"PREVALENCE OF GESTATIONAL DIABETES MELLITUS USING IADPSG AND DIPSI CRITERIA - A CROSS SECTIONAL STUDY"

REG.NO. BJ0112006

Dissertation

Submitted to the
KLE University, Belgaum, Karnataka

In Partial Fulfillment
of the requirements for the degree of

MASTER OF SURGERY
in
OBSTETRICS AND GYNAECOLOGY

**DEPARTMENT OF OBSTETRICS AND GYNAECOLOGY,
JAWAHARLAL NEHRU MEDICAL COLLEGE,
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ENDORSEMENT

This is to certify that the dissertation entitled **"PREVALENCE OF GESTATIONAL DIABETES MELLITUS USING IADPSG AND DIPSI CRITERIA – A CROSS SECTIONAL STUDY"** is a bonafide research work done by **CANDIDATE REG NO. BJ0112006**.

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LIST OF ABBREVIATIONS USED

β	-	Beta
ACOG	-	American College of Obstetricians and Gