by various evidences that universal screening irrespective of the risk status and early detection of GDM provide window for implementation of various management

methods to keep check on blood sugar level and maintain good glycemic control in pregnant women. Further research should focus on preconceptional diagnosis and management of glucose intolerance in the vulnerable Indian population to reduce the burden of adverse outcomes related to gestational diabetes mellitus.

Key words: Gestational diabetes, Maternal, Fetal, Outcome

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INTRODUCTION

GDM is one of the most common endocrine metabolic disorder seen among pregnant women. Due to exponentially rising prevalence of diabetic population,GDM has gained global importance. According to International Diabetes Federation (IDF) estimation, around 223 million women were living with diabetes in 2020, 20 million or 16 percent live births in women were affected by Hperglycemia in pregnancy (HIP) and around 84% was due to GDM.[1] Also 1 in 6 births was affected by GDM.[2]The prevalence in India reported to be between 4.6 %-14% in urban areas and 1.7% - 13.2% in rural areas.[3]

Gestational diabetes mellitus (GDM) is usually defined as "carbohydrate intolerance with recognition or onset during pregnancy". Women with GDM have high risk of developing type 2 diabetes, especially 3-6yrs after delivery.[4] Exposure to hyperglycaemia in utero make children susceptible to a high risk of becoming overweight or obese, associated with development of type 2 diabetes. Women with abnormal glucose intolerance i.e GDM are at high risk of developing adverse maternal and fetal outcomes. Thus GDM offers an window for development, testing and application of clinical strategies for prevention of diabetes related complications in pregnant women and their offsprings. As the Indian women are 11 times more at risk of developing GDM as compared to Caucasian women, Federation of obstetrics and gynaecology of India (FOGSI) and Government of India (GOI) recommend universal screening for all pregnant women for early detection of GDM and further intervention, improving maternal and fetal prognosis. [5]

Previous studies have reported that GDM was associated with several maternal and fetal complications and can result in significant morbidity and mortality. Maternal complications include PIH, preterm labour, polyhydramnios, abortions,

infections, APH, PPH, puerperal sepsis. Among fetal complications, macrosomia, RDS, hypoglycaemia, hyperbilirubinemia, hypocalcaemia, IUFD and congenital anomalies .[6]

Considering the higher prevalence of GDM as per previous in house study reporting prevalence of GDM as 18% in our hospital[7], this study was conducted to know the various maternal and perinatal outcome in pregnant women with GDM in KAHER'S Dr. Prabhakar Kore Hospital a tertiary centre in south India.

OBJECTIVE

- **Primary objective:** To study maternal and perinatal outcome in gestational diabetes mellitus women

REVIEW OF LITERATURE

Background

Diabetes is defined as a group of metabolic disorders characterized by defectsof insulin secretion and/or insulin action which leads to hyperglycaemia. There are different forms of diabetes, but the long term negative side effects of chronichyperglycaemia on different organs such as kidneys (nephropathy), eyes (retinopathy), blood vessels (angiopathy), nerves (neuropathy) and heart remain the same [8].

History of Diabetes Mellitus

Clinical features of diabetes were first described by the ancient Egyptians about 1550 B.C. In the Tomb of Thebes, a papyrus was discovered where polyuria was mentioned. It was sold to the German Egyptologist Georg Ebers in 1872 and named after him as the Ebers Papyrus. Even though the Ebers papyrus was written about 1550 BC, evidence suggests that it was copied from a series of books from 3400 BC [9,10].

Aretus of Cappodocia from ancient Greece (81-133 AD) was first to use the term "diabetes", which came from the Greek word for siphon [11]. The clinicaldiagnosis of diabetes with polyuria and glycosuria was described by the Hindu physicians Charaka, Sushrutha and Vaghbata. They found that the urine of those affected attracted flies and ants, and they called it "honey urine" [10].

The word mellitus (honey sweet) was added by the British physician Thomas Willis in 1675 when he as the first European discovered the sweetness of urine in patients with diabetes 12. In 1776, Doctor Matthew Dobson from Manchester did experiments showing that sugar was present in both urine and blood of diabetic

patients. Another important man in the history of diabetes was the Frenchman Claude Bernard, who through experiments in the early 19th century discovered the role of the liver in glycogenesis.

A German medical student, Paul Langerhans was the first who found the pancreatic islets cells in 1869, but did not know their function[9,10,11]. Later, in 1893, the French histologist Gustave Laguesse named the islet cells "islets of Langerhans" after their discoverer[14,15]. In 1889, German diabetologist Oscar Minkowski and pharmacist Joseph von Mering demonstrated that removal of the pancreas from a dog led to development of diabetes in the dog. Insulin was discovered not long thereafter. It was the young physician, Frederick Banting, who thought it might be possible to isolate the internal secretions of the pancreas by ligating the pancreatic ducts to induce atrophy of the acinar cells and thereby minimize contamination of the tissue extract with digestive enzymes.

Banting presented his suggestion to J.J.R. Macleod, a physiologist at the University of Toronto who provided Banting with a laboratory for the summer and some dogs for the experiments. Macleod also assigned Charles Best, a young student,to work as Banting's assistant.

During 1921, Banting and Best made remarkable progress, and by fall they had isolated material from pancreas extracts that dramatically prolonged the life of dogsmade diabetic by removal of the pancreas. In 1922, Banting and Best treated their first human patient, a 14-year old boy named Leonard Thompson, whose life was saved by the treatment [16].

Following this, the Eli Lilly Company was brought in to collaborate in the production and manufacture of insulin. By 1923, insulin was available in quantities adequate for relatively widespread treatment of diabetes.

In 1923, the Nobel Prize in Medicine was awarded to Banting and Macleod. To acknowledge Best's role in the discovery of insulin, Banting shared his prize withhim [9].

1940- first recognition that maternal hyperglycaemia influences pregnancy outcomes.

1952- Jorgen Pedersen puts forward the hyperglycaemia - hyperinsulinemia hypothesis to explain fetal macrosomia (later termed as Pedersen hypothesis)

1961- John B. O'Sullivan introduced the term Gestational Diabetes Mellitus (GDM).

1964- O'Sullivan and Mahan criteria for diagnosing GDM introduced; these were later modified by Carpenter and Coustan Early 1970s- Norbert Freinkel introduced the terms "facilitated anabolism" and "accelerated starvation"

1999-WHO criteria for diagnosis of GDM introduced

2005- ACHOIS study underlined the benefits of treating GDM

2006- DIPSI guidelines published

2008- HAPO study findings published

2010- IADPSG revises the diagnostic criteria for GDM

2012- ADA endorses IADPSG criteria

2013- NIH states that more evidence is needed before IADPSG criteria are adopted

While the first case of GDM was reported by Benniwitz in Germany in 1853 (he considered diabetes a "symptom of pregnancy" and noted that glycosuria resolvedafter delivery), the concept itself is relatively recent.

Classification of Diabetes Mellitus

In non-pregnant individuals, the type of diabetes is based on its presumed etiopathogenesis and its pathophysiological manifestations. Absolute insulin deficiency characterizes type 1 diabetes. In contrast, defective insulin secretion, insulin resistance, or increased glucose production characterizes type 2 diabetes. Both types are generally preceded by a period of abnormal glucose homeostasis. Pancreatic beta cell destruction can begin at any age, but type 1 diabetes is clinically apparent most often before age 30. Type 2 diabetes usually develops with advancing age but isincreasingly identified in younger obese adolescents [1]. The table-1 represents the etiological classification of diabetes mellitus.

Table 1: Etiological Classification of Diabetes Mellitus

Type 1: β-cell destruction, usually absolute insulin deficiency Immune-mediated Idiopathic
Type 2: Ranges from predominantly insulin resistance to predominantly an insulin resistance to predominantly an insulin secretory defect with insulin resistance.
Other Type: Genetic mutations of β-cell function – MODY 1-6, others Genetic defects in insulin action Genetic syndromes – Down, Klinefelter, Turner Diseases of the exocrine pancreas – pancreatitis, cystic fibrosis Endocrinopathies – Cushing syndrome, Pheochromocytoma, others Drug or chemical induced – glucocorticosteroids, thiazides, β-adrenergic agonists,others Infections – congenital rubella, cytomegalovirus, coxsackievirus. **Gestational diabetes** MODY = maturity-onset diabetes of the young.Modified from powers, 2012.

Type 1 Diabetes Mellitus

Type 1 diabetes is sometimes called insulin-dependent, immune-mediated or juvenile-onset diabetes. This form of diabetes is caused by a cellular mediated autoimmune destruction of the insulin producing beta-cells in the pancreas. The reason why this occurs is not fully understood and is related to multiple genetic predispositions and environmental factors.

Markers of the autoimmune process such as ICA, IAA, GADA and IA-2A are present in 85–90% of individuals at their onset of autoimmune diabetes. There is also a strong association between type 1 diabetes and the human leukocyte antigen (HLA) region on chromosome 6p2 and the DQA and DQB genes. The disease can affect people of any age, but usually occurs in children or young adults and the progression of the disease is variable. Younger patients usually have a more rapid progression, often together with ketoacidosis. Patients with type 1 diabetes always need insulin treatment, since the majority of the beta-cells are destroyed.

At present, type 1 diabetes cannot be prevented. For women with type 1 diabetes, pregnancy can lead to different complications. In a UK study, the perinatal mortality in babies of women with type 1 diabetes was 3.2% and the prevalence of major congenital anomalies was 4.8% [18]. A study from the Netherlands showed congenital malformations in 8.8% (5.5% for major congenital malformations) and perinatal mortality in 2.8% of babies to women with type 1 diabetes [19].

Type 2 diabetes mellitus

Type 2 diabetes is sometimes called non-insulin dependent diabetes or adult- onset diabetes, and is characterized by relative insulin deficiency and insulin resistance, either of which may be the predominant feature. At least initially, and often through many years, these patients do not need insulin treatment. The diagnosis

is more common among older people and over represented among obese patients. Type 2 diabetes can remain undetected for many years and is often incidentally discovered after associated complications or at regular health controls [8,19]. By maintaining a healthy weight and being physically active, type 2 diabetes can be prevented, or at least delayed in many cases. As in type 1 diabetes, pregnancies with type 2 diabetes can lead to complications. In a UK study during 1990-2002, the rate of perinatal mortality was 2.5% and congenital malformation was 9.9%. Another large study from UK showed a perinatal mortality of 3.2% and that the prevalence of major congenital anomalies was 4.3% [19]. When comparing pregnancy outcomes in type 1 and type 2 diabetes, some studies show almost the same rate of malformation and mortality [18,20,21], or even higher rates in type 2 than in type 1 diabetes.

Gestational Diabetes Mellitus

GDM was for many years defined as "any degree of glucose intolerance with onset or first recognition during pregnancy". Even though GDM often resolves after delivery, the definition applied whether or not the condition persisted after pregnancy. Therefore, it did not exclude the possibility that the glucose intolerance could have antedated or begun concomitantly with the

pregnancy. Though the limitations of this definition were apparent for many years, the definition remained. Because the number of women with overweight, obesity and diabetes continue to increase, the number of pregnant women with undiagnosed type 2 diabetes has increased. Therefore, the International Association of Diabetes and Pregnancy Study Groups (IADPSG) recommend that high risk women, where diabetes is found at their initial prenatal visit, receive the diagnosis overt diabetes instead of the GDM diagnosis.

Women diagnosed with GDM might have:

- Normal glucose tolerance prior to pregnancy which becomes abnormal with pregnancy and returns to normal following delivery
- Mild glucose intolerance ("pre-diabetes") before pregnancy which worsens duringpregnancy
- Previously undiagnosed type 2 diabetes
- Previously undiagnosed type 1 diabetes (rare)

The term GDM is used classically to define the onset or first recognition of abnormal glucose tolerance during pregnancy. This may include previously unrecognised diabetes that may have antedated pregnancy. The ACOG (2011) and theNational Institute of Health (2013) endorse this definition.

GDM, depending on the population studied, affects 1-14% of all pregnant women.[1] In Sweden 2% of pregnancies are complicated by GDM. GDM is often more common in populations with a high frequency of type 2 diabetes, such as India and China. It is well known that women with GDM have

a substantial risk of developing type 2 diabetes later in life, but the risk of developing type 1 diabetes is also increased.

India leads the world with largest number of diabetic subjects earning the dubious distinction of "the diabetes capital of the world". It was estimated to have had 31.7 million people having diabetes in year 2000 which is projected to be 79.4 million by year 2030. Both the figures are highest in the world. According to the Diabetes Atlas 2009 published by the International Diabetes Federation, the number of people with diabetes in India in year 2010 was reported to be around 50.8 million which is expected to rise to 69.9 million by 2025 unless urgent preventive steps are taken. The so-called Asian Indian Phenotype refers to certain unique clinical and biochemical abnormalities in Indians which includes but is not limited to increased insulin resistance, greater abdominal adiposity i.e., higher waist circumference despite lower body mass index. This phenotype makes Indians more prone to diabetes. Although genes are there to be blamed, but the primary driver of the epidemic of diabetes is the rapid epidemiological transition associated with changes in dietary patterns and decreased physical activity as evident from the higher prevalence of diabetes in the urban population. The 1997 WHO estimates of the prevalence of diabetes in adults showed an expected total rise of > 120% from 135 million in 1995 to 300 million in 2025. These numbers also include GDM, and should alert physicians to the need to direct special attention to this population, especially in developing countries.

History of Gestational Diabetes Mellitus

Gestational diabetes mellitus (GDM) was first described in 1823 by the German physician Heinrich Bennewitz, who described thirst and polyuria in a pregnant woman. He considered that diabetes actually was a symptom of the pregnancy, since the symptoms and the glycosuria disappeared after pregnancy[22].

Studies in the 1940s and 1950s showed that a lesser degree of maternal hyperglycaemia during pregnancy also was a risk for pregnancy outcome and increased perinatal mortality [23,24,25,26].

The case report of Fredrica Pepe, age 22, who was admitted to the Berlin Infirmary at 7 months into her fifth pregnancy on 13th November 1823, is probably the first description of GDM in literature. This case report was a part of thesis of Heinrich Gottleib Bennewitz for the degree of Doctor of Medicine, which he publiclydefended at the University of Berlin on 24 June, 1824.

The Belgian researcher Hoet JP published a study called "Carbohydrate Metabolism during Pregnancy" in Frenchand was the first to use the term "metagestational diabetes". The paper was translated into English by doctor F.D.W. Lukens and published in Diabetes 1954 [27].

The modern term "gestational diabetes" was used by John B O'Sullivan in 1961 and is said to have been used instead of the more neutral "Carbohydrate Intolerance of Pregnancy".

In 1964 John B. O'Sullivan performed a 100 gram 3-hour oral glucose tolerance test (OGTT) in 752 pregnant women during mainly the second or third trimester. From this material the first, second and third standard deviation (SD) upper limits for these glucose values were published, which were the first

statistically based criteria for assessing the upper limit of glycaemic normality in pregnancy.

The O'Sullivan criteria, published with statistician Claire Mahan, were the standard for diabetes detection in pregnancy for the next 40 years [27]. Jorge H. Mestman showed an increased rate of perinatal mortality associated with abnormal glucose tolerance in southern California. The population consisted of more than 60% Latin women [28]. In October 1979, doctor Norbert Freinkel (representing the American Diabetes Association) and doctor John Josimovich (representing the American College of Obstetricians and Gynaecologists) met in Chicago at the First International Workshop Conference on Gestational Diabetes Mellitus. Experts from around the world attended this meeting and shared their clinical experience, research, and opinions about GDM. During this and the next coming International Workshop Conferences on GDM held in 1984 and 1990 a definition of GDM was established[29].

The first series of pregnancy in Diabetes was reported by Duncan (1862). Glycosuria associated with pregnancy and lactation was recognized by Blot (1856).

Different urinary sugars especially Lactose was isolated in pregnancy and puerperium by Hofmeister (1877). Dubreuil and Anderodias (1920) identified that the islets of Langerhans in still born foetuses born to diabetic mothers, were hypertrophied. Insulin was first isolated by Fredrick and Banting (1921) and soon after its advent it was administered by Graham in England and Revenoin USA. Skipper (1933) was the first to classify diabetes in pregnancy according to onset, degree of severity and diabetic content. Priscilla White (1949) mentioned the high risk factor for developing GDM and also classified GDM.

Changes during pregnancy with Gestational Diabetes Mellitus Metabolism

In a pregnancy complicated by GDM, the same metabolic changes occur as ina normal pregnancy. During pregnancy, the mother's metabolism is adapted to supplynutrients to the foetus for its growth. Glucose is the main nutrient that crosses the placenta and constitutes the primary energy source for the foetus. Early during pregnancy basal plasma glucose, hepatic gluconeogenesis and insulin levels are unchanged [30]. But during late pregnancy the mother develops basal hypoglycaemia, which is due to the high rate of placental transfer, despite reduced glucose consumption (because of insulin resistance) and enhanced gluconeogenesis. The placental transfer of glucose is carried out by facilitated diffusion by different glucosetransporters (GLUT) and concentration dependent kinetics [31].

In the first half of pregnancy, there is storage of energy and nutrients due to maternal changes. The appetite of the mother is increased and the insulin sensitivity isnormal or increased. This leads to an increase in the lipid store [30,32].

During the second half of pregnancy, the stored reserves are used for foetal and placental growth. The insulin resistance also increases during this time and leads to a decreased uptake of glucose by maternal tissues sensitive to insulin, such as muscle and adipose tissues [33].

Insulin resistance

The mechanisms behind insulin resistance induced by the pregnancy per se are still not fully understood. In pregnant rats (are believed to be similar to humans) the degradation of insulin by the placenta is increased, which leads to

accelerated insulin removal [32]. There are also different hormonal and metabolic changes during the second half of pregnancy which facilitate insulin resistance. One is the high plasma level of progesterone during the second part of pregnancy.

GDM is associated with both insulin resistance and an impaired insulin secretion. There is a lack of insulin during a period of time with high insulin needs, to compensate the insulin resistance that develops during the third trimester of pregnancy. In the maternal tissues where glucose uptake is insulin-dependent, the uptake is decreased because of the lack of insulin and postprandial hyperglycaemia develops. Since the maternal-placental foetal transfer of glucose is concentration dependent, the hyperglycaemia of the mother leads to an increased placental transfer of glucose to the foetus. This leads to foetal hyperglycaemia and hyperinsulinism. Because insulin is one of the main growth factors for the foetus, the hyperinsulinism leads to macrosomia and can cause delivery complications such as shoulder dystocia[34].

The hyperinsulinism remains in the newborn after delivery and once the umbilical supply of glucose has disappeared, the risk of hypoglycaemia is increased. Early feeding of the newborn is important as well as the monitoring of their blood glucose levels, since untreated hypoglycaemia can lead to brain damage [35].

Classification of diabetes in pregnancy

Women can be classified into those who were known to have diabetes before pregnancy—**pregestational or overt,** and those diagnosed during

pregnancy— **gestational diabetes**. African American, Native American, Asian, and Hispanic women are at higher risk for gestational diabetes compared with white women. IADPSG in 2010 recommended a change in the classic definition and this was endorsed by ADA in 2012.

The IADPSG classifies diabetes in pregnancy as 2 categories – overt and gestational (Metzinger et al, 2010).

1. **Overt diabetes:** a diagnosis of overt diabetes can be made in women who meet any of the following criteria in their initial prenatal visit:

a. Fasting plasma glucose $\geq$ 126mg/dL (7.0 mmol/L), or

b. $A_1C \geq 6.5\%$ using a standardised assay, or

c. Random plasma glucose $\geq$ 200mg/dL (11.0mmol/L) that is subsequently confirmed by elevated plasma glucose or A_1C as described above.

2. **Gestational diabetes:** a diagnosis of gestational diabetes can be made in womenwho meet either of the following criteria (ADA, 2011):

a. FBS $\geq$ 92mg/dL (5.1 mmol/L), but <126 mg/dL (7.0 mmol/L) at any gestational age (fasting plasma glucose $\geq$ 126 mg/dL [7.0 mmol/L] is consistent with overt diabetes)

b. At 24-28 weeks of gestation: 75g, 2-hour OGTT with atleast one abnormal value: FBS $\geq$92 mg/dL, but <126 mg/dL, one hour value $\geq$180 mg/dL and2 hour value $\geq$153mg/Dl

White Classification in Pregnancy

The classification proposed by Priscilla White for diabetic pregnant women was the mainstay of management until mid-1990s. The one previously

recommended by the American College of Obstetricians and Gynaecologists (1986) is shown in table2.

Table 2: Classification Scheme Used from 1986 through 1994 for DiabetesComplicating Pregnancy

Class	Onset	Plasma Glucose Level		
		Fasting	2-Hour Postprandial	Therapy
A_1	Gestational	< 105 mg/dL	< 120 mg/dL	Diet
A_2	Gestational	> 105 mg/dL	> 120 mg/dL	Insulin
Class	**Age of Onset[Yr]**	**Duration [Yr]**	**Vascular Disease**	**Therapy**
B	Over 20	< 10	None	Insulin
C	10 to 19	10 to 19	None	Insulin
D	Before 10	> 20	Benign retinopathy [a]	Insulin
F	Any	Any	Nephropathy	Insulin
R	Any	Any	Proliferative retinopathy	Insulin
H	Any	Any	Heart	Insulin
[a]When diagnosed during pregnancy: proteinuria ≥ 500 mg/24 hr before 20 weeks gestation.				

The American College of Obstetricians and Gynaecologists (2012, 2013) no longer recommended the White classification. Instead, the current focus is whether diabetes antedates pregnancy or is first diagnosed during pregnancy [39,40]. Currently the classification proposed by the American Diabetes Association (ADA), as shown intable-3 is recommended [8].

SCREENING AND DIAGNOSIS OF GESTATIONAL DIABETES MELLITUS

Detecting the evidence of diabetes mellitus in pregnancy is a major challenge as the condition is associated with diverse range of adverse maternal and neonatal outcomes. Various screening guidelines have been introduced depending upon the suitability of test to the population characteristics, cost and screening accuracy.

Table 3: Proposed Classification System for Diabetes in Pregnancy

Gestational Diabetes: Diabetes diagnosed during pregnancy that is not clearly overt [type 1 or type 2] diabetes	
Type 1 Diabetes	**Type 2 Diabetes**
Diabetes resulting from β-cell destruction, usually leading to absolute insulin deficiency.	Diabetes from inadequate insulin secretion in the face of increased insulin resistance.
a. Without vascular complications.	a. Without vascular complications.
b. With vascular complications (specifywhich)	b. With vascular complications (specifywhich)
Other type diabetes: genetic in origin, associated with pancreatic disease, drug-induced, of chemically induced.	
* Data from American Diabetes Association, 2012.	

Still there are lots of controversies as to which test to be used, when should the screening be done and on whom it should be applied. Multiplicity of the guidelines given is the reflection of lack of available evidence demonstrating a benefit of specified health outcome with any of national and international standard screening criteria. Till the search for ideal screening strategy is ongoing, factors like clinical judgement and available resources play important

role in choosing best possible mode for evaluation of glucose intolerance in pregnant women

American Diabetes Association (Carpenter and Couston) recommends 3 hour 100 gm OGTT and Gestational Diabetes Mellitus is diagnosed if any two values meet or exceed FBS > 95 mg/dl, 1 hr PG > 180mg/dl, 2 hr PG > 155 mg/dl and 3 hr PG >140 mg/dl. This criteria was originally validated against the future risk of developing diabetes and not on the foetal outcome [8]. Carpenter himself now recommends a 2hour OGTT with 75 gm glucose. The reason for this is that when a glucose tolerance test is administered to non-pregnant individuals, it is standard to use the 75 gram 2-hour OGTT. Using a different glucose challenge in pregnant versus non-pregnant patients leads to confusion in the laboratory and may result in errors in applying the proper diagnostic criteria. Further, the 75-g, 2-hour OGTT is in use during pregnancy in many countries around the world, typically using the same thresholds as in non- pregnant individuals. The ACOG recommends a two-step approach, as proposed by the National Diabetic Data Group (NDDG) and Carpenter and Coustan (1982). This involves a 50-g oral glucose challenge test in the non-fasting state. If the 50g OGCT is positive, the second step is undertaken. The diagnostic test for GDM is the 100g, 3 hour OGTT.

To standardize the diagnosis of GDM, the World Health Organisation (WHO) proposed using a 2 hour 75 g OGTT with a threshold plasma glucose concentration of greater than 140 mg/dl at 2 hour, similar to that of IGT, outside pregnancy and greater than 200 mg/dl as Overt diabetics. The single-step approach proposed by the WHO in 1999 and the IADPSG 2010 is most commonly used to diagnose GDM, where a 75g OGTT is administered to the

fasting women. In 2011, ADA endorsed the IADPSG recommendations. Using the WHO approach, fasting and 2-hr post-load glucose levels are measured, and using the IADPSG approach, fasting, 1 hr, 2 hr levels are evaluated. The WHO criteria require one or more values and the IADPSG considers any single abnormal value as diagnostic of GDM.

The DIPSI (2005, 2006) guidelines recommend a simplified screening diagnostic test. When the pregnant women walks in to the antenatal clinic in the fasting state, she is given a 75g oral glucose load, and at 2 hours a venous blood sample is collected for estimating plasma glucose. This one-step procedure of challenging women with 75-g glucose and diagnosing GDM is considered by its proponents to be simple, economical and feasible, especially in developing countries.

The 2008 U.S. Preventive Services Task Force (USPSTF) evidence review on screening for GDM concluded that at that time, "evidence was insufficient to assess the balance of benefits and harms of screening for GDM either before or after 24 weeks gestation". The report suggested that, until there was better evidence, clinicians should discuss screening for GDM with their patient and make case-by-case decisions. Discussions should include information about the uncertainty of benefits and harms as well as the frequency of positive screening test results [41].

The 2001 practice guidelines of the American College of Obstetricians and Gynaecologists (ACOG) endorsed risk factor-based screening for GDM, recognizing that low-risk women may be less likely to benefit from screening

with glucose measurements. Women were considered **low risk** of GDM if they met all thefollowing criteria-

(1) Younger than 25 years;

(2) Not a member of an ethnic group at high risk for development of type 2 diabetesmellitus;

(3) BMI $\leq$ 25 kg/m^2

(4) No history of previous glucose intolerance or adverse pregnancy outcomes

(5) Associated with GDM; and

(6) No first degree relative with known diabetes.(Blood glucose screening not routinely required in these)

ACOG plans to update its 2001 practice guidelines on GDM based on the proceedings of the 2012 National Institutes of Health consensus conference on GDMdiagnosis.

Until 2011, the American Diabetes Association (ADA) also endorsed no screening for pregnant woman who met all the criteria mentioned above for low risk of GDM. In 2011 the ADA changed their recommendations to endorse glucose testingfor GDM in all pregnant women who do not have a diagnosis of pregestational diabetes.

The pregnant women who belong to low risk have already been mentioned above, the criteria for moderate and high risk are as follows (Adapted from the FourthInternational Workshop Conference on GDM, August 1998):

Moderate risk: One or more of the following:

(1) Age > 25 years

(2) Member of an ethnic group with high prevalence of GDM

(3) Diabetes in first degree relative

(4) Overweight prior to pregnancy

(5) Weight high at birth

(In these women, blood glucose testing be done at 24–28 weeks, one or two step procedure)

High risk:

(1) Marked obesity

(2) Strong family history of type 2 DM

(3) Previous history of GDM or impaired glucose tolerance or glycosuria or macrosomic baby

(In these women, glucose testing should be done as soon as possible)

Common practices of glucose screening for GDM in North America involve a two-step approach in which patients with abnormal results on a screening test receivea subsequent diagnostic test. Typically, a 50 g oral glucose challenge test (OGCT) is initially administered between 24 and 28 weeks' gestation in a non-fasting state, in women at moderate risk (i.e., women who do not meet all low risk criteria but lack two or more risk factors for GDM). The test is administered earlier in gestation for women at high risk of GDM (i.e.,

multiple risk factors for GDM) and repeated at 24–28 weeks' gestation if initial surveillance is normal. Patients who meet or exceed a screening threshold (usually 130 mg/dL or 140 mg/dL) receive a more involved diagnostic test—the oral glucose tolerance test (OGTT), in which a 75 g or 100 g oral glucose load is administered in a fasting state, and plasma glucose levels are evaluated after 1, 2, or 3hours. A diagnosis of GDM is made in pregnant women when one or more glucose values fall at or above the specified glucose thresholds.

Alternatively, a one-step method in which all patients or high-risk patients forego the screening test and proceed directly to the OGTT has been recommended[2].

The absence of a universally accepted gold standard for the diagnosis of GDM has resulted in a variety of recommended diagnostic glucose thresholds that have been endorsed by different stakeholders (Table-4). These criteria reflect changes that have occurred in laboratory glucose measurements over the years and in new evidence that suggests the ability of different glucose thresholds to predict poor pregnancy outcomes. The different diagnostic criteria and thresholds result in different estimates of the prevalence of GDM.

Table 4: Diagnostic criteria and plasma glucose thresholds for gestational diabetes mellitus

Organization	Year	Testing Schedule	Abnormal Value(s)	Threshold (equal to or greater than)			
				0 (h)	1 (h)	2 (h)	3 (h)
ADA	1999	50 g OGCT	1	-	140 mg/dL 7.8mmol/L	-	-
		100 g OGTT	2 or more	105 mg/dL 5.8 mmol/L	190 mg/dL 10.5 mmol/L	165 mg/dL 9.1 mmol/L	145mg/dL 8.0 mol/L
ADA Low risk excluded	2000-2010	50 g OGCT	1	-	130 mg/dL 7.2 mmol/L (or) 140 mg/dL 7.8mmol/L	-	-
		100 g (or) 75g OGCT after overnight fast ≥ 8hr	2 or more	95 mg/dL 5.3 mmol/L	180 mg/dL 10.0mmol/L	155 mg/dL 8.6 mmol/L	140mg/dL 7.8 mol/L (3 hr value only for100g test)
IADPSG ADA	2011	75 g OGTT	1 or more	92 mg/dL 5.1 mmol/L	180 mg/dL 10.0mmol/L	153 mg/dL 8.5 mmol/L	-
1. CC 2.4th IWC(same) 3.5thIWC(same as 4th, but 75g is accepted with same glucose thresholds)	1.1982 2.1998 3.2007	50 g OGCT	1	-	130 mg/dL 7.2 mmol/L	-	-
		100 g OGCT	2 or more	95 mg/dL 5.3 mmol/L	180 mg/dL 10.0 mmol/L	155 mg/dL 8.6 mmol/L	140mg/dL 7.8 mmol/L
NDGG	1979	50 g OGCT	-	-	-	-	-
		100 g OGCT	2 or more	105 mg/dL 5.8 mmol/L	190 mg/dL 10.5mmol/L	165 mg/dL 9.1 mmol/L	145 mg/dL 8.0 mmol/L
WHO	1999 WHO consultation	75 g OGTT	1	6.1 mmol/L forIGT of pregnancy; 7.0 mmol/L for Dxof DM	-	140 mgl/dL 7.8mmol/L for IGT of pregnancy; 200 mg/dL 11.1 mmol/L for Dx ofDM	-

Organization	Year	Testing Schedule	Abnormal Value(s)	Threshold (equal to or greater than)			
				0 (h)	1 (h)	2 (h)	3 (h)
WHO	1985 WHO study groupreport	75 g OGTT	1	7.8 mmol/L 140 mg/dL for IGT of pregnancy;	-	140 mgl/dL 7.8mmol/L for IGTof pregnancy; 200mg/dL 11.1 mmol/L for Dx ofDM	-
CDA	2003, 2008	50 g OGCT	1	-	140 mgl/dL 7.8mmol/L (or) 186 mg/dL, 10..3 mmol/L for Dx ofGDM	-	-
		75 g	2 or more	95 mg/dL 5.3 mmol/L	191 mg/dL 10.6mmol/L	160 mg/dL 8.9mmol/L	-
ACOG - risk factor, 4th IWC	2001	50 g	1	-	130 mgl/dL 7.2 mmol/L (or) 140 mg/dL, 7.8mmol/L	-	-
		100 g CC	2 or more	95 mg/dL 5.3 mmol/L	180 mg/dL 10.0mmol/L	155 mg/dL 8.5mmol/L	140mg/dL 7.8 mmol/L
		100 g NDGG	2 or more	105 mg/dL 5.8 mmol/L	190 mg/dL 10.5 mmol/L	165 mg/dL 9.1 mmol/L	145mg/dL 8.0 mmol/L
3rd IWC	1991	100 g OGTT	2 or more	105 mg/dL 5.8 mmol/L	190 mg/dL 10.5 mmol/L	165 mg/dL 9.1 mmol/L	145mg/dL 8.0 mmol/L
ADIPS	1998	50 g or 75 g non-fasting	1	-	140 mgl/dL 7.8mmol/L (50 g) (or) 144 mg/dL, 8.0 mmol/L (75g)	-	-
		75 g fasting	1	99 mg/dL 5.5 mmol/L	-	144 mgl/dL 8.0 mmol/L (or) 162 mg/dL, 9.0 mmol/L	-
EASD	1996	75 g	1	108 mg/dL 6.0	-	162 mg/dL 9.0	-

				mmol/L		mmol/L*	
USPSTF (Grade 1 Recommendation)	2008**	Risk Assessment50 g OGCT	1	-	130 mgl/dL 7.2 mmol/L (or) 140 mg/dL, 7.8 mmol/L	-	-
		100 g OGTT	2 or more	NR	NR	NR	NR
DIPSI	2006	75g	1	-	-	140mg/dL 7.8 mmol/L	-

ACOG = American College of Obstetricians and Gynaecologists; ADA = American Diabetic Association; ADIPS = Australian Diabetics in Pregnancy Society; CC = Carpenter Coston; CDA= Canadian Diabetic Association; DM = Diabetic Mellitus; Dx =Diagnosis; EASD = European Association of study of Diabetics; GDM = Gestational Diabetics Mellitus; IADPSG = International Association of Diabetes in Pregnancy Study Groups; IGT = Impaired Glucose Tolerance; IWC = International work shop conference; NDDG = National Diabetes Data Group; NR = Not Responded; OGCT = Oral Glucose Challenge Test; OGTT = Oral Glucose Tolerance Test; USPSTF = US Preventive services Task Force; WHO = World Health Organization; DIPSI = The Diabetes in Pregnancy Study Group in India

Low Risk defined age < 25 years, normal body weight, no first degree relative with DM, no history of abnormal glucose, no history of obstetrical outcomes, not of high risk ethnicity of DM.

*New Zealand

** Screening for GDM: USPSTF recommendation statement Ann Intern Med 2008;148(10):759-65.

Universal Screening versus selective screening

Although uncertainty exists as to the value of diagnosis and treatment of GDM, universal screening for this entity is widely practiced. In order to reduce the burden of screening on women and the health care system, the concept of selective screening was introduced. Selective screening originally consisted of taking a personal and family history in order to identify a high-risk population in need of further directed testing. Women with any of the risk factors like Previous history of gestational diabetes or glucose Intolerance, A family history of diabetes, age >25 yrs, BMI >25 kg/m^2, Previous macrosomia (> 4,000 g), Previous unexplained stillbirth, Previous neonatal hypoglycaemia, hypocalcaemia, or hyperbilirubinemia. Advanced maternal age, Obesity, Repeated glycosuria in pregnancy, Polyhydramnios, Suspectedmacrosomia were advised to perform a 50 g glucose challenge test. Screening by riskfactors alone has a sensitivity of 63% and a specificity of 56%. In other words, 37%–50% of women with GDM may go undiagnosed using this approach. Due to this lowsensitivity, most guidelines prior to 1995 recommended universal biochemical screening. Recent data and reviews of existing data suggesting that women at low riskfor GDM could be exempt from biochemical screening led the American Diabetes Association to revise their guidelines to recommend that women who are 25 years old or younger, who are Caucasian and are not obese (< 20% over desired body weight orBMI 27 kg/m^2) could be exempt from screening. This revised concept of selective screening will still result in screening 90% of all pregnant patients. Thus many clinicians continue the practice of universal screening [7].

In 2004, a cross-sectional study reported that universal screening was the most common practice in the United States, with 96 percent of obstetricians routinely screening for GDM. In contrast, the guidelines of ACOG and the ADA at that time stated that women at low risk for GDM were unlikely to benefit from screening. Since only 10 percent of pregnant women were categorized as low risk, some argued that selective screening contributed to confusion, with little benefit and potential for harm. Recently a large multinational epidemiologic study - Hyperglycemia and Adverse Pregnancy Outcomes (HAPO) study, demonstrated that risk of adverse maternal, fetal, and neonatal outcomes continuously increased as a function of maternal glycaemia at 24–28 weeks, even within ranges previously considered normal for pregnancy. These results have led to careful reconsideration of the diagnostic criteria for GDM. International association of diabetes and pregnancy study groups (IADPSG) developed diagnostic cut points for the fasting, 1-hr, and 2-hr plasma glucose measurements after 75 g OGTT at 24–28 weeks of gestation that conveyed an odds ratio for adverse outcomes of at least 1.75 compared with women with the mean glucose levels in the HAPO study [6].

They also recommended that a single glucose value, rather than at least two abnormal values at or above diagnostic glucose thresholds on the OGTT be accepted as sufficient for a diagnosis of GDM. Since overt diabetes is often asymptomatic, may not have been screened for before conception, has a prevalence that is increasing dramatically in reproductive-age women, and carries a higher risk for poor pregnancyoutcomes, the IADPSG also recommended that all women, or at least women from high-risk groups for type 2 diabetes mellitus, be

screened for overt diabetes at their first prenatal visit and excluded from the diagnosis of GDM using one of thefollowing criteria:

- fasting plasma glucose ≥ 126 mg/dL (7.0 mmol/L),
- glycated hemoglobin (HbA1c) ≥ 6.5 percent (Diabetes Chronic Complications Trial/United Kingdom Prospective Diabetes Study standardized) or
- A random plasma glucose ≥ 200 mg/dL (11.1 mmol/L) confirmed by one of thefirst two measures.

The following algorithm was suggested combining the recommendations ofthe ADA and IADPSG in 2011 for diabetes in pregnancy [8,38]:

a. Testing of all women at the first antenatal visit <13 weeks – early detectionreduces complications, (OR)
b. Test women who have ANY risk factors:

1) Non-Caucasian
2) BMI > 25 (at risk BMI may be lower in some ethnic groups)
3) History of GDM or prediabetes, unexplained stillbirth, malformed infant
4) Previous baby 4000 gm or more
5) First-degree relative with diabetes mellitus
6) Glucosuria
7) Medications that raise glucose (e.g. steroids, beta mimetics, atypical antipsychotics)
8) Polycystic ovarian syndrome, cardiovascular disease, hypertension, hyperlipidaemia.

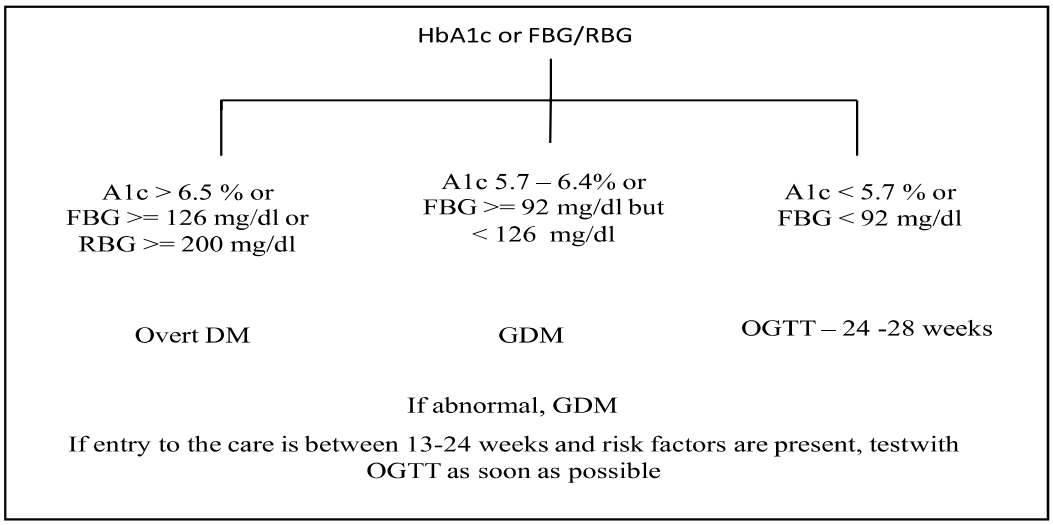


Figure 1: Diagnosis of GDM/Overt DM

The IADPSG recommendations do not specify that the first phase of testing should necessarily be universal rather it recommends that the decision be made on thebasis of the background rate of abnormal glucose tolerance in the local population, aswell as the resources.

Seshiah et al [2] has debated that the ADA/IADPSG suggestions and cut-offs have certain disadvantages:

- The HAPO study was essentially conducted in the Caucasian population (except Bangkok, Hong-Kong). They hypothesize that ethnically Asians have higher insulin resistance in pregnancy which may result in higher blood glucose levels.
- Also, most pregnant women do not come fasting for the antenatal visit. Thus the dropout rate is very high when she is asked to come back for an OGTT, especially in developing countries where the number of antenatal visits are so few.
- Glycosylated Hb is not possible in low resource settings because of its cost and lack of technically qualified staff.

To overcome these problems in the developing countries, the Diabetes in Pregnancy Study Group India (DIPSI) recommended a 'single step' diagnostic procedure for all patients (universal screening).

Gestational Weeks at Which Screening is Recommended

GDM usually develops during late pregnancy (after 1st trimester), however, if screening is delayed till that time, there is a chance that pre-existing diabetes may bemissed. It is ideal to perform the 1st screening as early as possible (during the 1st antenatal check-up). Practically all the pregnant women should undergo screening for glucose intolerance. The usual recommendation for screening is between 24 and 28 weeks of gestation. The recent concept is to screen for glucose intolerance in the firsttrimester itself as the fetal beta cell recognizes and responds to maternal glycemic level as early as 16th week of gestation [36]. If found negative at this time, the screeningtest is to be performed again around 24th – 28th week and finally around 32nd – 34thweek [37].

DIPSI Recommended Method

As a pregnant woman walks into the antenatal clinic in the fasting state, she has to be given a 75g oral glucose load and at 2 hrs a venous blood sample is collected for estimating plasma glucose. This one step procedure of challenging women with 75 gm glucose and diagnosing GDM is simple, economical and feasible [37]. This is themethod which is followed in the current study.

Clarity in Labelling the Different Magnitude of Abnormal Glucose Intolerance on Pregnancy

Increasing maternal carbohydrate intolerance in pregnant women without GDM is associated with a graded increase in adverse maternal and foetal outcomes implying that foetal morbidity starts at a lower maternal glycemic level (< 140 mg/dl). A number of prospective and retrospective studies have substantiated the observation that the frequency of adverse foetal outcome increases with 2hr PG > 120mg/dl and taking care of these women had resulted in a better foetal outcome. Thus, the data is robust and indicates that 2 hr > 120mg/dl needs cognizance. The term 'Impaired Gestational Glucose Tolerance (IGGT)' is used by few authors to indicate pregnant women whose 2 hr PG is > 120mg/dl. It may be appropriate to use the term 'Decreased Gestational glucose tolerance (DGGT)' instead of impaired gestational glucose tolerance. The use of the term 'Decreased' is appropriate as it implies only 'Low' whereas the term 'Impaired' means both high and low.

Further, quiet frequently we come across, labelling any abnormal value in the OGTT not meeting the diagnostic criteria of GDM as IGT [2]. The use of this term 'IGT' during pregnancy may be confusing, as this terminology is also being used in non-pregnant adult with 2 hr PG > 140 mg/dl. This level is also applied to diagnose GDM by WHO criteria.

Hence it may be prudent to label 2 hr plasma glucose value > 140 mg/dl as GDM and a 2 hrplasma glucose value > 120 mg/dl as 'Decreased Gestational Glucose Tolerance' (DGGT). The term IGTshould not be used to denote any abnormal valueduring pregnancy. The table-5 represents the same.

Table 5: With 75 gm OGTT (WHO Criteria)

With 75 gm OGTT (WHO Criteria)		
	In Pregnancy	**Outside Pregnancy**
2 hr >= 200 mg/dl	Diabetes	Diabetes
2 hr >= 140 mg/dl	GDM	IGT
2 hr >= 120 mg/dl	DGGT	-

PREGESTATIONAL DIABETES (OVERT DIABETES)

The increasing prevalence of type 2 diabetes in general, and in younger people in particular, has led to an increasing number of affected pregnancies. Thus, the number of pregnant women with diabetes that was undiagnosed before pregnancy is increasing. Many women found to have gestational diabetes are likely to have type 2 diabetes that has previously gone undiagnosed. In fact, 5 to 10 percent of women with gestational diabetes are found to have diabetes immediately after pregnancy.

Diagnosis of pregestational diabetes

Women with high plasma glucose levels, glucosuria, and ketoacidosis present no problem in diagnosis. Similarly, women with a random plasma glucose level > 200 mg/dL plus classic signs and symptoms such as polydipsia, polyuria, and unexplained weight loss or those with a fasting glucose level exceeding 125 mg/dL are considered by the ADA (2012) to have overt diabetes [8]. Women with only minimal metabolic derangement may be more difficult to identify. To diagnose overt diabetes in pregnancy, the International Association of Diabetes

and Pregnancy Study Groups (IADPSG) Consensus Panel (2010) recommends threshold values for fasting or random plasma glucoseand glycosylated hemoglobin (A1c) levels at prenatal care initiation (**Table-6**) [38]. There was no consensus on whether such testing should be universal or limited to those women classified as high risk. Regardless, the tentative diagnosis of overt diabetes during pregnancy based on these thresholds should be confirmed postpartum.

Risk factors for impaired carbohydrate metabolism in pregnant women include a strong familial history of diabetes, prior delivery of a large new-born, persistent glucosuria, or unexplained fetal losses.

Table 6: Diagnosis of Overt Diabetes in Pregnancy

Measure of Glycemia	**Threshold**
Fasting Plasma glucose	At least 7 mmol/L(126 mg/dL)
Hemoglobin A1c	Atleast 6.5%
Random Plasma glucose	Atleast 11.1 mmol/L (200 mg/dL) plus confirmation
* Apply to women without known diabetes antedating pregnancy. The decision to perform blood testing for evaluation of glycemia on all pregnant women or only on women with characteristic indicating a high risk for diabetes is based on backgroundfrequency of abnormal glucose metabolism in the population and on local circumstances.	

EFFECTS OF PREGESTATIONAL DIABETES MELLITUS ON MOTHERAND FETUS

FETAL EFFECTS

Spontaneous Abortion

Several studies have shown that early miscarriage is associated with poor glycemic control. In an analysis of 127 Spanish women with pregestational diabetes, poor glycemic control, defined by glycohemoglobinA1c concentrations >7%, was associated with a threefold increase in the spontaneous abortion rate [42].

Preterm Delivery

Overt diabetes is an undisputed risk factor for preterm birth. Eidem and associates (2011) analyzed 1307 births in women with pregestational type 1 diabetes from the Norwegian Medical Birth Registry. More than 26 percent were delivered preterm compared with 6.8 percent in the general obstetrical population. Moreover, almost 60 percent were indicated preterm births, that is, due to obstetrical or medicalcomplications.[43]

Malformations

The incidence of major malformations in women with type 1 diabetes is doubled and approximates 5 percent. These account for almost half of perinatal deaths in diabetic pregnancies. In the National Birth Defects Prevention Study, the risk of anisolated cardiac defect was fourfold higher in women with pregestational diabetes compared with the twofold increased risk of non-cardiac defects [44]. The etiology was multifactorial. Three molecular chain reactions have been linked to maternal hyperglycemia and can explain the mechanism behind poor glycemic

control and increased risk for major malformations [45]. These include alterations in cellular lipid metabolism, excess production of toxic superoxide radicals, and activation of programmed cell death. Most common but nonspecific manifestation of diabetic embryopathy is neural tube defect. Most specific but rare manifestation is caudal regression syndrome (figure-2).

Figure 2: Neonate with caudal regression syndrome

All pregestational diabetics should be advised folic acid supplementation. In all pregestational diabetics, the congenital malformation remains as a major cause for pregnancy loss. Congenital malformations are uncommon in GDM since it usually develops in late pregnancy, long after organogenesis is complete. Risk of malformation is similar to that in women with pre-existing diabetes if GDM is diagnosed in the 1st trimester.Table 7: Major birth defects in infants of diabetic mothers (6 -10%)

Table 7: Major birth defects in infants of diabetic mothers (6 -10%)

CNS and Skeletal	CVS	Renal	GIT	Others
Neural tube defects	VSD, ASD	Renal agencies	Duodenal atresia	Single umbilical artery
Anencephaly	Coarctation of aorta	Hydronephrosis	Anorectal atresia	
Microcephaly	Transpositionof great vessels	Double ureter	Omphalocoel e	
Caudal regression syndrome	Situs Inversus	Polycystic kidneys	Tracheoes-ophageal fitsula	
Sacral agenesis	Fallot's tetrology			

Altered Foetal Growth

Diminished growth may result from congenital malformations or from substrate deprivation due to advanced maternal vascular disease. Maternal hyperglycemia prompts foetal hyperinsulinemia, particularly during the second half of gestation. This in turn stimulates excessive somatic growth or macrosomia. Except forthe brain, most foetal organs are affected by the macrosomia that characterizes the foetus of a diabetic woman. Such infants are described as being anthropometrically different from other large-for-gestational age (LGA) infants. Specifically, those whose mothers are diabetic have excessive fat deposition on the shoulders and trunk, which predisposes to shoulder dystocia or caesarean delivery. The incidence of macrosomia rises significantly when mean maternal blood glucose concentrations chronically exceed 130 mg/dL [46].

Unexplained Foetal Demise

The risk of foetal death is three to four times higher in women with type 1 diabetes compared with that of the general obstetrical population [43]. These stillbirths are "unexplained" because common factors such as obvious placental insufficiency, abruption, foetal-growth restriction, or oligohydramnios are not identified. These infants are typically LGA and die before labor, usually after 35 weeks gestation or later. These unexplained stillbirths are associated with poor glycemic control.

Explicable stillbirths due to placental insufficiency also occur with increased frequency in women with overt diabetes, usually in association with severe preeclampsia. In a retrospective analysis of more than 500,000 singleton deliveries, Yanit and associates (2012) found that the foetal death risk was seven fold higher in women with hypertension and pregestational diabetes compared with the threefold increased risk associated with diabetes alone.[47]

Hydramnios

Diabetic pregnancies are often complicated by excess amniotic fluid. According to Idris and co-workers (2010), 18 percent of 314 women with pregestational diabetes were identified with hydramnios, defined as an amnionic fluidindex (AFI) greater than 25 cm in the third trimester [48]. They also found that women with elevated glycohemoglobinA1c values in the third trimester were more likely to have hydramnios.

NEONATAL EFFECTS

Respiratory Distress Syndrome

Historically, newborns of diabetic mothers were thought to be at increased riskfor respiratory distress from delayed lung maturation. Indeed, in their analysis of 19,399 very-low-birth weight neonates delivered between 24 and 33 weeks gestation,

Bental and colleagues (2011) were unable to demonstrate an increased rate ofrespiratory distress syndrome in newborns of diabetic mothers [49].

Hypoglycemia:

Newborns of a diabetic mother experience a rapid drop in plasma glucose concentration after delivery. This is attributed to hyperplasia of the fetal beta-islet cells induced by chronic maternal hyperglycemia. Low glucose concentrations—defined as ≤45 mg/dL—are particularly common in newborns of women with unstable glucose concentrations during labor.

Hypocalcemia:

Defined as a total serum calcium concentration ≤8 mg/dL in term newborns. hypocalcemia is one of the potential metabolic derangements in neonates of diabetic mothers. Theories include aberrations in magnesium–calcium economy, asphyxia, and preterm birth.

Hyperbilirubinemia and Polycythemia:

The pathogenesis of hyperbilirubinemia in neonates of diabetic mothers is uncertain. A major contributing factor is new-born polycythemia, which increases

the bilirubin load. Polycythemia is thought to be a fetal response to relative hypoxia. According to Hay (2012), the sources of this fetal hypoxia are hyperglycemia- mediated increase in maternal affinity for oxygen and fetal oxygen consumption [46]. Together with insulin-like growth factors, this hypoxia leads to increased fetalerythropoietin levels and red cell production.

Cardiomyopathy.

Infants of diabetic pregnancies may have hypertrophic cardiomyopathy that primarily affects the interventricular septum. In severe cases, this cardiomyopathy may lead to obstructive cardiac failure. Russell and coworkers (2008) performe serial echocardiograms on fetuses of 26 women with pregestational diabetes. In the first trimester, fetal diastolic dysfunction was evident compared with that of non diabetic controls. In the third trimester, the fetal interventricular septum and right ventricular wall were thicker in fetuses of diabetic mothers. The authors concluded that cardiac dysfunction precedes these structural changes.[50]

Long-Term Cognitive Development

Finally, results from the childhood Autism Risks from Genetics and the Environment (CHARGE) study indicated that autism spectrum disorders or developmental delay were more common in children of diabetic women [51].

Inheritance of Diabetes

The risk of developing type 1 diabetes if either parent is affected is 3 to 4 percent. Type 2 diabetes has a much stronger genetic component. If both parents have type 2 diabetes, the risk of developing it approaches 40 percent.[1]

MATERNAL EFFECTS

Preeclampsia

Hypertension that is induced or exacerbated by pregnancy is the complication that most often forces preterm delivery in diabetic women. The incidence of chronic and gestational hypertension—and especially preeclampsia—is remarkably increasedin diabetic mothers. In the study cited earlier by Yanit and colleagues (2012), preeclampsia developed three to four times more often in women with overt diabetes. Moreover, those diabetics with coexistent chronic hypertension were almost 12 times more likely to develop preeclampsia [47]. Other risk factors for preeclampsia include any vascular complication and pre-existing proteinuria, with or without chronic hypertension.

Diabetic Nephropathy

Clinically detectable nephropathy begins with microalbuminuria—30 to 300 mg/24 hours. This may manifest as early as 5 years after diabetes onset. Macroalbuminuria—more than 300 mg/24 hours—develops in patients destined to have end stage renal disease. Hypertension almost invariably develops during this period, and renal failure ensues typically in the next 5 to 10 years. The incidence of overt proteinuria is nearly 30 percent in individuals with type 1 diabetes and ranges from 4 to 20 percent in those with type 2 diabetes. Investigators for the Diabetes Control and Complications Trial (2002) reported that there was a 25-percent decrease in the rate of nephropathy for each 10-percent decrease in hemoglobin A1c levels[52].

Diabetic Retinopathy

Retinal vasculopathy is a highly specific complication of both type 1 and type 2 diabetes. The first and most common visible lesions are small micro aneurysms followed by blot haemorrhages that form when erythrocytes escape from the aneurysms. These areas leak serous fluid that creates hard exudates. Such features aretermed benign or background or non- proliferative retinopathy.

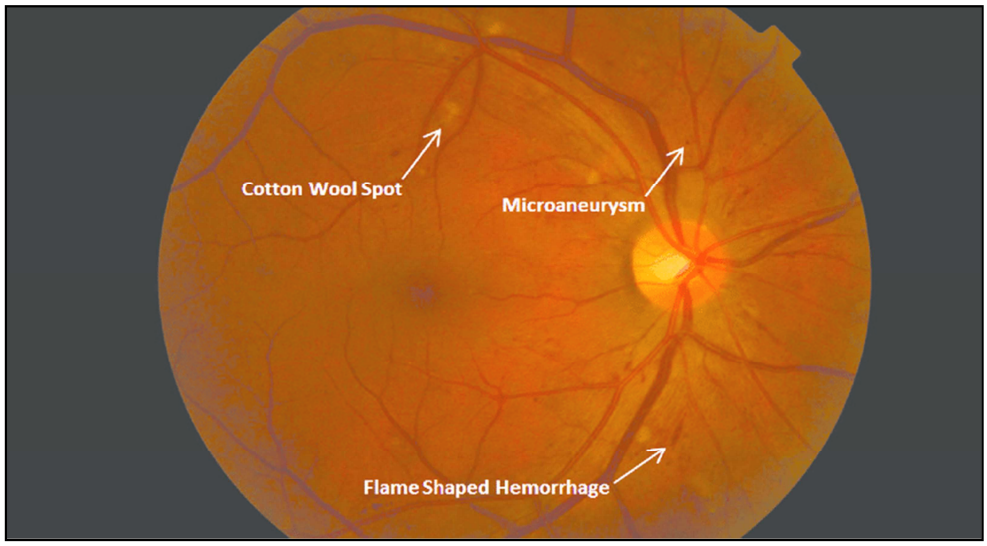


Figure 3: Diabetic retinopathy

With increasingly severe retinopathy, the abnormal vessels of background eye disease become occluded, leading to retinal ischemia and infarctions that appear as cotton wool exudates. These are considered pre-proliferative retinopathy. In responseto ischemia, there is neovascularization on the retinal surface and out into the vitreous cavity. Vision is obscured when there is haemorrhage. Laser photocoagulation before hemorrhage reduces the rate of visual loss progression and blindness by half. The procedure is performed during pregnancy when indicated.

The National Institute for Health and Clinical Excellence (2008) established guidelines recommending that pregnant women with preexisting diabetes should routinely be offered retinal assessment after the first prenatal visit [53].

Diabetic Neuropathy

Peripheral symmetrical sensorimotor diabetic neuropathy is uncommon in pregnant women. But a form of this, known as diabetic gastropathy, is troublesome during pregnancy. It causes nausea and vomiting, nutritional problems, and difficultywith glucose control. This complication is associated with a high risk of morbidity and poor perinatal outcome.

Diabetic Ketoacidosis

It develops in approximately 1 percent of diabetic pregnancies. It is most oftenencountered in women with type 1 diabetes. It is increasingly being reported in women with type 2 or even those with gestational diabetes.

DKA results from an insulin deficiency combined with an excess in counter- regulatory hormones such as glucagon. This leads to gluconeogenesis and ketone body formation. The ketone body β-hydroxybutyrate is synthesized at a much greater rate than acetoacetate, which is preferentially detected by commonly used ketosis detection methodologies. Therefore, serum or plasma assays for β-hydroxybutyrate more accurately reflect true ketone body levels. The incidence of fetal loss can be as high as 20 percent with DKA[1].

Infections

Almost all types of infections are increased in diabetic pregnancies. Common infections include Candida vulvovaginitis, urinary and respiratory tract infections, and puerperal pelvic sepsis.

MATERNAL AND FETAL EFFECTS OF GESTATIONAL DIABETES MELLITUS

The concerns from GDM include:

- Increased perinatal morbidity and mortality
- A mother at high risk for future development of type 2 DM
- The development of metabolic problems- type 2 diabetes in the offspring.

Fetal Macrosomia

Maternal hyperglycemia prompts fetal hyperinsulinemia, particularly during the second half of pregnancy. This in turn stimulates excessive somatic growth. The perinatal goal is to avoid difficult delivery from macrosomia and concomitant birth trauma associated with shoulder dystocia. In a retrospective analysis of more than 80,000 vaginal deliveries in Chinese women, Cheng and associates (2013) calculated a 76-fold increased risk for shoulder dystocia in newborns weighing ≥ 4200 g compared with the risk in those weighing <3500g. [54]

Figure 4: Neonate with Macrosomia

The excessive shoulder and trunk fat that commonly characterizes the macrosomic infant of a diabetic mother theoretically predisposes such infants to shoulder dystocia or caesarean delivery. The HAPO study investigators also reported dramatic increases of cord-serum C-peptide levels with increasing maternal glucose levels following a 75-g OGTT. C-peptide levels >90th percentile were found in almost a third of newborns in the highest glucose categories [11]. Other factors implicated in macrosomia include epidermal growth factor, fibroblast growth factor, platelet-derived growth factor, leptin, and adiponectin.

Neonatal Hypoglycaemia

Neonatal hyperinsulinemia may provoke hypoglycaemia within minutes of birth. The incidence varies depending on the threshold definition. An NIH workshop conference on neonatal hypoglycaemia supported use of such operational thresholdsbut cautioned that these are not strictly evidence-based [46].

Maternal Obesity

In women with gestational diabetes, maternal BMI is an independent and more substantial risk factor for fetal macrosomia than is glucose intolerance. Stuebe and colleagues (2012) completed a secondary analysis of women with either untreated mild gestational diabetes or normal glucose tolerance testing results. They found that higher BMI levels were associated with increasing birth weight, regardless of glucose levels [55]. Also, maternal obesity is an important confounding factor in the diagnosis of gestational diabetes. In their metaanalysis, Torloni and co-workers (2009) estimated that the gestational diabetes prevalence increases by approximately 1 percent for every 1 kg/m^2 increase in BMI. Weight distribution also seems to play a role because the risk of gestational diabetes is increased with truncal obesity. Suresh and colleagues (2012) verified that increased maternal abdominal subcutaneous fat thickness as measured by sonography at 18 to 22 weeks' gestation correlated with BMI and was a better predictor of gestational diabetes.[56]

MANAGEMENT

Guiding Principles

All pregnant mothers who test positive for GDM for the first time should be started on Medical Nutrition Therapy (MNT) for 2 weeks. After 2 weeks on MNT, a 2 hrs post prandial blood sugar (PPBS) should be done. If 2hr PPBS<120 mg/dL, repeat test every 2 weeks in second trimester & every week in third trimester. If 2hr PPBS

≥120mg/dL medical management (Insulin Therapy) to be started. A team approach is ideal for managing women with GDM. ACOG 2001 recommends administration of insulin if FBS ≥ 95mg/dL or 1 hr PPBS >130-140 mg/dL or 2 hr PPBS ≥120 mg/dL.

Medical Nutrition Therapy (MNT)

All pregnant women with GDM should get Medical nutrition therapy (MNT) as soon as diagnosis is made. MNT for GDM primarily involves a carbohydrate controlled balanced meal plan which promotes Optimal nutrition for the mother and the fetus. Nutrition assessment in GDM should be individualised to allow an accurate appraisal of the woman's nutritional status. This assessment includes defining her Body Mass Index (BMI) or percentage of desirable pre-pregnancy bodyweight and optimal pattern of weight gain during pregnancy.

Thus, GDM is managed initially with MNT and if it is not controlled with MNT,insulin therapy is added to the MNT.

a) Meal pattern: It should provide adequate calories and nutrients to meet the needs of pregnancy. The expected weight gain during pregnancy is 300 to 400 gm/week and total weight gain is 10 to 12 kg by term. Approximately 30 to 40 Kcal/kg ideal body weight or an increment of 300 kcal/day above the basal requirement is needed.

b) Calorie Counting: Pregnant diabetic woman are advised to distribute their calorie consumption especially the breakfast. This implies splitting the usual breakfast into two equal halves and consuming the portions with a two hour gap in between. By this the undue peak in plasma glucose levels after ingestion of the total quantity of breakfast at one time is avoided. The total calorie requirement should

consist of 45% carbohydrate, 30% protein and 25% fat (mainly unsaturated fats)

c) Exercise: Around 30 minutes of mild to moderate exercise daily helps in improving glycemic control by improving insulin sensitivity at the skeletal muscle level. This reduces overall insulin requirement. Light exercise in the form of walking especially after a meal helps reduce postprandial glucose levels.

Insulin Therapy

Insulin is essential if medical nutrition therapy fails to achieve euglycemia. Various criteria have been proposed for the initiation of insulin therapy. Fourth International Workshop on GDM recommended lowering capillary blood glucose concentration to 140 mg/dl at 1 hour and 120 mg/dl at 2 hours[29], Whereas ADA recommended the option of measuring 1 hour post meal values with cut off of 120mg/dl [8]. If the FPG concentration on the OGTT is >120mg/dl, then the patient is started on insulin immediately along with MNT. Other GDM women are seen within 3days and are also taught self-monitoring of blood glucose (SMBG). SMBG is to be performed in fasting and 1 ½ hours after each meal. Insulin is started within 1 to 2 weeks, if the majority (i.e., at least four of seven per week) of fasting values exceed 90 mg/dl. Similarly, if the majority of post prandial values after a particular meal exceed 120 mg/dl, insulin is started.[21]

Insulin therapy has remained gold standard in the treatment of GDM and pregestational diabetes. Most associations recommend the use of short acting regular insulin (onset of action 30 minutes lasting for 6–8 hours) and intermediate acting NPH insulin (onset of action 1 hour, lasting for 10–14 hours). Recent research has added newer rapid acting insulin lispro and aspart whose action begins within 15 min. The different types of insulin available are mentioned in table-8.

Table 8: Types of Human Insulin and Insulin Analogues

	Source	Onset (hr)	Peak (Hr)	Duration (hr)
Short Acting				
Humulin R (Lilly)	Human	0.5	2-4	5-7
Velosulin H (Novo Nordisk)	Human	0.5	1-3	8
Velosulin R (Novo Nordisk)	Human	0.5	2.5-5	6-8
Lispro	Analog	0.25	0.5-1.5	4-5
Aspart	Analog	0.25	1-3	3-5
Intermediate Acting				
Humulin Lente (Lilly)	Human	1-3	6-12	18-24
Humulin NPH (Lilly)	Human	1-2	6-12	18-24
Novolin I (Novo Nordisk)	Human	2.5	7-15	22
Novolin N (Novo Nordisk)	Human	1.5	4-20	24
Long Acting				
Humuline Ultralente (Lilly)	Human	4-6	8-20	> 36
Glargine	Analogue	1	-	24
Detemir	Analogue	1-2	-	24

The requirement for insulin increases with gestational age, glycaemic control, obesity and other factors. Dose of insulin varies from 0.6–1U/kg/day in divided doses depending on the trimester of pregnancy. ACOG recommends insulin therapy for those who are not able to achieve the gylcemic targets mentioned previously with MNT. Commonly used formula for insulin therapy is:

Total daily insulin requirement= 2⁄3 in the morning+ 1⁄3 at night such that Morning dose=2⁄3 NPH+ 1⁄3 short acting;

Predinner dose= 1⁄2 NPH + 1⁄2 short acting

According to NHM GUIDELINES 2014, Injection Human premix insulin 30/70 is to be administered with a 40 IU Insulin syringe – 40 IU.[57]

Target Blood Glucose Levels

Maintenance of Mean Plasma Glucose (MPG) level ~105 mg% is ideal for good fetal outcome[22]. This is possible if FPG and post prandial peaks are around 90 mg/dl and 120 mg/dl respectively (MPG should not be < 86mg/dl as this may cause small for gestational age infants).

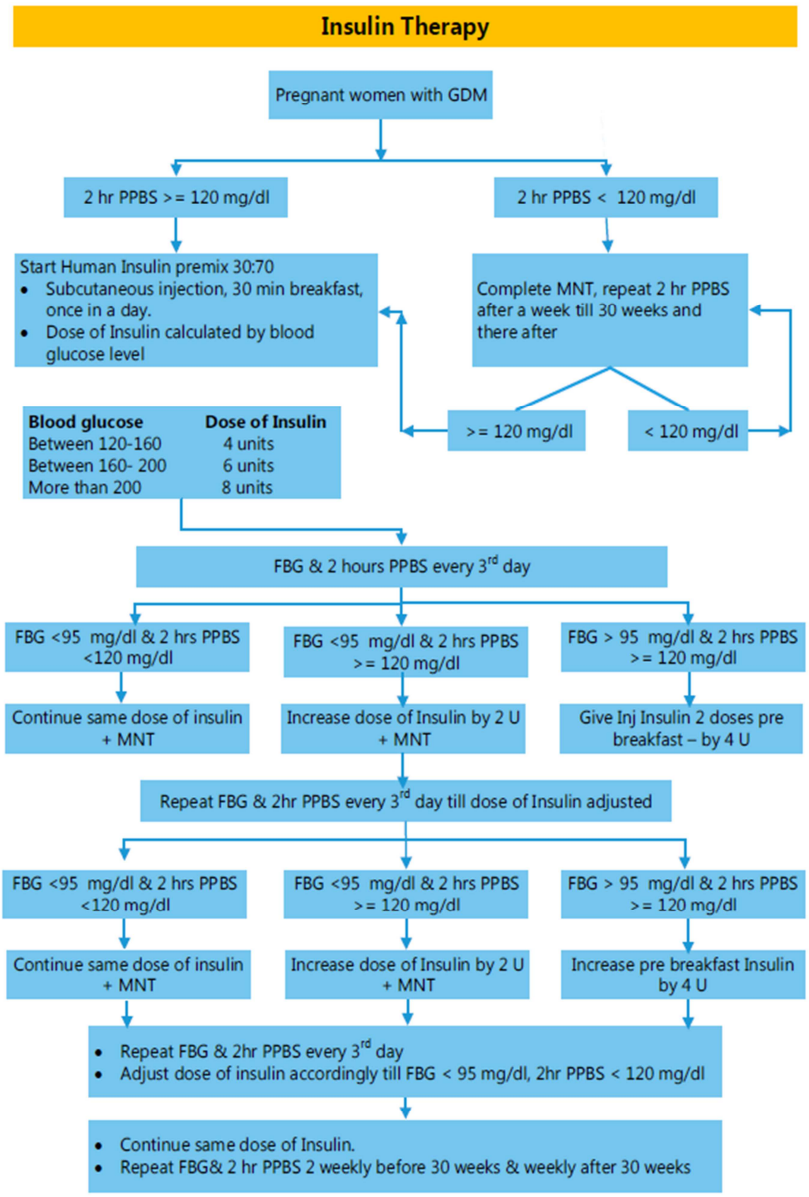


Figure 5: National guidelines for Insulin therapy

Oral Anti-diabetic Drugs

Recently reports have shown good fetal outcome in GDM women who were on glyburide (micronized form of Glibenclamide). Metformin has been found to be useful in women with polycystic ovarian disease (PCOD) who failed to conceive. Continuing this drug after conception is still a controversy. Currently, oral agents arenot routinely recommended during pregnancy. Both these drugs are considered as category B drugs. The ADA and ACOG do not endorse the use of these drugs. NICE guidelines endorse the safety of use and efficacy of these 2 drugs, but adds that informed consent should be obtained before initiating or continuing these drugs.

OBSTETRIC CONSIDERATIONS

Fetal Surveillance in Diabetes

First Trimester

Early ultrasound for confirmation of viability and dating must be done. This helps in later confirmation of gestational age in preterm cases or for planning time of delivery. First trimester screening including maternal serum PAPP-A and βHCG with ultrasound evaluation for detecting chromosomal anomalies in the fetus must be offered between 11 and 14 weeks of gestation (NICE guidelines) Nuchal translucency (NT) >3.5mm is associated with cardiac anomalies in chromosomally normal fetuses. Cardiac anomalies are commonly associated with diabetes. Hence NT is an important marker. Also, an early scan at 11–12 weeks can detect neural tube defects which areseen in diabetes.

Second Trimester

A detailed anomaly scan must be done between 18 and 20 weeks of gestational age. Maternal serum alpha protein levels are increased in neural tube defects. However, there is no added benefit if the anomaly scan is normal.

Fetal echocardiography at around 24 weeks as a routine in all diabetic mothers may not be cost effective. Major cardiac anomalies can be detected (especially those of the conotruncal septum) by the 4 chamber and outflow tract view on ultrasound. Thus, fetal echocardiography may be justified in women with diabetes with increased nuchal translucency in the first trimester with a normal karyotype or when a cardiac anomaly is suspected in the anomaly scan or when the cardiac visibility is restricted on ultrasound due to maternal obesity or in pregestational diabetes mellitus with poorglycemic control in the first trimester.

In cases of diabetes complicated with hypertension or restricted fetal growth, an umbilical artery Doppler should be done for assessing uteroplacental insufficiency. In such cases, Doppler studies help to predict perinatal outcome and plan subsequent management. In the absence of risk factors, umbilical artery Doppler is ineffective inpredicting adverse fetal outcome.

Third Trimester

The NICE guidelines suggest 4 weekly monitoring of fetal growth from 28–36weeks. This would help to detect macrosomia and polyhydramnios. The ACOG suggests that accuracy of ultrasound biometry for suspected macrosomia is similar toclinical palpation by Leopold's manoeuvres.

Diabetic mothers must be explained the importance of daily fetal movement counts (DFMC) which is easy, inexpensive and non-invasive method for fetal well- being. Any reduced movements must be immediately reported and investigated further. Presence of polyhydramnios sometimes poses difficulty in perceiving fetal movements.

Biophysical profile takes into consideration fetal movement, breathing, tone, amniotic fluid largest vertical pocket and nonstress test. A good BPP score is reassuring. However, it cannot predict adverse perinatal outcome or sudden fetal death.In case of hypertension or vasculopathy, depending on the severity, the frequency of these tests will vary. If pregnancy goes beyond 38 weeks, then weekly orbiweekly intense fetal monitoring with modified BPP must be done.

TIMING OF DELIVERY

a. Induction of labour

Sudden intrauterine fetal demise in the third trimester of diabetic pregnancy is common. To avoid this risk, preterm delivery is recommended. But with this, respiratory distress syndrome (RDS) is likely to occur.

Some centres allow women with uncomplicated diabetes to go into spontaneous labor irrespective of the gestational age, but most still advocate delivery at 38 weeks as perinatal mortality and morbidity appear to increase after this time. Some state that uncomplicated cases of gestational diabetes (not pregestational) may be allowed to wait for spontaneous labour up to 40 weeks with adequate fetal surveillance. NHM recommends delivery after 39 weeks. If a Pregnant mother with GDM with well controlled plasma glucose has not delivered

spontaneously, induction of labour should be scheduled at or after 39 weeks pregnancy.

The main concern with vaginal delivery is the risk of shoulder dystocia due to macrosomia. However, it has been seen that 50% of cases with brachial plexus palsy occur in absence of shoulder dystocia. The ACOG and NICE guidelines suggest that vaginal delivery is not contraindicated for suspected macrosomia unless the estimated fetal weight is >4.5kg. In the Indian population, the average baby weight is less.

Balaji et al in their study on the diagnosis of GDM in Indian women considered birth weight of 90th centile, which is 3.45kg, as macrosomia [57], vaginal delivery should be allowed after a proper assessment of pelvic adequacy. Depending on the bishops score, labour can be induced using mechanical methods, prostaglandins or oxytocin. Suspected macrosomia is also not a contraindication for allowing vaginal birth after previous caesarean.

b. Elective caesarean section.

This is indicated when there is string suspicion of macrosomia which may leadto shoulder dystocia and an associated complication like severe pre-eclampsia. In the Indian context, when the average birth weights are less in nondiabetic women, there isno clear cut-off fetal weight at which an elective caesarean section or induction of labour be offered to minimise risk of shoulder dystocia. Taking the average weight ofIndian babies, $\geq$ 3500 g can be considered as macrosomia.

INTRA PARTUM MANAGEMENT

If labour is to be induced in GDM, the usual evening insulin dose should be taken the night before, but no subcutaneous insulin is given the following morning when induction begins. Once labour begins, insulin is not necessary. In gestational diabetics the requirement of insulin is likely to fall precipitously and no insulin may be required immediately after expulsion of placenta. The pregnant women should bestarted on 2 hourly monitoring of plasma glucose. The aim is to maintain blood glucose levels between 72-126mg/dl. It is important to monitor vitals and fluid intake and output, urinary ketones with blood glucose levels 1–2 hourly. IV infusion with normal saline (NS) to be started & regular insulin to be added according to blood glucose levels as per Table-9.

Table 9: Dose of regular insulin according to blood glucose levels

Blood glucose level	Amount of Insulin added in 500 ml NS	Rate of NS Infusion or RL
60-90 mg/dl	5% DNS	100ml/hr
90 - 120 mg/dl	0	100 ml/hr (16 drop/min)
120 -140 mg/dl	4 U	100 ml/hr (16 drop/min)
140 - 180 mg/dl	6 U	100 ml/hr (16 drop/min)
> 180 mg/dl	8 U	100 ml/hr (16 drop/min)

DELIVERY

As soon as the infant is born, the following actions are mandatory:

1. Early clamping of the cord, i.e. within 20 seconds of delivery, to avoid erythrocytosis;
2. Evaluate vital signs; Apgar scores at 1 and 5 minutes;
3. Clear oropharynx and nose of mucus; later empty the stomach - be aware that stimulation of the pharynx with the catheter may lead to reflex bradycardia and apnoea;
4. Avoid heat loss, keep neonate warm, transfer to incubator pre-warmed to 34^{o}C;
5. Perform a preliminary physical examination to detect major congenital malformations;
6. Start early feeding, preferably breast milk, at 4-6hours after delivery: aim at full caloric intake (125kcal/kg/24 hours) at 5 days, divided into six to eight feeds a day;

7. Capillary blood glucose should be monitored at 1 hour of age and before the first four breast feedings (and for up to 24 hours in high- risk neonates). The cut-off of 45mg% is now currently used as the working definition for hypoglycaemia.

POST-PARTUM MANAGEMENT

1. In the postpartum period, the insulin requirements drop and so the dosage of the medications will need to be changed. Most women with GDM can be taken off medications and managed on diet alone. It is important to maintain good glycemic control in the post partum period
2. Life-style modifications and diet are emphasised in the post partum period and at discharge.
3. Breast feeding is the preferred option for all GDM and pregestational diabetes women.
4. Along with nutritional and immunological advantages, breast feeding has been associated in the general population with a reduction in the rates of childhood obesity.

PRECAUTIONS DURING CAESAREAN

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

- After appropriate consent and blood availability, a light meal and night dose of insulin are given.
- Elective section of a diabetic patient should preferably be performed as the first case in the morning as the patient is fasting.
- Morning insulin dose is skipped and fasting glucose level should be recorded.

- If required sliding scale of insulin can be started and continued in the postoperative period.
- Severely obese may require thromboprophylaxis.
- Special precautions while performing the caesarean are adequate incision to allow delivery of the big baby, use of forceps to deliver high floating head, to check for and suture extensions of the uterine incision which may take place especially while performing a second stage caesarean.
- Postoperative glucose monitoring must be continued and patient must be mobilized as early as possible.

FOLLOW UP

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

The Cut offs for normal blood glucose values are:

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

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

CONTRACEPTIVE ADVICE

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

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

MATERIALS AND METHODS

STUDY SETTING:

"KAHER's Dr.Prabhakar Kore Charitable Hospital and MRC", Belagavi attached to Jawaharlal Nehru medical College, Belagavi

STUDY DESIGN:

A cross-sectional prospective study

STUDY PERIOD:

1st January 2020 to 31st March 2021.

SOURCE OF DATA:

All the pregnant women attending, the obstetric unit of "KAHER's Dr.Prabhakar Kore Charitable Hospital and MRC", Belagavi who were diagnosed as GDM as per the institutional protocol (IADPSG criteria) and who gave written informed consent were enrolled in the study.

SELECTION CRITERIA:

Inclusion criteria-

- Antenatal women diagnosed with GDM attending the teaching hospital –attached to KLE University's J.N. Medical College, Belagavi.

Exclusion criteria-

- Overt diabetes
- Non diabetic women

ETHICAL CLEARANCE:

Approved by "Ethical and Research committee, KAHER's Jawaharlal Nehru Medical College" Belagavi, prior to its commencement. (Annexure-1)

INFORMED CONSENT:

All participants fulfilling the selection criteria were explained regarding the study purpose in their own vernacular language and written informed consent was obtained prior to their enrollment in the study.

SAMPLE SIZE:

Sample size calculation based on prevalence is

$$n = \frac{z_\alpha{}^2 P(1-P)}{d^2}$$

where P is the percentage of prevalence and d is the percentage likely difference in the prevalence.

z_α is linked with the level of significance.

For 5% level of the significance $z_\alpha = 1.96$.

With P = 69.39% and d = 10% of P, the sample size is 169

To make the study more confirmative, the sample size can be raised.

Since the present study is an observational study, there will be a single group with the above sample size

DATA COLLECTION

All antenatal women diagnosed with GDM as per the IADPSG criteria, as per institutional protocol for diagnosis of GDM will be included in study.. The demographic and baseline data of the study participants were recorded. The antenatal history, present and past history, presence or absence of other disorders like hypertension, thyroid disorder, pre-gestational diabetes were noted. Detailed general examination and obstetric examination were carried out. In addition to all the basic obstetric investigations, HBA1C test was carried out. Each woman had a data file and a serial number that was used for data entry in her subsequent clinic visits and delivery. Data was entered (from data files and questionnaires) to the final electronic database using unique identification number . Obstetric ultrasound was performed as per the routine recommendations (dating, target ultrasound and growth scan) and also if indicated at any time during antenatal and intranatal period. The management of GDM was carried out by a multidisciplinary team of endocrinologist, obstetrician and neonatologist. Dietary modification through education and motivation is given priority in every clinic visit with one-to-one counselling. Medical nutrition therapy (MNT) was advised by a dietician , in accordance with their socio-cultural background. Blood glucose monitoring by regular 7-point blood sugar series (BSS) that includes pre meal and 2-hour post meal capillary blood glucose by using glucometer in or else venous plasma glucose testing for 24 hours by hospital admission. Once the women had euglycemia with therapy, she was discharged and followed up outpatient. On each antenatal visit, blood glucose was further estimated and the GDM was managed as per the endocrinologist's opinion. When diet and exercise both fail to achieve the target glycaemia, metformin or insulin therapy were initiated. The obstetric management included regular reviews and focused

antenatal care by the obstetric team at the same clinic. For obstetric management, national guidelines and departmental protocol on management of diabetes in pregnancy were used. The outcome of pregnancy and presence or absence of antenatal, intranatal and postnatal complications were recorded. All women were assessed by the specialist obstetrician at 36 weeks of gestation to plan for timing and mode of delivery. Women with good glycemic control and requiring MNT alone were planned to await spontaneous onset of labour up to 40 weeks of gestation. For the others, induction of labour or caesarean delivery was planned at 38-39 weeks of gestation depending on patients glycemic control and individual obstetric factor.

STATISTICAL ANALYSIS:

For the continuous quantitative variables mean and standard deviation will be calculated. The inter group continuous variables will be compared using suitable tools of statistics like unpaired student's t test. Two quantitative variables, within a group, will be compared using student's paired t test.

Discrete variables will be represented by median. Suitable graphs will be used to depict the comparison.

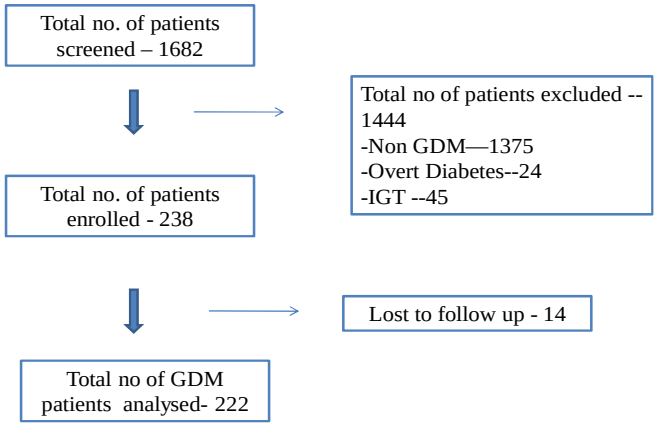
For all the tests, p value less than 5% (0.05) will be considered significant.

RESULTS

A prospective observational study was carried out in women attending obstetric clinic and diagnosed as GDM at KAHER'S Dr Prabhakar Kore hospital attached to JNMC during the period from January 2020 to March 2021. .Data was collected, analyzed and tabulated.

In total 222 women with GDM were analyzed.

Flow chart



In this study, 1682 number of women were screened and 238 women were enrolled for study who met inclusion criteria. A total of 1444 women were excluded, out of which 24 were diagnosed as overt DM, 45 were labelled as IGT and 1375 women were Non GDM.

Prevalence of GDM is usually high in Indian population due to its ethnicity and other factors like rapid urbanization and sedentary lifestyle. Prevalence of GDM was 14% in this study.

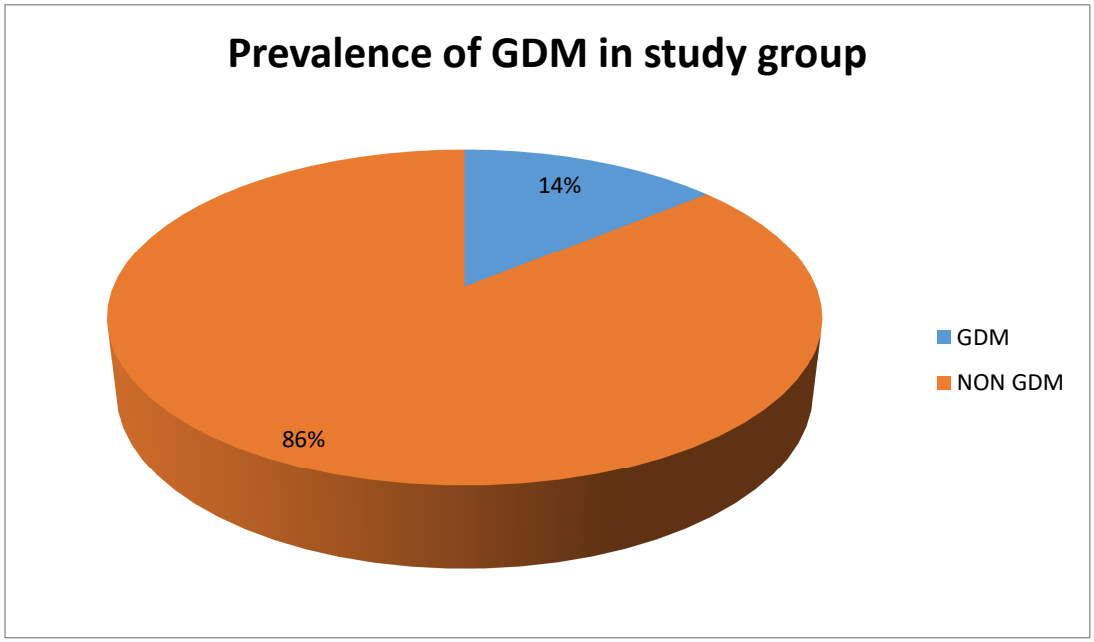


Figure - 6 prevalence of GDM in women attending the hospital during study period

Table 10 - Maternal socio demographic characteristics of study participants in women with GDM

CATEGORY	SUB-CATEGORY	NUMBER	PERCENTAGE
REGISTRATION STATUS	REGISTERED	131	59.11
	UN REGISTERED	91	40.88
AGE(YRS)	<20	9	4.04
	20-25	60	27.02
	26-30	94	42.22
	>30	59	26.5
RESIDENCE	RURAL	80	36.23
	URBAN	142	63.77

Most of the women in the study group were registered cases accounting for 60% which shows a good follow up of participants and most of the study participants i.e 94 participants were in the age group of 26yrs-30yrs. with mean age of incidence was 28 yrs showing that GDM was common in advanced maternal age group .and 63% women were from urban area.

Below Figure 7 and Figure 8 shows distribution of study participants according to age group and registration status respectively.

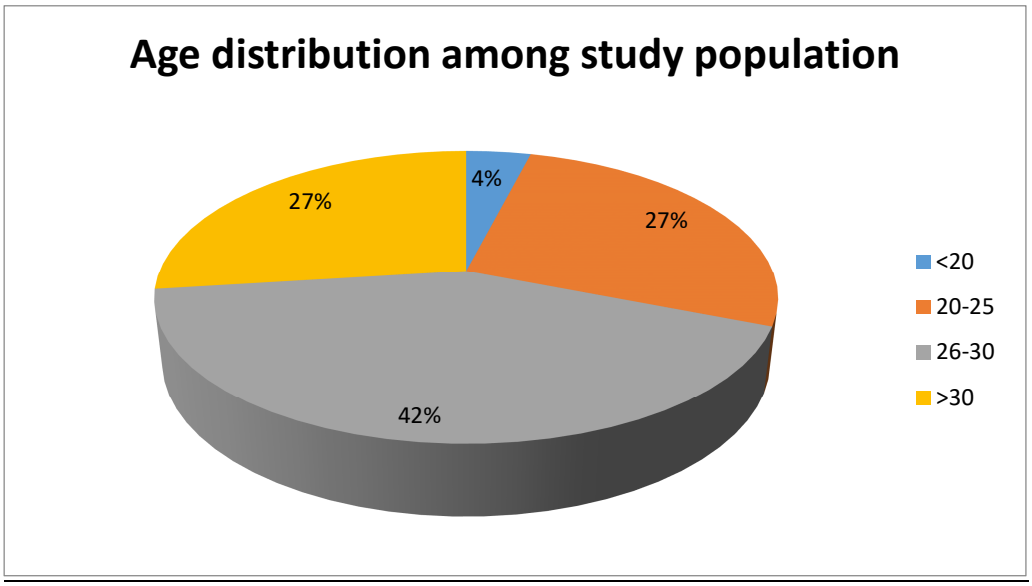


Figure 7 - Distribution of age among study population

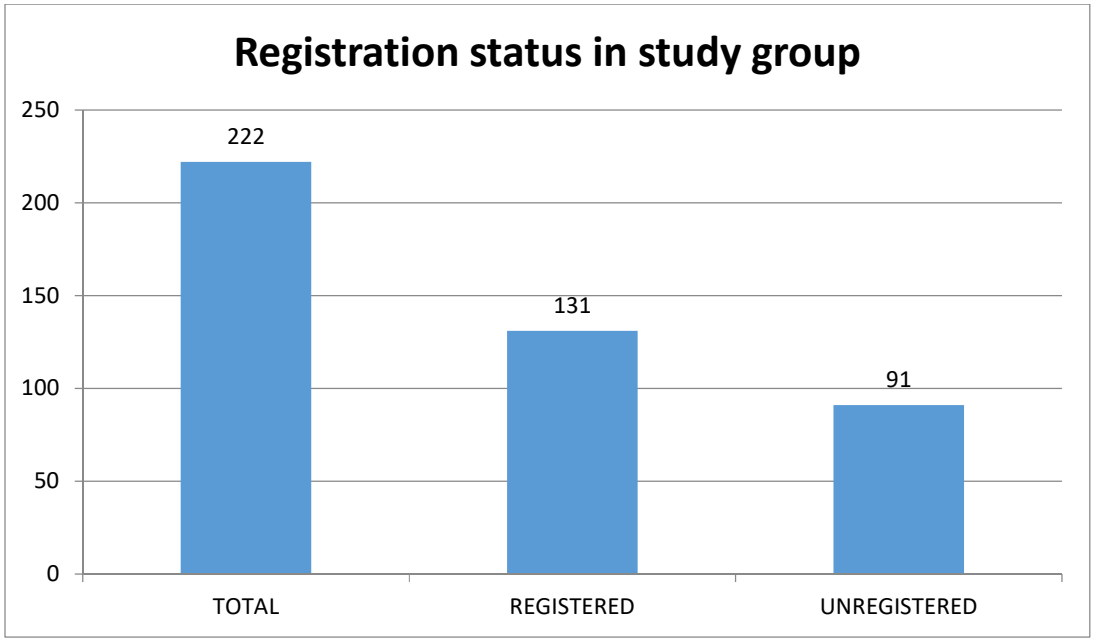


Figure 8 – Division of women according to Registration status

Table 11 - Clinical characteristics of study population among women with GDM

CATEGORY	SUB-CATEGORY	NUMBER	PERCENTAGE (%)
Obstetric index (GRAVIDA)	1	90	40.33
	2	64	28.8
	3	35	15.7
	>3	33	14.8
PAST H/O	GDM	36	23.23
	BIG BABY	16	10.32
	ABORTIONS	59	32.7
	IUD	8	4.4
	PRETERM LABOUR	6	3.3
FAMILY H/O DM		30	19.35
H/O of HYPOTHYROIDISM		42	18.44

Our study shows among women with GDM, 40% were primigravidas and 60% were multigravidas. As per the results shown from the data of past history, absence of GDM in the past history doesn't exclude its occurrence in the present pregnancy as 76% of cases were not diagnosed cases of GDM in their previous pregnancies. 10% of women had delivered big baby in previous pregnancy(>3.5kg),32% women had abortions in previous pregnancies and 6 participants had preterm labour As GDM is more commonly associated with positive family history ,around 20% of GDM cases were having positive family history of diabetes mellitus. H/o of hypothyroidism was seen in 18% cases showing presence of dual endocrinopathy in cases with GDM.

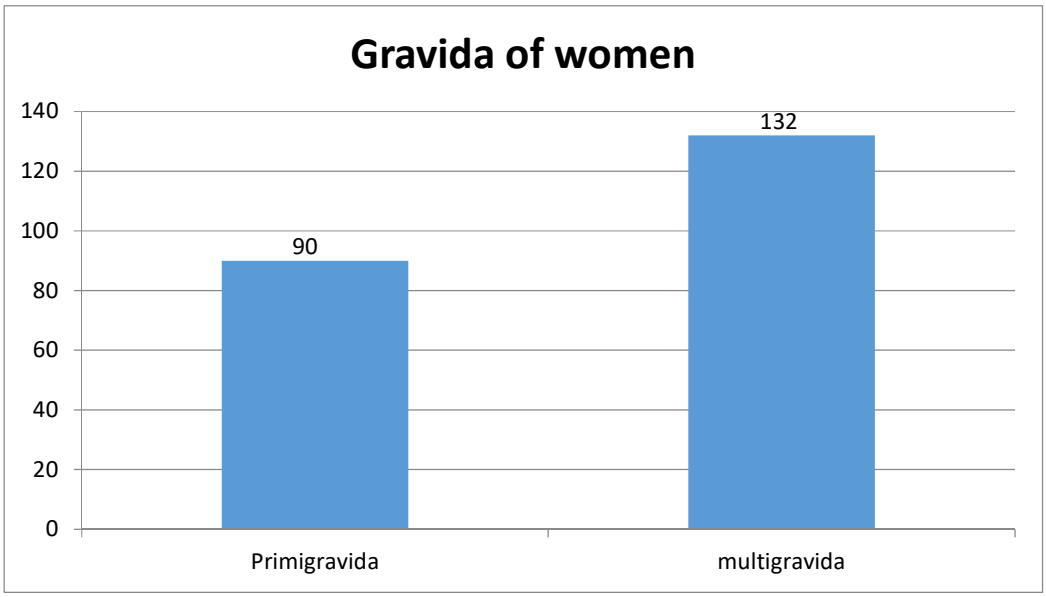


Figure 9 –Distribution according to gravid status of women

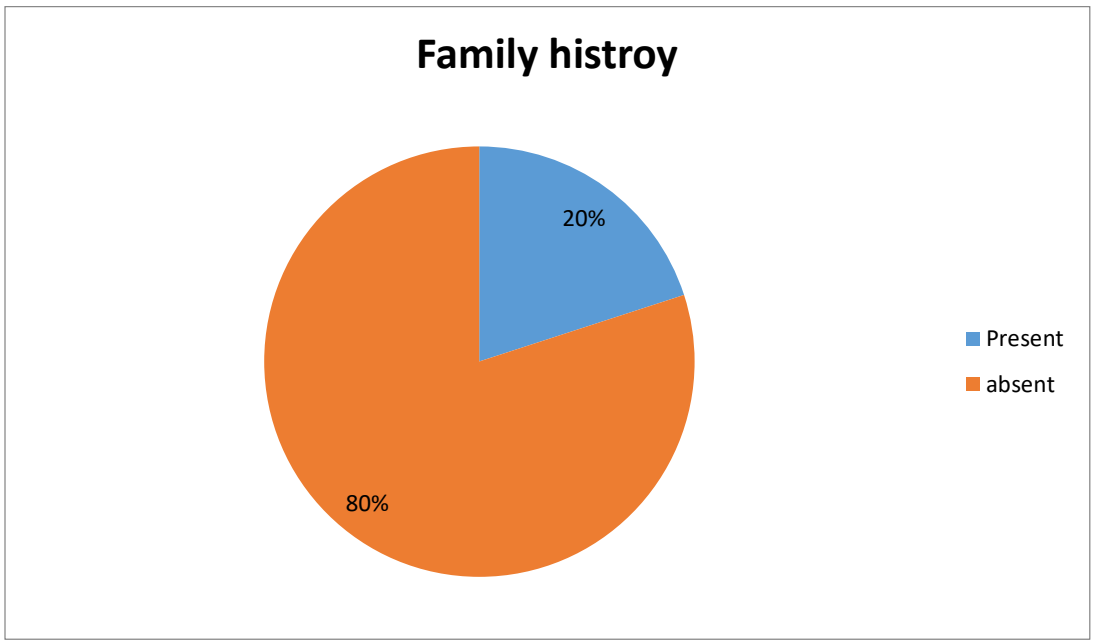


Figure 10 - Correlation of positive family history with GDM

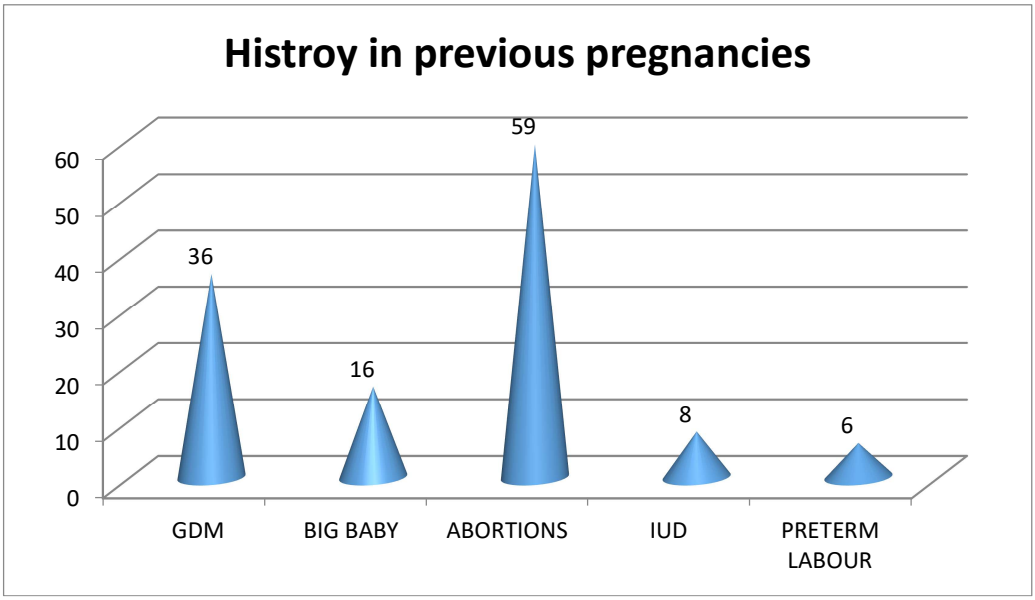


Figure 11 - Distribution according to history of previous pregnancies

Table 12 – BMI of women with GDM

BMI(Kg/m2)	NUMBER	PERCENTAGE
<18.5	6	2.7
18-24.9	51	22.8
25-29.9	149	67.1
>30	16	7.2

As shown in table, most of the cases were in the range of overweight BMI accounting to 67%, GDM is found most commonly associated with higher BMI Around 74% of GDM women were with BMI greater than 25 kg/sqmt.

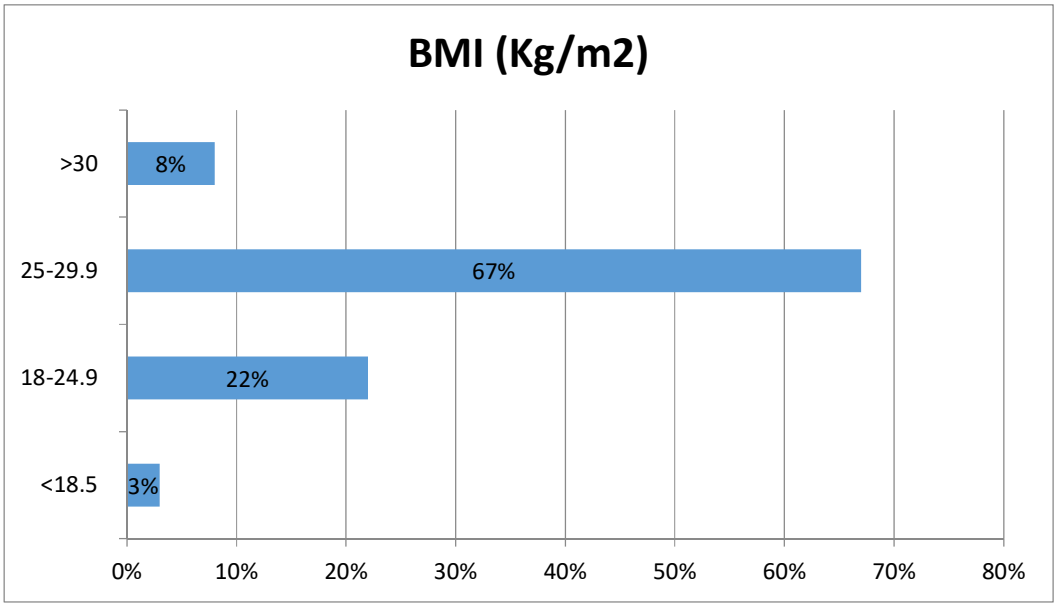


Figure 12 -Distribution according to BMI of women with GDM

Table 13-Distribution according to gestational age at diagnosis

GESTATIONAL AGE(WEEKS)	NUMBER	PERCENTAGE
<12	8	3.6
12-24	83	37.3
24-28	115	51.8
>28	16	7.2

In our study, most of the women (51%) were diagnosed at gestational age of around 24wks -28 wks where insulin resistance will be at its maximum.

Table 14 - Distribution according to mode of management

MANAGEMENT	NUMBER	PERCENTAGE
DIET	163	76.6
DIET +INSULIN	48	21.6
DIET+ORAL HYPOGLYCAEMIC DRUGS	2	0.9

Most of the GDM cases were managed with diet alone accounting to 77%,22% of cases needed step up of management by controlling sugars with insulin and 2 cases were on OHA with tab metformin who were unregistered cases.

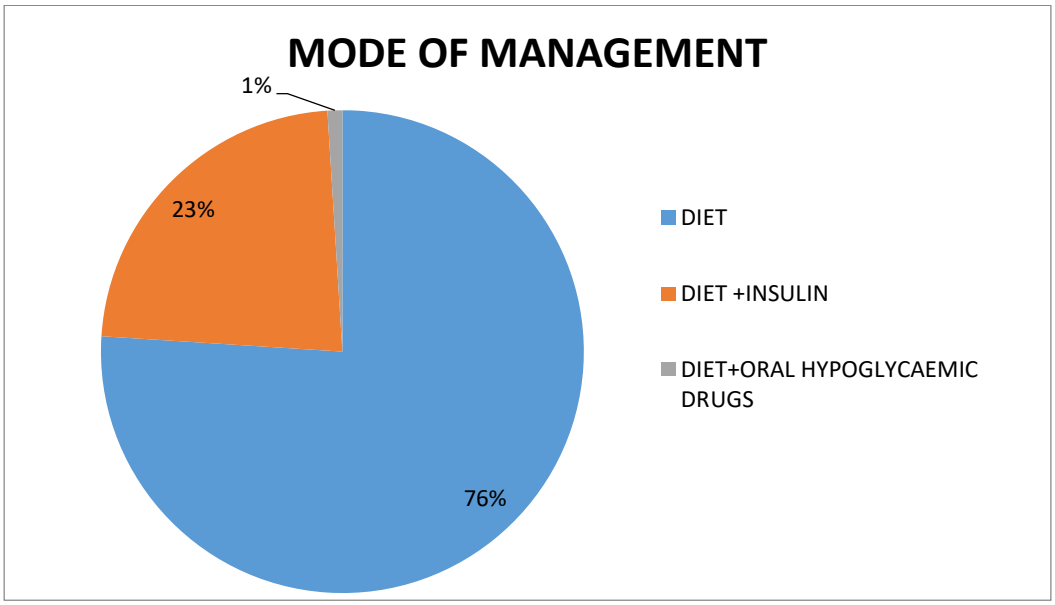


Figure 13 -Distribution according to mode of management in women with GDM

Table 15-Maternal complications and outcome among women with GDM

COMPLICATION		NUMBER	PERCENTAGE
PIH		38	18.89
PRE-TERM LABOUR		9	4.1
PROM		22	9.4
POLYHYDROAMNIOS		13	6.1
INFECTIONS	Urinary tract infections	18	8.1
	Vaginal infections	10	4.5
ABORTIONS		0	0
PPH		2	1.1
PURPUERIAL SEPSIS		0	0

As we know GDM is associated with adverse maternal outcome as shown in table, 50% of women with GDM had composite adverse maternal outcome. PIH is seen to the major adverse outcome in our study accounting for 19% .Followed by PROM seen in around 9 women ,infections was seen in 8% of women of which 18 women had UTI and main organism cultured was E coli and 10 women had vulvovaginitis of which candida was the main organism seen. No abortions were encountered in the study group ,2 women had PPH who were managed medically.

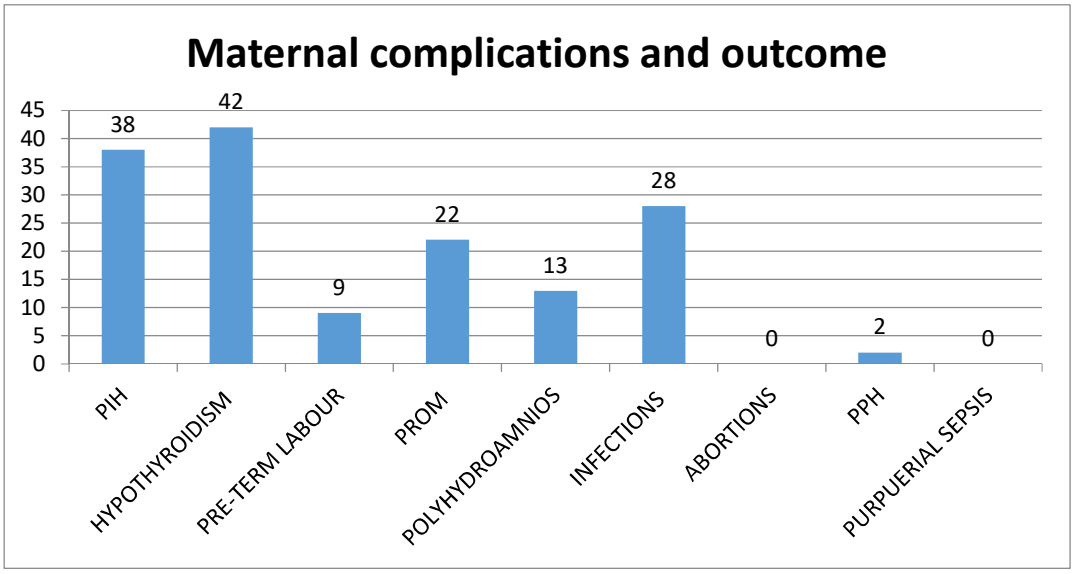


Figure 14-Distribution according to maternal complications

Table 16-Distribution according to gestational age at delivery

GESTATIONAL AGE(WEEKS)	NUMBER	PERCENTAGE
<32	2	0.9
33-36	20	9
37 – 40	184	82.8
>40	16	7.2
TOTAL	222	100.00

The above table shows, most women delivered at gestational age of 37-40wks.

Table17-Distribution according to mode of delivery

MODE	SUBCATEGORY	NUMBER	PERCENTAGE
VAGINAL	SPONTANEOUS	28	12.6
	INDUCED	16	7.6
	INSTRUMENTAL	4	1.3
CAESAREAN SECTION	ELECTIVE	56	25.8
	EMERGENCY	118	53.1

Among the study population, 78% women underwent LSCS in comparison to 22 % women who delivered vaginally out if which 4 had instrumental delivery (ventouse)

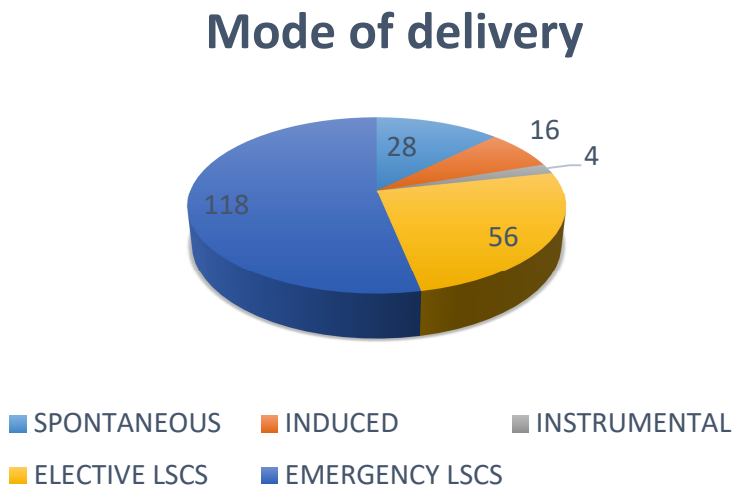


Figure 15 -Distribution according to mode of delivery

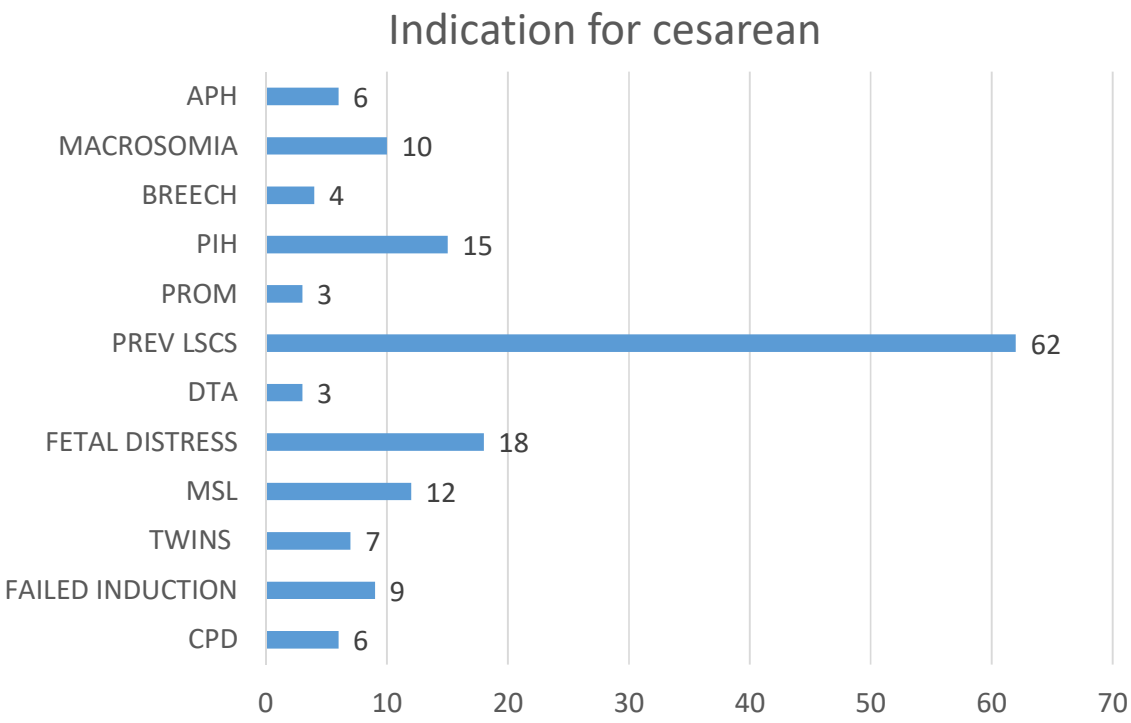


Figure 16- Indications for caesarean section among women with GDM

Previous LSCS was the most common indication among women with GDM who underwent LSCS accounting to 35% and this serves as main reason for which caesarean rate was high among women with GDM in our study.

Table 18-Distribution according to Baby characteristics

CATEGORY	SUB CATEGORY	NUMBER	PERCENTAGE
SEX	MALE	117	52.7
	FEMALE	104	47.2
BIRTH WEIGHT	<2KG	19	8.5
	2-2.5KG	38	17.1
	2.6-3KG	104	46.8
	3.1-3.5KG	48	21.6
	3.6-4KG	6	2.66
	≥4KG	0	0

In our study, 52% were male babies and 47% babies weight were in the range of 2.6-3kgand 3% babies weight was more than 3.6kg

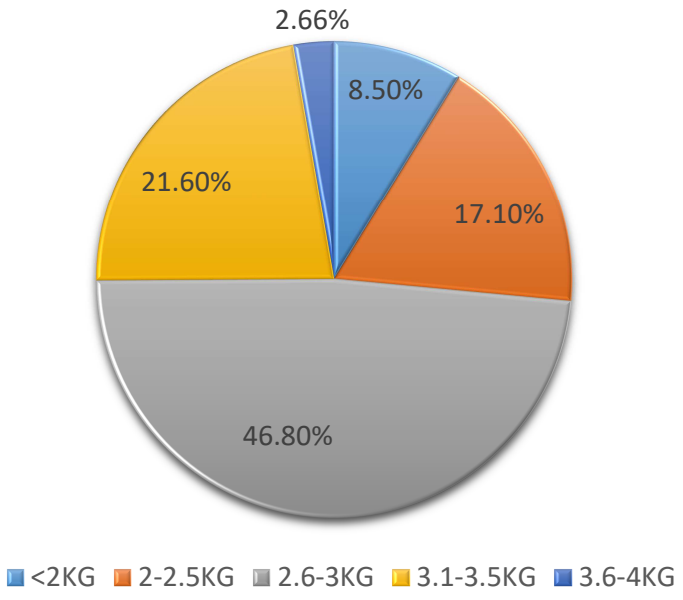


Figure 17- Distribution according to birth weight

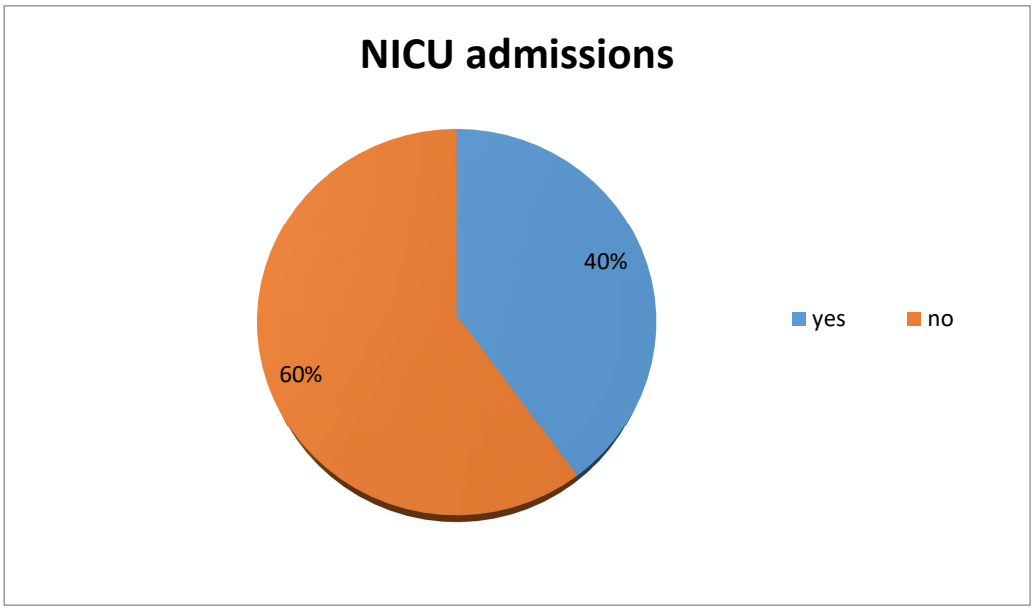


Figure 18- NICU admissions among babies delivered to GDM women

In our study, total of 86 babies were shifted to NICU for various indicatios, most of them were absorbed for 48-72hrs, given antibiotics and IV fluids, the indications were hypoglycaemia, hyperbilirubunemia, respiratory distress syndrome.

Table 19-Distribution according to perinatal complications and outcome

COMPLICATION	NUMBER	PERCENTAGE(%)
HYPERBILIRUBINEMIA	24	10.8
HYPOGLYCAEMIA	38	17.8
HYPOCALCEMIA	22	9.9
RDS	20	9.0
MACROSOMIA	28	12.6
IUGR	6	2.7
CONGENITAL MALFORMATIONS	4	1.8
NEONATAL SEPSIS	4	1.8
IUD	3	1.3

Fetal hypoglycaemia was observed in 18% of babies, most common neonatal outcome among women with GDM.11% offspring had hyperbilirubinemia,12% babies were macrosomic .3 women had IUFD, out of which 2 were FSB and 1 was MSB .4 babies developed sepsis of which 2 were preterm and other 2 babies had meconium aspiration. Congenital malformations were seen in 4 babies, all 4 were delivered to women who were unregistered and below table show various congenital malformations

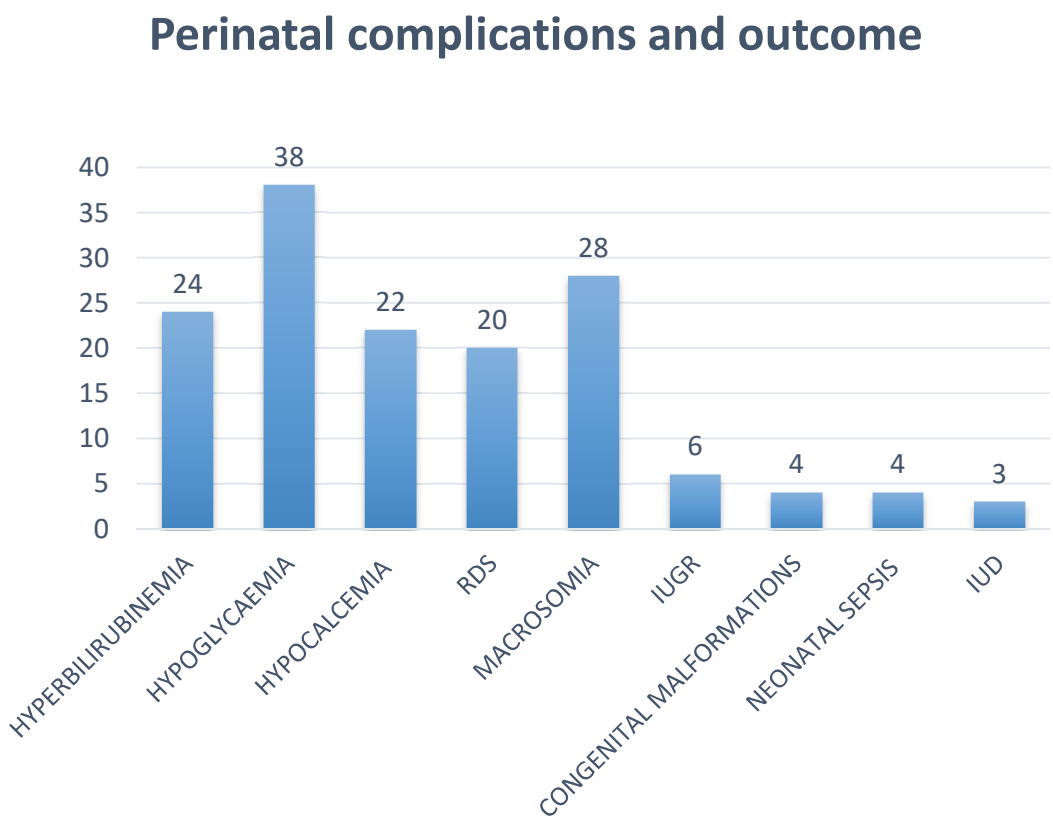


Figure 19- Perinatal complications and outcome among babies delivered to women with GDM

Table 20- Distribution according to congenital malformations

Congenital malformations	Number
TGA	1
B/L CTEV	1
Cervicomeningocele	1
Choanal atresia	1

5. DISCUSSION

The present study is a prospective observational study conducted in KAHER'S Dr Prabhakar kore hospital attached to JNMC to know the effect of GDM on women with its maternal and perinatal outcome in study period from January 2020 to March 2021. The prevalence of GDM in our study was found to be 14%. According to IDF estimates, the prevalence of HIP is 16.2% globally, GDM accounted for 85.1%, while other types of diabetes first detected during pregnancy being 7.4% and pregestational diabetes being 7.5% [58]. We can attribute our higher rate to South Asian ethnicity and universal screening and diagnosis in all the pregnant women attending the health facility. The data was comparable to study done by Abdulbari et al[59] showed prevalence of 16.3%, 27.5% in study done by Carmelo capula et al[60].

Our study shows that prevalence was higher among registered cases,most women belonged to urban area which shows that urbanization has led to increase in prevalence of GDM.

In this study, maximum patients (43%) were clustered in the age group of 26-30 years and 30% of patients were over 30 years of age. A study in Jammu done by Wahi P et al[61] also stated when compared with women of normal OGTT, women with GDM were older. The finding were similar with the study done by Hedderson et al [62] which had maximum incidence between 25-34 years. Landon Mark B et al.[63] study had similar finding of 29 years. Jacobson John D and Cousins Larry et al[64] study also had similar observation of 28 years. Langer Oded et al [65] study found mean age of GDM to be 27years.

Thus GDM affects older women more than younger ones, in concert with the pathophysiology of the disease. In the present study, 40% patients were primigravida while 60% patients were multigravida. The study by Rajput et al[66], showed that higher parity would have a higher rate of GDM. Positive family history as a risk factor was noted in 20% patients in this study. In the study conducted in UK by Nanda et al[67], positive family history was found in 23.9% patients.

Among many possible mediators of the increased risk of GDM, maternal age and prepregnancy BMI are the predominant factors. . Prepregnancy BMI is the second predominant mediator of the increased risk of GDM. Overweight and obese women were at higher risk of developing GDM. Since higher BMI values are one of the main risk factors in type 2 diabetes mellitus, it is no wonder that a similar correlation is observed between GDM and BMI. In our study most of the women were in the range of 25-25.9(overweight) Comparable associations have been published in studies done by Wapner RJ, et al.[68,69]

Hypothyroidism was noted in 42 patients (18%) in the present study. These patients were previously diagnosed hypothyroid, already on thyroid supplementation in adequate dose. According to Toulis KA et al[70], a modestly increased risk of GDM might be present in pregnant women with subclinical Hypothyroidism compared to euthyroid pregnant women.

The incidence of composite adverse maternal outcome was higher (52.9%) among women with GDM. Specifically, the incidence of caesarean delivery, PIH, PROM, was higher among women with GDM. This indicated that GDM can result a higher maternal morbidity.

Women with GDM were three times at high risk for PIH. In our study 18% of cases of GDM had association with PIH, in accordance with many studies done in

past like Pennison Erin H and Egerman Robert S et al[71] found an incidence of PIH to be 20.9%. The study done by Gajjar F Maitra N K et al[72] found this to as high as 60%.The finding was consistent with several other studies [73,74] which reported that GDM increased the incidence of PIH. . The association might be due to the nature of co-existing mutual risk factors, such as obesity, advanced maternal age and family history of diabetes and hypertension [75]. To maintain stable blood glucoselevels, β cells in the pancreas subsequently increase the production of insulin, which results in hyperinsulinemia[76] Evidence suggests that insulin resistance contribute to sodium retention and vasoconstriction in the pathogenesis of hypertensive disorders during pregnancy[77,78,79,80] .A better understanding of the association between these conditions may lead to implement more effective strategies on mutual risk factors during prenatal care.

Our study showed the incidence of preterm labour to be 9%. Similar observation of 13.9% was made by Ostlund Ingrid et al[81]. 7.4% preterm labour was seen in B Dittakaran et al[82] study.

Our study identified that mothers with GDM had more risk for PROM accounting to 9.4% .Other studies demonstrated that there was a direct relationship between GDM and PROM[83,84,85,86]. This might be due to the secondary complications of polyhydraminos and macrosomic babies caused by GDM and leads the head of the fetus to be arrested at the pelvic inlet, and the entire force exerted by the uterus is directed to the portion of membranes in contact with the internal os. Thus, early rupture of membranes is more likely to occur [87]

The incidence of polyhydramnios in our study was to be 6%. . Jacobson John D et al[88] found polyhydramnios in 2.1% cases while Hedderson Monique M et al[89] found it in only 0.7% cases. Maternal UTI due to E.coli and vaginal candidiasis was

detected in 28% women. Mamata Bhat et al[90] found incidence of infections to be 13%. 76% of the cases in our study were on diet therapy alone while 32% were on both diet and insulin therapy and rest 2% were on diet and OHA.. This is similar to the study done by Garner Peter et al[91] where 50% were on insulin. Jacobson John D et al[92] had only 13 out of 97 cases on insulin. Adams Kristina M et al[93] had 76 cases on insulin and 297 cases only on diet therapy.

Farrar D et al[94] suggested, using the "step-up" approach ensures that interventions are only offered if required, resulting in less burden for the woman and care providers, which is particularly important given the increasing prevalence of GDM.

We found the risk of caesarean delivery was higher among pregnant women with GDM accounting to 78% in our study. Previous studies [95,96,97] also demonstrated that the risk for caesarean delivery was high among GDM patients. For instance, GDM increased the incidence of caesarean sections (CS) from 30% [98] to 35% [99]. Though GDM alone is not an indication for CS before 38 weeks of gestation, it becomes evident that CS is a priority choice for many obstetricians due to different maternal and fetal complications arising from GDM [100]. Despite evidences showing the benefits of vaginal delivery, CS has been preferred for most diabetic pregnant women with previous operations for fear of the rupture of the uterus that may be associated with the risk of fetal macrosomia[101,102].

Additionally, a study by Odar E et al[103] revealed that the modes of delivery were similar, but genital injuries were more common among women with GDM. The primary indications of CS might be fetal macrosomia that resulted from GDM. On the other hand, CS can prevent poor obstetric outcomes and be a life-saving procedure for both the mother and the fetus [104]. However, there is a growing concern about

unnecessary CS that leads to risks for maternal morbidity, neonatal death and neonatal admissions into intensive care units [105]

Perinatal Complications noted in GDM mothers include fetal macrosomia, impaired fetal growth, metabolic and electrolyte abnormalities, cardiovascular and CNS anomalies. The link between a mother's GDM and negative outcomes for the newborn and for the mother are well established and broadly accepted [106,107,108]. Therefore, it is not surprising that neonates born to women with diabetes were much heavier, were more often born prematurely and were more often delivered by C-section than children of mothers without diabetes. In general, the higher number of premature deliveries and C-sections among women with GDM can be explained by faster intrauterine growth due to overexposure to the energy source. 40% of the total neonates were admitted to NICU for various indications

All the admitted babies were on intravenous fluids, antibiotics and monitored and observed for 48-72 hrs. .Das Vinita et al [109] study had significant incidence of NICU admission (p=0.001). Ostlund Ingrid et al[110] noted this to be 29%. Crowther Caroline A et al[111] found it to be as high as 71%.

Neonatal hypoglycaemia is one of the most frequent adverse effects of exposure to GDM. Children suffering from neonatal hypoglycaemia may develop motor impairments and learning and behavioural difficulties [112,113]. There is an established and accepted relationship between neonatal hypoglycaemia and GDM, which, in turn is facilitated by mother's high BMI values [114].The prevalence of the neonatal hypoglycaemia depends on nutritional status, gestational age and onset of feeding. Approximately 2 to 4% of mature newborns are affected, compared to 5 to 10% of premature babies and up to 50% of babies in GDM pregnancies. Comparing these figures with the data of our study where 18% of the babies had hypoglycaemia,

we observe a much lower incidence of hypoglycaemia in neonates born to GDM mothers. These findings may be an indication for the appropriate therapy applied to this group of expecting mothers. However, the metabolome and epigenome of the offspring are affected by maternal BMI and glycaemia, suggesting long-term consequences for the next generation [115,116].

The present study found the incidence of hyperbilirubinemia to be 10%. A similar observation of 10% was made by Landon Mark M et al[117] . B Dittakarn et al [118] found it to be 20%.

The occurrence of respiratory distress syndrome was not increased in the neonates of GDM mothers in our study (9%) compared to literature.[119,120] A comprehensive review by Piper[121] in 2002 explained the importance of glycemic control where diabetic women with good glucose control had babies with lung maturation similar to that of non-diabetic population. Another important factor may be avoidance of iatrogenic prematurity by termination after 38 weeks.

Macrosomia is a known complication of GDM and it is a proven fact that post prandial hyperglycaemia is associated with increased incidence of macrosomia. The macrosomia rate in our study was 12% which was higher than various studies reported in literature like 5.9% in the study conducted by Landon MB et al[122] 6% by Garner Peter et al [123] This observed difference can be due to better glycemic control or due to different genetic, demographic and maternal metabolic factors that are known to affect foetal growth. The difference may also be due to different diagnostic criteria applied in different literatures. Although glycemic control plays an important role in determining foetal size, excessive maternal weight gain and obesity also strongly influence neonatal birth weight, even in women without glucose intolerance.[123] 1.8% of congenital anomalies in the newborn were noticed in our study. Congenital

malformations were seen in 4 babies, all 4 were delivered to women who were unregistered. A similar observation of 5.9% was made by Al-Hakeem Malak M. et al[124] noticed significant p-value (0.001) in the incidence of congenital anomalies.

Literature does not show an increased incidence of congenital anomalies in gestational diabetes compared to that of general population, again related to hyperglycaemia at periconceptional period and during period of organogenesis.[123] In the study maternal mortality was nil. Perinatal mortality was also nil. Still birth rate was found to be 2%.

CONCLUSION

GDM is found to be associated with higher incidence of various adverse maternal and perinatal outcomes, like preeclampsia, infections, increased risk of caesarean delivery in mother and macrosomia, hypoglycaemia and hrperbilirubinemia in neonate as shown and supported by the results of our study. Considering high prevalence in Indian population, it is recommended and suggested by various evidences that universal screening irrespective of the risk status and early detection of GDM provide window for implementation of various management methods to keep check on blood sugar level and maintain good glycemic control in pregnant women. It will reduce the adversities of GDM on both mother and fetus and thus helps in prevention of transgenerational transfer of adversities to the neonate. Further research should focus on preconceptional diagnosis and management of glucose intolerance in the vulnerable Indian population to reduce the burden of adverse outcomes related to gestational diabetes mellitus.

SUMMARY

GDM is the most common endocrine medical disorder seen during pregnancy. Presence of GDM in women will give rise to various adverse maternal and fetal outcome .Our study was done to give an insight into these outcome. Prevalence of GDM was found to be 14% among study population during our study period.

The present study shows that highest incidence of GDM occurs at around 28 years and that most of the cases can be managed with diet & exercises as first line of therapy. Our study shows that the incidence of pregnancy complications like PIH in 38 women (19%), infections in 28 women (12%), polyhydramnios in 13 women (6%) are increased significantly in these cases.

The present study shows the increased rate of LSCS in GDM cases (78%), the indications being not only GDM but also the associated risk factors like PIH, big baby etc. Previous LSCS being the most common indication for cesarean section in GDM.

Among the neonatal metabolic complications, we observed that hypoglycaemia in 38 neonates (18%), hyperbilirubinemia in 24 of neonates (10%), RDS in 20 neonates (9%) are increased in babies of GDM mothers and macrosomia was seen in 28 babies (12%). 40% of babies needed NICU care either for the morbidity or for observation upto 48-72 hours.

Early recognition and intervention with good glycemic control in GDM cases decreases both maternal and fetal morbidity.

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ANNEXURE - I - ETHICAL CLEARANCE

K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed – to- be- University)
Accredited 'A' Grade by NAAC (2nd Cycle) Placed in Category 'A' by MHRD (GoI)
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
Website: http//www.jnmc.edu
E-Mail : dome@jnmc.edu
Phone: (+ 91-(0)831 Office : 2472550
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref: MDC/DOME/ 299 **Date: 24/12/2019**

To

REG. NO. BJ0119009

PG student in Obstetrics and Gynecology,
J. N. Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"MATERNAL AND PERINATAL OUTCOME IN WOMEN WITH GESTATIONAL DIABETES MELLITUS – A ONE YEAR CROSS SECTIONAL STUDY",** is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

(Dr. Anita Dalal)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

(Dr. Roopa M Bellad)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

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ANEXXURE II- CONSENT FORM

Title: **Maternal and perinatal outcome in gestational diabetes mellitus women– A one year cross sectional study**

Principal Investigator: -

DR _____

Associate Professor,

Department of Obstetrics and Gynaecology,

J.N. Medical College, Belagavi

Co-investigator

REG. NO. BJ0119009

Post Graduate

Department of Obstetrics and gynaecology

J N medical college, Belagavi

CONSENT FOR PARTICIPATION IN RESEARCH STUDY

Mrs. _____ we are requesting you to enroll yourself in study titled **"**Maternal and perinatal outcome in gestational diabetes mellitus women-A one year cross sectional study**"** Conducted by **REG. NO. BJ0119009** Post Graduate in M.S. Obstetrics and Gynaecology under the guidance of DR. _____ Associate Professor, Department of Obstetrics and Gynaecology, J.N. Medical College, Belgaum under KLE University, Belgaum.

Objectives /purpose of study:

Respected Madam we request you to participate in our study as you are eligible for participating and your participation in this study is important as it helps to study maternal and perinatal outcome in gestational diabetes mellitus women

Your participation in research is voluntary. Your decision whether to participate in the study or not will not change present or future health care services

offered to you and will not affect your relationship with J.N. Medical College. If you decide to participate you are free to withdraw at any time. All pregnant women meeting the inclusion criteria will be recruited in our study.

Purpose of study: To study maternal and perinatal outcome in gestational diabetes women

Type of Study: cross sectional study

Financial Incentives for participation:

No financial incentives are being offered to enrolled patients. It is purely being done with the idea of research and all the cost of the study will be borne by the investigator.

Privacy and Confidentiality:

The only people who will know that you are the research subject will be the members of the research team. No information about you or information provided by you during the research will be disclosed to others without your written permission except:

1. In emergency to protect your rights and welfare.
2. If required by law.

Authorization to Publish Results:

When the results of the research are published or discussed, in a conference, no information will be displayed that would disclose your identity. Any information that is obtained in connection with this study and that can be identified with you will remain confidential. Results of the study will be used to improve maternal outcome.

Right to refuse or withdraw from study:

You do not have to participate in this research if you do not wish to.You can withdraw at any time from the study. There will be no penalty for withdrawal. Your treatment and care in this hospital will not change irrespective of whether you agree to participate or not. You can be removed from the study if necessary.

Institutional/sponsor's policy:

In the event of any injury related to the study, treatment will be made available through KAHER, Belagavi. There is no compensation or payment for such medical treatment by law. If you are injured you may contact **REG. NO. BJ0119009,** Post graduate student, Department of Obstetrics and Gynaecology, KAHER or by Ph. No: _____.

Contact details:

In case you have any questions related to the study, in future or in case of study related injury or illness, you can contact **REG. NO. BJ0119009,** Post graduate student, Department of Obstetrics and Gynaecology, KAHER, Ph. No: _____ or Dr. _____ , Dept. Of Obstetrics and Gynaecology, KAHER Belagavi

If you have any queries about your rights as a study participant, you may contact Dr. Roopa M Bellad, Prof. of Pediatrics as Chairman of J. N. Medical College Institutional Ethics Committee on Human Subjects Research, Phone No.0831 2473777 ext-1527 at J. N. Medical College, Belagavi.

Consent statement:

I, _____ voluntarily agree for participating in this study. By signing this consent form I am not giving up any of my legal rights, I may withdraw from the study anytime. I am signing the consent form after having read or been read form in my own vernacular language, including the risks and the benefits and having all my questions answered.

Participant Name : _____

Signature of the Left Thumb Print of Participant : _____

Investigators Name: _____ Signature: _____

Witness Name : _____ Signature: _____

Date: _____

PROFORMA

Case No : **Patient id:**
Name : **Occupation :**
Age : **phone number(pt):**
Address :
socio- economic status:

A) CHIEF COMPLAINTS:

B) HISTORY OF PRESENTING COMPLAINTS:

H/o ---------- months of amenorrhea
Able to perceive fetal movements (y/n)

C) MENSTRUAL HISTORY:

LMP
EDD
C.EDD(if):
GA at the time of diagnosis:

D)OBSTETRIC HISTORY

Married life
Consanguinity
Obstetric score**:** G P L A
1-
2-
3-
4

E.H/O of GDM in previous pregnancy: yes/no

F.Present pregnancy: complaints (history)	**Yes**	**No**	**GA at having**
1)H/O Of Excessive Vomiting			
2)H/O Polyuria			
3)H/O Polydypsia			
4)H/O Headache			
5)H/O Blurring Of Vision			
6)H/O Bleeding P/v			
7)H/O Leaking P/v			
8)H/O Burning Micturation			
9)H/O White Discharge P/v			
10)H/O Constipation			

11)Any other complaints specify: ☐

G. PAST HISTORY

1. Diabetes ☐
2.Hypertension ☐
3.Cardiac Disease ☐
4.Thyroid Disorders: ☐
5.If any Other Specify ☐

H. FAMILY HISTORY

1.Diabetes Mellitus ☐
2.Hypertension ☐
3.Cardiac Disease ☐
4.Any Other ☐

I. EXAMINATION OF PATIENT:

On examination

1. Height in cms : _____
2.Weight : _____
BMI: _____

3. Pulse rate _____
4. BP in mm of Hg _____
5. Pallor _____

Systemic examination

a. CVS _____
b. RS _____
c. Per abdomen :

Inspection:		**YES/NO**
Abdomen-	1.Normal	☐ ☐
	2.Over distended	☐ ☐

Palpation:
Fundal height:
Liquor:.1.Adequate
2.Scanty
3.Excess

Auscultation: --------- FHS (/min)
EFW(by Johnson formula)----------

Per vagina(if in labor):
Dilatation:
Length :
Effacement :
Position :
Consistency :
Vertex station :
Membranes :

J) DIAGNOSIS:

H)Investigations

1)Blood grouping and RH typing ----------
2)Hb ----------
3)RBS ----------
4)Urine routine and microscopy ----------
5)HIV ----------
6)HBsAg ----------
7)VDRL ----------
8)Glucose challenge test: Fasting ----------
1hr ----------
2 hr ----------
Diagnosis GDM (Y/N) IGT(Y/N)

9)FBS/PPBS
10)HBA1C
(at the time of diagnosis and at the time of developing any complaints if any)

11)S.TSH ----------
12)TPO antibodies ----------
13)Free t3 ----------
14)Free t4 ----------

15)USG Findings : **Yes** **No**

1)Macrosomia ☐ ☐
2) Polyhydramnios ☐ ☐
3)IUGR ☐ ☐
4)Placenta Previa ☐ ☐
5)Multiple Gestation ☐ ☐

J)Treatment :

	YES	NO
Controlled with:		
Insulin	☐	☐
Meal	☐	☐
Oral hypoglycemic drugs	☐	☐

K)Pregnancy outcome:

Gestational age at delivery
Vaginal delivery: Assisted ☐ Spontaneous ☐

Induced (indication)----------

Caesarean section(indication) ----------

Maternal complications

a)During pregnancy:
1) Abortion ☐
2) Preterm labor ☐
3)Infection ☐
4)Polyhydramnios ☐
5)PIH ☐
6)Hypothyroidsm ☐
7)Eclampsia ☐
8)APH ☐

b)During labor:

1)Prolongatoin of labor due to big baby
2)Shoulder dystocia
3)Perineal injuries
4)Postpartum haemorrhage
5)Operative interference (if any)

c)Puerperium:
1) Pueriperial sepsis
2)Secondary PPH
3)Any other complaints:

L)Perinatal outcome:

1)Live birth
2)Still birth
3)IUD
4)Congenital anomalies
If any specify

Baby details
Sex:
Weight:

APGAR: 1 MIN
5 MIN
NICU admission **Y/N**

M)NEONATAL COMPLICATIONS:

1) Hypoglycemia
2) Fetal macrosomia
3) Congenital malformations
4) Jaundice
5)Hypocalcemia
6)Respiratory distress syndrome
7)Electrolyte Imbalance

Sr.	IP NO	Age	R/UR	Residence	Socio economic status	BMI	Parity	past h/o abortion	h/o IUD	Past h/o	past h/o	past h/o cardiac disease	h/o	hypothyroidism in present pregnancy	Gestation at the time of diagnosis	HBA1C	TSH	Split	Insulin	OHA	PIH	PTL	PROM	Hydramnios	APH	Infection	vaginal	vaginal		c section	c- section		Gestation at	LIVE BIRTH /IUD	Sex	Birth	Nicu admission	Congenital malformation	Hyper-Bilirubinemia	Hypoglycemia	RDS	Macrosomia	Prematurity	IUGR	Sepsis	Peurperal	PPH
No.										GDM	hypothyroidism		bigbaby					Meals					/PPROM				spontaneous	induced	Instrumental delivery	elective	emergency	indication	delivery			Wgt										sepsis	
1	992893	39	R	Urban	2	20	Primi								18	6.20%	2.3	yes					Yes								emergency	DCDA twins with PPROM	33W 2 D		A - Male B - Fema	- 2.1 kg B - 1.6 K	YES			Yes	Yes						
2	993245	20	UR	Rural	2	22	Primi								14	5.30%	1.5	yes													emergency	MSL	39W +6		Male	2.8kg											
3	993162	29	R	Urban	1	22.5	G5A4	Yes						yes	24	5.20%	3.1	yes													emergency	msl	37 weeks		Male	3 kg	Yes			Yes						yes	
4	993809	25	R	Urban	2	19	Primi								26	5.80%	1.2	yes			yes					UTI					emergency	Failed induction	37 w		Female	2.8 kg	YES		yes		yes						
5	992636	33	R	Urban	2	24	Primi							yes	25	6.40%	4.1		yes		yes										emergency	DGDM with macrosomia	34 w		Male	3.6kg	yes					yes					
6	994250	26	R	Urban	2	25.9	G3P1L1A1	yes		yes					24	6.10%	2.3		yes											elective		Previous lscs	37 w +2		male	3.8kg						yes					
7	994078	30	UR	Rural	2	26	G4P2L1A1	Yes		yes			yes		26	5.10%	2.3	yes												elective		breech	38 w		Female	3.1 kg	yes			yes							
8	994505	23	R	Urban	2	24.8	Primi								25	4.70%	3.4	yes									spontaneous						38 w		male	2.5kg	yes				yes						
9	994553	24	R	Urban	2	24	G3P1L1					yes			16	6.50%	3.4	yes												elective		Previous lscs	37 w		Female	2.7 kg	yes		yes	yes	yes						
10	993658	30	UR	Rural	2	26	G2P1L1				yes				24	5.20%	3	yes												elective		Previous lscs	38 W		Male	2.8 kg											
11	994948	23	R	Urban	2	26	G2P1L1							YES	24	5.20%	4		yes				yes							elective	emergency	fetal macrosomia	34 w 5d		Female	2.8 kg	yes					yes					
12	996868	25	R	Rural	2	28	G2P1L1								26	5.30%	2.3	yes					YES								emergency	cord prollapse	39 weeks 2d		Female	2.8 kg											
13	996579	23	R	Urban	2	22	Primi								24	5.20%	2.4	yes									spontaneous						39 weeks 2d		Female	3 kg			yes								
14	997172	29	UR	Urban	2	24	Primi								24	5.80%	2.2		yes							vulvovaginitis					emergency	macrosomia	38 weeks		Male	3.4kg	yes					yes					
15	997107	25	UR	Urban	1	26	Primi								26	6.10%	2.4		yes											elective		breech	38 weeks +2		male	3.2kg	yes		yes	yes							
16	997280	28	R	Urban	1	24	G4P3L2D1		yes	yes				yes	18	5.80%	2.1	yes									spontaneous						39 weeks		Female	2.8 kg	yes			yes							
17	994764	24	R	Urban	2	20	Primi								24	5.40%	3.1	yes				yes					spontaneous						22weeks + 3		Female	490g	yes						yes				
18	997148	27	UR	Urban	2	25	G3P1L1A1	Yes					yes		24	5.20%	2.4	yes			yes	yes									emergency	PREV LSCS GDM with gestational HTN	33W3D		Female	2.4 kg	YES						YES				
19	997025	26	UR	Rural	2	27	G2P1L0		yes						24	6.20%	3.2	yes													emergency	fetal distress	38 weeks		male	2.8 kg	yes				yes						
20	998545	27	R	Urban	2	25	G2P1L1								20	5.10%	2.4			YES							spontaneous						39 weeks 2 d		Female	2.6 kg											
21	998491	29	R	urban	2	24	G2P1L1								16	5.20%	2.1	yes													emergency	Previous lscs	37 weeks		Female	2.8 kg			yes								
22	998175	36	R	Rural	2	27	Primi							yes	24	5.40%	4.4	yes				yes	yes								emergency	precious pregnancy with prom	33weeks + 6		Male	2.4 kg	yes				yes		yes				
23	998954	25	R	Rural	2	25	G2A1	Yes						Yes	20	5.80%	3.1	yes													emergency	fetal distress	39 weeks +3		Male	3.2 kg											
24	999078	28	R	Urban	2	22	G2P1L1								24	5.40%	2.2	yes												elective		Previous lscs	37 weeks		male	2.9 kg	yes		yes	yes							
25	999994	28	R	Urban	2	24	Primi							yes	26	6%	4.1		yes							UTI				elective		MSL	38 weeks +1		Female	2.7 kg											
26	1000000	23	R	Rural	1	23	Primi								24	5.10%	2.2	yes													emergency	Failed induction	37 weeks +2		Female	2.9 kg											
27	1000023	45	R	Rural	2	21	Primi								20	5.20%	1.7	yes			yes									elective		DCDA twins with precious pregnancy	36 weeks		twin A - male Twin B	- 2.3 kg Twin B -	yes				yes						
28	999978	26	UR	Rural	2	25	G2P1L1								16	5.20%	2.3	yes						oligohydramnios		vulvovaginitis		induced					40 weeks		Male	3.1kg											
29	1000064	30	UR	Urban	2	27	G4P2L2A1	Yes		yes			yes		24	5.40%	2.3	yes												elective		previous lscs	38 weeks		male	2.8kg											
30	1002027	33	R	Urban	2	24	G2P1L1								24	5%	2.1	yes				yes			yes						emergency	abruptio placenta	34 weeks		female	3 kg	yes			yes			yes				
31	1001837	20	R	Urban	2	22	Primi								26	6%	3.1	yes												elective		DCDA twins	37 weeks		twin A - female Twin B	- 2.4 kg Twin B -	YES					YES					
32	1008214	23	UR	Urban	2	25	Primi								22	5%	2.4	yes													emergency	fetal distress	40 weeks		Female	3.1 kg	yes		yes							yes	
33	1001522	28	R	Urban	1	24	G4P1L1A2	Yes							24	5.80%	3.1	yes			yes					vulvo vaginitis					emergency	breech	36 weeks +4		Female	2.7 kg											
34	1003914	25	UR	Urban	1	22	Primi							yes	28	5.20%	4.2	yes			yes						spontaneous						36 weeks+ 3		Male	3.1kg	yes						yes				yes
35	1002885	26	UR	Urban	1	21	G5P1L1A3								30	6.20%	3.1		yes												emergency	previous lscs with fetal macrosomia	36 weeks +5		Female	3.9 kg	yes					yes					
36	1003262	24	R	Rural	1	23	G2P1L1								27	5.10%	2.4	yes								UTI				elective		previous lscs	37 weeks + 5		Male	3.4 kg	yes		yes	yes							
37	1003335	26	R	Rural	1	24	G2P1L0		yes					yes	30	5.20%	5.1	yes			yes										emergency	severe PE	28 weeks		male	1kg					yes		yes	yes			
38	1003928	20	R	Urban	2	21	G2A1	Yes							22	5.20%	3.1	yes								UTI		induced					37 weeks +6		Female	2.6kg											
39	1002026	28	R	Urban	2	21.4	Primi								20	6.00%	2.1	yes									spontaneous						39 weeks		Male	2.8 kg											
40	1004749	26	UR	Urban	2	20	G2P1L0		yes						24	6.10%	2.3	YES										induced					37 weeks + 6		male	2.9 kg											
41	1005313	33	UR	Rural	2	24	G4P1L1A2	Yes						yes	23	6%	4.5	yes			yes										emergency	severe PE	36 weeks +4		Male	3.7 kg	yes					yes					
42	1006509	23	R	Rural	2	25	G4P2L2A1	Yes							8	5.20%	2.4	yes				yes						induced					36 weeks+2		Male	2.6 kg	yes		yes		yes						
43	1007120	28	R	Urban	2	26	G4P3L3								16	5%	2.3	yes												elective		DCDA twins	36 weeks +5		twin A - female Twin B	- 2.5KG TWIN B	no										
44	1007249	20	R	Urban	2	27	G2A1	Yes							20	5.40%	3.2	yes													emergency	Pathological trace	38 weeks +1 day		Male	3kg	yes					yes					
45	1008246	26	R	Urban	2	23.3	G3P1L1A1	Yes							20	5.20%	1.4	yes			yes							induced					37 weeks +2		Male	2.8kg											
46	1008630	29	R	Urban	2	22	G2P1L1								26	4.60%	2.1	yes													emergency	previous lscs in labor	39 weeks +3		Female	2.9kg	yes		yes								
47	1008715	34	R	Urban	2	24	G2P1L1								25	4.80%	2.3										spontaneous		instrumental (poor maternal bear down)				40weeks +1		male	3.1kg	yes	choanal atresia									
48	1009030	28	UR	Rural	2	22.4	Primi							yes	22	5.50%	3.1		yes					oligohydarmnios		UTI				elective		oligohydramnios	36 weeks+6		Female	2.8kg											
49	1009228	26	UR	Urban	1	27	Primi								20	5.10%	2.2	yes													emergency	fetal distress	39 weeks +6		Female	2.9kg											
50	1009261	27	UR	Urban	1	25	Primi							yes	18	4.80%	2.1					yes	yes				spontaneous						28 weeks +5		male	1.1 kg	yes						yes				
51	1009331	21	R	Urban	1	22	Primi								28	5.20%	1.8	yes									spontaneous						38 weeks		male	2.8 kg	yes				yes						
52	1009507	32	R	Urban	1	24.6	G2P1L1								16	5.20%	2	yes												elective		previous lscs	39 weeks +1		male	3.6 kg											
53	1009578	21	UR	Rural	1	24.6	Primi								22	4.20%	3.2	yes					yes								emergency	pprom	36 weeks + 6		Male	2.7kg											
54	1009599	28	R	Rural	2	23.6	G5P2A2	yes		yes					24	4.80%	3.1	yes												elective		precious pregnancy	37 weeks +6		Female	2.2kg	yes				yes						
55	1009621	27	R	Rural	2	22.2	Primi								24	5.20%	3.4	yes													emergency	MSL	37 weeks +6		male	2.9 kg	yes				yes						
56	1009737	29	UR	Rural	1	22.1	G2P1L1							yes	22	6%	5.2		yes		yes										emergency	previous lscs	39 weeks		Female	3.4 kg											
57	1009769	34	R	Urban	1	22.6	Primi								20	5.40%	2		yes				yes								emergency	precious pregnancy with PPROM	36 weeks		Female	2.5 kg											
58	1009803	27	R	Rural	2	22.8	G3P1A1	yes							26	5.10%	3.2	yes			yes				yes						emergency	revealed abruption	39 weeks		male	2.3 kg			yes								
59	1009919	22	R	Urban	2	22.4	G3P1L1A1	Yes		yes					30	5.00%	1.9	yes					yes				spontaneous						38 weeks +4		Female	2.6 kg	yes							yes			
60	1010243	22	R	Rural	2	22.4	G2A1	Yes							24	5.20%	1.9	yes			yes										emergency	uncontrolled hypertension	36 weeks		female	2.1 kg	yes	ervical meningeo ceal									
61	1010506	33	UR	Urban	1	22.1	G3P2L2			yes					24	4.30%	2										spontaneous						38 weeks		male	2.6 kg	yes									yes	
62	1010502	31	R	Urban	1	24	G3P2L2			yes			yes		22	6.00%	2.3	yes										induced					41 weeks		Female	3.1 kg											
63	1010427	25	R	Urban	2	27	G2A1	Yes						yes	20	5.40%	0.34	yes			yes										emergency	HELLP syndrome with gestational HTN with GDM	38 weeks +2		Male	2kg											
64	1010591	30	R	Urban	2	25	Primi								26	5.80%	2.3	yes												elective		GDM with precious pregnancy	38 weeks		female	3.3 kg											
65	1010631	30	R	Urban	2	24	Primi								20	5.30%	3.1	yes			yes				yes						emergency	Abruption with DIC	32 weeks	IUD (FSB)	female	1.5 kg											
66	1011007	24	R	Rural	2	24.9	Primi								24	5.60%	1.9	yes													emergency	Deep transverse arrest	37 weeks +1		male	3.2 kg											
67	1010985	21	UR	Rural	2	23.7	Primi								28	4.80%	2.5	yes									spontaneous						40 weeks		male	3.2 kg			yes								
68	1011211	29	UR	Rural	1	25.8	G2P1L1								21	3.60%	2.5	yes			yes										emergency	severe PE	38 weeks +2		male	3.1 kg											
69	1011349	30	UR	Urban	1	20	G2P1L1								27	4.80%	3.1	yes													emergency	thick MSL	40 weeks +6		Female	3.1 kg	yes				yes						
70	1011346	33	R	Urban	1	22	Primi								22	5.40%	3.2	yes								UTI				elective		precious pregnancy	36 weeks 2 days		male	2.3 kg	yes						yes				
71	1011462	30	R	urban	1	22.5	G2A1	Yes						yes	24	6.00%	6.1										spontaneous		instrumental (poor maternal bear down)				38 weeks + 4		Female	3.1 kg											
72	1011678	24	R	Urban	2	19	G2A1	Yes							34	6.10%	2.1	yes					YES								emergency	non progress of labour	37 weeks + 4		male	2.8 kg			yes								
73	1011650	26	R	Urban	2	24	G4P1L0A2	YES							29	6.20%	3.2	yes												elective		previous lscs	37 weeks 6 days		male	3.4 kg											
74	1012009	23	R	Urban	2	25.9	G2A1	Yes							32	5.80%	2.5	yes						oligo hydramnios				induced					39 weeks 5 days		female	2.2 kg								YES			
75	1011846	20	UR	Urban	2	26	Primi								30	5.50%	2.4	yes						oligo hydramnios				induced					39 weeks		Female	2 kg								yes			
76	1012078	27	UR	Urban	2	24.8	G4P1L1A2	Yes							29	5.40%	2.2		yes												emergency	Failed induction	39 weeks		male	2.6 kg	yes		yes								
77	1012866	28	UR	Urban	2	24	G3P2L2								37	5.30%	3.1		yes												emergency	previous 2 lscs	37 weeks +2		male	2.9 kg	yes			yes							
78	1012684	23	R	Urban	2	26	G4P1L1A2	Yes						yes	21	5.40%	5.3	yes									spontaneous						36 weeks 4 ddays		Male	3 kg											
79	1013236	36	UR	Rural	2	26	G2P1L1								32	5.80%	3.1	yes												elective		previous lscs	37 weeks		female	2.3 kg											
80	1013242	32	R	Rural	2	28	G5P1L1A3	Yes		yes			yes		25	5.60%	3.2	yes												elective		previous lscs	38 weeks 1 day		male	3.1 kg											
81	1013303	37	UR	Rural	2	22	G3P1L1A1	Yes							24	4.30%	2.4	yes												elective		previous lscs	38 weeks 1 day		Male	3 kg											
82	1013596	32	UR	Rural	2	24	Primi								26	5.10%	2.2	yes													emergency	thick MSL	40 weeks 2 days		male	3.5 kg	yes			yes							
83	1013895	23	UR	Urban	2	26	Primi							yes	24	5.30%	5.6	yes												elective		CPD	39 weeks		Female	3.4 kg											
84	1014364	26	UR	Urban	2	24	G2P1L1								26	5.60%	2.3	yes												elective		previous lscs	38 weeks 1 day		Male	3.1 kg											
85	1014534	32	UR	Urban	2	20	Primi								22	5.10%	2.1		yes												emergency	Failed induction	38 weeks 2 days		Male	2.7 kg											
86	1014574	22	UR	Urban	2	25	G3P2D2		h/o 1 IUD						23	5.20%	3.1	yes													emergency	previous lscs with scar tenderness	35 weeks 1 day		Female	2.5 kg	yes						yes				
87	1014736	36	R	Urban	2	27	Primi							yes	10	5.5	4.3	yes													emergency	non progress of labour	39 weeks 1 day		male	3 kg											
88	1015044	34	R	Urban	2	25	G2P1L1								26	5.70%	3.1	yes			yes										emergency	previous lscs	37 weeks		Male	3.5 kg											
89	1014320	36	R	Rural	2	24	G3P1L1A1	Yes							20	5.80%	2.3		yes		yes										emergency	DCDA twin	33 weeks 5 days		twin A - female Twin B	- 2.6KG TWIN B	yes						YES				
90	1015031	36	R	Rural	2	27	G3P2L1								18	5.30%	3.2		yes							vulvovaginitis					emergency	uncontrolled hypertension	33 weeks 5 days		male	1.8 kg	yes				yes		yes				
91	1015625	34	R	Rural	2	25	G5P2L1A2	Yes		yes	yes			yes	24	5.10%	4.2		yes											elective		previous 2 lscs	37 weeks		Male	3.4kg											
92	1016244	43	R	Rural	1	22	Primi								28	5.30%	2.2		yes											elective		precious pregnancy	37 weeks		Male	3kg											
93	1015979	30	R	Rural	1	24	Primi								24	5.50%	2.3	yes													emergency	MCDA twins	34 weeks 2 day		twin A - female Twin B	- 1.8 kg Twin B -	yes				yes		yes				
94	1016459	30	UR	Rural	1	23	G3P1L1A1	Yes							26	5.90%	1.2	yes				yes					spontaneous						28 weeks 2 days		Male	1.3 kg	yes				yes		yes				
95	1016915	34	UR	Rural	1	21	G3P1L1A1	Yes					yes	yes	20	5.60%	3.9	yes												elective		previous lscs	37 weeks 1 day		Male	3.1 kg											
96	1016844	29	UR	Rural	1	25	G2A1								22	5.40%	2.2	yes													emergency	suspicious trace	34 weeks 4 days		Female	1.7kg	yes						yes	yes			
97	1017256	32	R	Urban	1	27	G4P1L1A2	yes						yes	10	6.10%	4.2		yes											elective		previous lscs with GDM	36 weeks 2 days		Female	2.9 kg											
98	1017260	31	R	Urban	2	24	G3A2	yes							12	5.80%	2.3		yes											elective		precious pregnancy	37 weeks 4 days		Female	3.3 kg											
99	1017301	40	UR	Urban	2	22	Primi								24	5.20%	2.4		yes				yes								emergency	prolonged pprom	33 weeks		Female	1.7kg	yes		yes		yes		yes			yes	
100	1017374	27	UR	Urban	2	25	G4P2L2A1	yes							26	5.20%	3.1		yes												emergency	uncontrolled sugars	34 weeks		male	2.7 kg	yes		yes			yes					
101	1017926	27	R	Urban	2	24	G4A3	yes							22	5.30%	3	yes													emergency	fetal distress	41 weeks 3 days		Female	2.9 kg											
102	1018177	35	R	Urban	2	22	G3P1L1A1	yes							28	5.40%	3.2	yes												elective		previous lscs	38 weeks		Female	3.1kg											
103	1018241	30	R	Urban	1	21	Primi							yes	16	5.10%	5.2		yes												emergency	persistent fetal tachycardia	36 weeks 6 days		Female	2.4kg	yes		yes		yes						
104	1018530	31	UR	Urban	2	23	Primi								24	5.60%			yes											elective		precious pregnancy	38 weeks 1 day		Female	2.7kg											
105	1018845	24	UR	Urban	2	24	Primi								26	5.40%		yes													emergency	fetal distress	39 weeks		Female	2.8 kg					yes						
106	1019494	26	R	Urban	2	21	Primi								20	5.30%		yes													emergency	thick MSL	39 weeks 4 days		Female	3kg											
107	1019962	31	UR	Rural	2	21.4	G3P1L1A1	yes							28	5.20%		yes													emergency	previous lscs	39 weeks		male	3.4 kg											
108	1020110	32	R	Rural	2	20	G2P1L1							yes	27	5.70%	5.1		yes											elective		previous lscs	38 weeks 2 days		male	2.7 kg	yes							yes			
109	1020918	24	R	Rural	2	24	Primi							yes	26	5.40%	5.2	yes								UTI		induced					36 weeks	IUD (MSB)	Female	1.6kg											
110	1020966	26	R	Urban	2	25	G2P1L1								22	5.30%		yes					yes				spontaneous		ntouse delivery (poor maternal bear down)				36 weeks 6 days		male	2.9 kg	yes	TGA									
111	1021048	22	R	Urban	2	26	Primi								22	5.20%		yes													emergency	fetal distress	37 weeks		male	2 kg	yes				yes			yes			
112	1021265	40	UR	Urban	2	27	Primi							yes	27	5,7	4.1		yes				yes								emergency	precious pregnancy with pprom	32 weeks 1 day		Male	2 kg	yes						yes				
113	1021412	26	UR	Urban	2	23.3	G2P1L0		yes						20	5.20%		yes					yes								emergency	prolonged pprom	37 weeks 2 days		female	2.3 kg											
114	1022209	30	R	Urban	1	22	Primi								28	5.10%		yes			yes										emergency	antepartum eclampsia	28 weeks 1 day		male	1.1kg	yes				yes			yes			yes
115	1022719	32	UR	Urban	1	24	G3P1L1A1	Yes							22	5.30%		yes										induced					37 weeks 1 day		female	2 kg											
116	1022678	26	R	Rural	1	22.4	Primi								28	5.50%		yes													emergency	Failed induction	39 weeks 1 day		Female	2.5 kg											
117	1023277	26	UR	Rural	1	27	Primi								30	5.70%		yes						polyhydramnios						elective		macrosomia with polyhydramnios	38 weeks		Female	3.7 kg	yes		yes			yes					
118	1023726	24	R	Rural	2	25	Primi								25	5.90%		yes			yes							induced	y (poor maternal bear down)				38 weeks 1 day		Female	3 kg											
119	1023921	29	UR	Urban	2	22	G2A1	Yes							26	5.20%			yes					polyhydramnios		vulvovaginitis					emergency	uncontrolled sugars	34 weeks 5 days		Female	3 kg						yes					
120	1024115	24	UR	Urban	2	24.6	Primi								22	5.40%			yes				yes								emergency	prolonged pprom	29 weeks 6 days		male	1.4 kg	yes						yes				
121	1024320	28	R	Rural	2	24.6	G2P1L1								18	5.60%		yes					yes								emergency	previous lscs in pprom	34 weeks 3 days		Female	2 kg											
122	1024309	32	R	Rural	2	23.6	Primi								14	5.70%		yes													emergency	Pathological trace	39 weeks 3 days		female	3kg			yes								
123	1024118	23	R	Urban	2	22.2	G2P1L0		yes					yes	24	5.40%	4.2	yes													emergency	MSL	37 weeks 4 days		male	2.7kg											
124	1024634	27	UR	Rural	2	22.1	G2A1					yes			26	5.30%		yes													emergency	Pathological trace	40 weeks		female	2.9 kg											
125	1024656	36	UR	Urban	1	27	G4P1L1A2	Yes						yes	25	5.70%	5.1	yes						polyhydramnios						elective		non progress of labour	36 weeks 4 days		male	3.1 kg											
126	1024835	19	UR	Urban	2	26	Primi								24	5.30%		yes									spontaneous		ntouse delivery (poor maternal bear down)						male	2.8 kg											
127	1025176	25	R	Rural	1	24	G5P1L0A3		yes					yes	26	5.30%	5.3	yes			yes		yes	olyhydramni	yes						emergency	BOH with polyhydramnios	35 weeks 4 days		male	2.4 kg											
128	1025806	30	R	Rural	2	22.4	G4P1L1A2	Yes							10	5.00%		yes								UTI					emergency	placenta previa	39 weeks		female	2 kg	yes							yes			
129	1025773	26	R	Rural	1	22	Primi								16	5.30%		yes			yes			oligohydramnios							emergency	fetal distress	38 weeks 2 day		female	1.3 kg					yes		yes	yes			
130	1025909	30	R	Rural	2	24	G2P1L1								24	5.40%		yes			yes										emergency	previous lscs	38 weeks 2 day		male	2.8 kg											
131	1026183	33	R	Urban	2	27	G2P1L1						yes		24	5.20%			yes											elective		previous lscs	37 weeks 6 days		Female	2.9 kg											
132	1026485	23	R	Urban	2	25	G3P1L1A1	Yes							26	6.10%		yes												elective		previous lscs	38 weeks		Female	2.5 kg	yes		yes	yes							
133	1026468	36	UR	Urban	2	24	G4P2L2A1	Yes							36	5.40%		yes									spontaneous						37 weeks 1 day		male	3.2 kg											
134	1026753	24	UR	Urban	2	24.9	G4P2L2A1	Yes							24	6.30%		yes								uti	spontaneous						38 weeks		male	2.8kg											
135	1027547	20	R	Urban	2	23.7	Primi								26	6.10%		yes									spontaneous						39 weeks 2 days		male	2.9 kg										yes	
136	1027807	32	UR	Urban	2	25.8	G3P1L1A1	Yes							18	5.30%			yes		yes			polyhydramnios		vulvo vaginitis					emergency	Pathological trace	38 weeks		Female	3 kg											
137	1027542	32	R	Urban	2	21	G3P1L1A1	Yes						yes	24	6.10%	5.2	yes						polyhydramnios							emergency	previous lscs	36 weeks 4 days		male	3.6 kg	yes		yes			yes					
138	1027727	25	UR	Urban	2	21.4	Primi								24	5.30%		yes			yes										emergency	MSL	38 weeks 2days		male	3.2 kg											
139	1027785	27	R	Urban	2	20	Primi								24	5.30%			yes												emergency	CPD	38 weeks		male	3.5 kg						yes					
140	1027786	28	UR	Rural	2	24	G5P1L1A3	Yes							30	6.10%		yes						polyhydramnios							emergency	previous lscs	33 weeks		male	2.7 kg	yes					yes	yes				
141	1028258	21	UR	Urban	2	25	G2A1	Yes							16	5.60%		yes			yes							induced					40 weeks 3 days	IUD (MSB)	male	3.8 kg											
142	1028578	28	UR	Urban	2	26	G2E1							yes	24	5.80%	5.3	yes													emergency	non progress of labour	40 weeks		Female	3.2 kg											
143	1028436	26	R	Urban	2	27	G3P2L2								20	5.10%		yes												elective		previous 2 lscs	34 weeks 2 days		male	3 kg	yes		yes	yes		yes					
144	1030171	29	R	Urban	2	23.3	Primi							yes	12	5.20%	4.3		yes											elective		precious pregnancy	38 weeks 5 days		male	2.7kg											
145	1030173	25	R	Urban	2	22	G3P1L1A1	Yes							26	5.40%		yes													emergency	previous lscs	40 weeks 3 days		Female	3 kg											
146	1031581	30	R	Urban	2	24	G2A1	Yes							24	5.30%				yes						uti					emergency	fetal distress	40 weeks 1 day		male	3.9 kg	yes			yes							
147	1032428	28	RU	Urban	2	22.4	G2P1L1								20	5.60%		yes			yes										emeregency	severe PE	40 weeks 2 days		male	3.5 kg	yes										
148	1033228	28	R	Urban	1	27	Primi					yes			16	6.10%			yes												emeregency	precious pregnancy	37 weeks 4 days		Female	2.8 kg											
149	1033462	35	UR	Rural	1	25	G3P1L1A1	Yes						yes	24	6.30%	4.5		yes							UTI				elective		breech	36 weeks		male	3.1 kg											
150	1033463	22	R	Rural	1	22	Primi								24	6.10%															emergency	CPD	39 weeks		Female	3.2 kg											
151	1033832	32	R	Rural	2	24.6	G3P1L1A1	Yes			yes			yes	26	5.80%	4.3		yes												emergency	previous lscs	36 weeks		male	2.5 kg											

152	1033942	34	R	Rural	1	24.6	G3P2L2								22	5.90%	1.2	yes			yes			oligohydramnios							emergency	previous lscs	38 weeks 2 days		Female	2.1 kg								yes			
153	1033705	30	UR	Rural	1	23.6	G3P2L2								24	5.40%	1.8	yes													emergency	non progress of labour	36 weeks 3 days		male	3 kg					yes	yes					
154	1034214	29	R	Rural	2	22.2	G3A2	Yes							28	5.60%	2.4		yes		yes									elective		precious pregnancy	37 weeks 5 days		Female	3 kg											
155	1034259	30	R	Rural	1	22.1	G2P1L1								30	5.30%	2.3				yes			polyhydramnios							emergency	macrosomia with polyhydramnios	35 weeks 6 days		Female	2.8 kg	yes		yes	yes		yes					
156	1034667	24	R	Rural	2	22.6	Primi								27	5.10%	2.5	yes												elective		Failed induction	38 weeks		male	2.7 kg											
157	1034931	28	R	Urban	1	22.8	Primi								18	5.20%	2.6		yes				yes	olyhydramnios							emergency	non reassuring NST	37 weeks 4 days		male	2.6 kg											
158	1036024	22	R	Urban	2	22.4	G2P1L1								22	5.70%	2.2	yes													emergency	previous LSCS	38 weeks		Female	2.7kg											
159	1036416	27	R	Urban	2	22.4	Primi								20	5.00%	2.6				yes										emergency	MCDA twins with severe PE	34 weeks	twin A - male Twin B A - 2.2 KG Twin B - 2 kg													
160	1036413	32	UR	Urban	2	22.1	G2A1	Yes							24	5.30%	3		yes							UTI					emergency	CPD	37 weeks 5 days		male	2.7 kg	yes		yes		yes						
161	1036623	20	UR	Urban	2	24	Primi								23	5.30%	3.1	yes			yes			polyhydramnios							emergency	antepartum eclampsia	38 weeks		Female	2.6 kg	yes				yes						
162	1037366	36	UR	Urban	1	27	G4P2L2A1	Yes							8	5.10%	3.4	yes													emergency	fetal distress	37 weeks 6 days		male	3 kg											
163	1037619	24	R	Urban	1	25	G2P1L1								16	5.60%	3.1	yes													emergency	fetal distress	38 weeks 3 days		male	2.6 kg	yes				yes						
164	1037339	24	R	Rural	1	24	Primi								20	5.30%	2.8	yes				yes									emergency	precious pregnancy	33 weeks 2 days		male	3.2 kg	yes	b/l CTEV				yes					
165	1037814	30	R	Urban	1	24.9	Primi								20	5.60%	2.5	yes			yes										emergency	fetal distress	37 weeks 1 day		male	2.9 kg	yes				yes						
166	1039742	30	UR	Rural	2	23.7	G4P1L1A2	Yes							26	5.30%	2.2	yes				yes									emergency	cord presentation	35 weeks 3 days		Female	2.8 kg						yes					
167	1039295	23	UR	Urban	2	25.8	G2A1	Yes							25	5.70%	3.2		yes												emergency	non progress of labour	35 weeks 2 days		Female	2.4 kg											
168	1040663	28	UR	Rural	2	20	G3P2L2							yes	22	5.30%	4.3	yes													emergency	previous 2 lscs	34 weeks		Female	2.2 kg	yes		yes		yes	yes					
169	1041582	28	UR	Urban	2	22	G2P1L1					yes			20	5.40%	3.1	yes								uti				elective		previous lscs	37 weeks		male	2.9 kg											
170	1041619	27	R	Rural	2	22.5	Primi							yes	18	5.50%	4.1	yes			yes										emergency	severe PE	39 weeks		male	3.6 kg											
171	1041796	21	R	Urban	2	19	G2A1	Yes							28	5.40%	2.5	yes													emergency	MSL	38 weeks 4 days		Female	2.8 kg											
172	1041787	30	R	Rural	2	24	G4P2L2A1	Yes							18	5.80%	2.6	yes													emergency	thick MSL	37 weeks 4 days		Female	3.1 kg											
173	1042182	23	R	Urban	2	25.9	Primi								10	5.30%	2.4	yes													emergency	thick MSL	39 weeks 2 days		male	3.3 kg											
174	1043199	25	R	Urban	2	26	G2P1L1								24	5.80%	2.5	yes									spontaneous						37 weeks 1 day		male	2.9 kg			yes								
175	1043557	39	R	Rural	2	24.8	G5P2L2A2	Yes							26	5.30%	2.6	yes			yes									elective		previous lscs	38 weeks 1 day		Female	3 kg											
176	1043613	25	UR	Rural	2	24	Primi								25	5.20%	2.9	yes									spontaneous						40 weeks		Female	2.8 kg											
177	1044042	30	UR	Rural	1	26	G2A1	Yes							24	5.30%	3	yes								UTI					emergency	CPD	40 weeks		Female	3.1kg	yes			yes							
178	1043625	31	UR	Urban	1	26	Primi								26	5.20%	3.2														emergency	CPD	36 weeks 6 days		male	2.5 kg	yes		yes		yes						
179	1013646	23	R	Rural	1	28	Primi								25	5.10%	1.2	yes									spontaneous						38 weeks 4 days		Female	2.8 kg						yes					
180	1045420	33	R	Urban	2	22	G2P1L1								16	5.80%	2.2		yes					polyhydramnios						elective		previous lscs	33 weeks 5 days		Female	2.8 kg	yes				yes	yes					
181	1004254	27	R	Urban	2	24	G4A3	yes							24	5.30%	3	yes													emergency	fetal distress	41 weeks 3 days		Female	2.9 kg											
182	1004823	35	R	Urban	2	26	G3P1L1A1	yes							14	5.40%	3.2	yes												elective		previous lscs	38 weeks		Female	3.1kg											
183	1005504	30	R	Urban	1	24	Primi							yes	26	5.10%	5.2		yes												emergency	persistent fetal tachycardia	36 weeks 6 days		Female	2.4kg	yes		yes		yes						
184	1005633	31	UR	Urban	2	20	Primi								24	5.60%			yes											elective		precious pregnancy	38 weeks 1 day		Female	2.7kg											
185	1005788	24	UR	Urban	2	25	Primi								24	5.40%		yes													emergency	fetal distress	39 weeks		Female	2.8 kg					yes						
186	1008499	26	R	Urban	2	27	Primi								26	5.30%		yes													emergency	thick MSL	39 weeks 4 days		Female	3kg											
187	1009211	31	UR	Rural	2	25	G3P1L1A1	yes							18	5.20%		yes													emergency	previous lscs	39 weeks		male	3.4 kg											
188	1010344	32	R	Rural	2	24	G2P1L1							yes	12	5.70%	5.1		yes											elective		previous lscs	38 weeks 2 days		male	2.7 kg	yes							yes			
189	1011101	24	R	Rural	2	27	Primi							yes	24	5.40%	5.2	yes								UTI		induced					36 weeks	IUD (MSB)	Female	1.9kg											
190	1011249	26	R	Urban	2	25	G2P1L1								24	5.30%		yes					yes			uti	spontaneous	ntouse delivery (poor maternal bear down)					36 weeks 6 days		male	2.9 kg	yes										
191	1011633	22	R	Urban	2	22	Primi								20	5.20%		yes													emergency	fetal distress	37 weeks		male	2 kg	yes				yes			yes			
192	1012282	40	UR	Urban	2	24	Primi							yes	16	5.7	4.1		yes				yes								emergency	precious pregnancy with pprom	32 weeks 1 day		Male	2 kg	yes						yes				
193	1012285	26	UR	Urban	2	23	G2P1L0		yes						24	5.20%		yes					yes								emergency	prolonged pprom	37 weeks 2 days		female	2.3 kg											
194	1012720	30	R	Urban	1	21	Primi								20	5.10%		yes			yes										emergency	antepartum eclampsia	28 weeks 1 day		male	1.1kg	yes				yes			yes			
195	990218	32	UR	Urban	1	25	G3P1L1A1	Yes							24	5.30%		yes										induced					37 weeks 1 day		female	2 kg											
196	990902	26	R	Rural	1	27	Primi								26	5.50%		yes													emergency	Failed induction	39 weeks 1 day		Female	2.5 kg											
197	993501	26	UR	Rural	1	24	Primi								24	5.70%		yes						polyhydramnios						elective		macrosomia with polyhydramnios	38 weeks		Female	3.7 kg	yes		yes			yes					
198	993769	24	R	Rural	2	22	Primi								8	5.90%		yes			yes							induced	y (poor maternal bear down)				38 weeks 1 day		Female	3 kg											
199	993918	29	UR	Urban	2	25	G2A1	Yes							16	5.20%			yes					polyhydramnios		vulvovaginitis					emergency	uncontrolled sugars	34 weeks 5 days		Female	3 kg						yes					
200	996694	24	UR	Urban	2	24	Primi								24	5.40%			yes				yes								emergency	prolonged pprom	29 weeks 6 days		male	1.4 kg	yes						yes				
201	996898	30	R	Urban	2	22	Primi								24	5.80%	2.3	yes												elective		GDM with precious pregnancy	38 weeks		female	3.3 kg											
202	999320	30	R	Urban	2	21	Primi								26	5.30%	3.1	yes			yes				yes						emergency		34	IUD (FSB)	female	1.8kg											
203	999051	24	R	Rural	2	23	Primi								22	5.60%	1.9	yes													emergency	Deep transverse arrest	37 weeks +1		male	3.2 kg											
204	1000611	21	UR	Rural	2	24	Primi								10	4.80%	2.5	yes									spontaneous						40 weeks		male	3.2 kg			yes								
205	1011211	29	UR	Rural	1	21	G2P1L1								28	3.60%	2.5	yes			yes										emergency	severe PE	38 weeks +2		male	3.1 kg											
206	1011349	30	UR	Urban	1	21.4	G2P1L1								30	4.80%	3.1	yes													emergency	thick MSL	40 weeks +6		Female	3.1 kg	yes				yes						
207	1011346	33	R	Urban	1	20	Primi								27	5.40%	3.2	yes								UTI				elective		precious pregnancy	36 weeks 2 days		male	2.3 kg	yes						yes				
208	1011462	30	R	urban	1	24	G2A1	Yes						yes	30	6.00%	6.1										spontaneous	instrumental (poor maternal bear down)					38 weeks + 4		Female	3.1 kg											
209	1011678	24	R	Urban	2	25	G2A1	Yes							22	6.10%	2.1	yes					YES								emergency	non progress of labour	37 weeks + 4		male	2.8 kg			yes								
210	1011650	26	R	Urban	2	25	G4P1L0A2	YES							20	6.20%	3.2	yes								uti				elective		previous lscs	37 weeks 6 days		male	3.4 kg											
211	1012009	23	R	Urban	2	24	G2A1	Yes							24	5.80%	2.5	yes						oligo hydramnios				induced					39 weeks 5 days		female	2.2 kg								YES			
212	1011846	20	UR	Urban	2	24.9	Primi								23	5.50%	2.4	yes						oligo hydramnios				induced					39 weeks		Female	2 kg								yes			
213	1012078	27	UR	Urban	2	23.7	G4P1L1A2	Yes							24	5.40%	2.2		yes												emergency	Failed induction	39 weeks		male	2.6 kg	yes		yes								
214	1012866	28	UR	Urban	2	25.8	G3P2L2								16	5.30%	3.1		yes												emergency	previous 2 lscs	37 weeks +2		male	2.9 kg	yes			yes							
215	1012684	23	R	Urban	2	20	G4P1L1A2	Yes						yes	20	5.40%	5.3	yes									spontaneous						36 weeks 4 ddays		Male	3 kg											
216	1013236	36	UR	Rural	2	22	G2P1L1								20	5.80%	3.1	yes												elective		previous lscs	37 weeks		female	2.3 kg											
217	1013242	32	R	Rural	2	22.5	G5P1L1A3	Yes		yes			yes		26	5.60%	3.2	yes												elective		previous lscs	38 weeks 1 day		male	3.1 kg											
218	1013303	37	UR	Rural	2	19	G3P1L1A1	Yes							25	4.30%	2.4	yes								UTI				elective		previous lscs	38 weeks 1 day		Male	3 kg											
219	1013596	32	UR	Rural	2	24	Primi								22	5.10%	2.2	yes													emergency	thick MSL	40 weeks 2 days		male	3.5 kg	yes			yes							
220	1013895	23	UR	Urban	2	25.9	Primi							yes	20	5.30%	5.6	yes												elective		CPD	39 weeks		Female	3.4 kg											
221	1014364	26	UR	Urban	2	26	G2P1L1								18	5.60%	2.3	yes												elective		previous lscs	38 weeks 1 day		Male	3.1 kg											
222	1014534	32	UR	Urban	2	24.8	Primi								28	5.10%	2.1		yes												emergency	Failed induction	38 weeks 2 days		Male	2.7 kg											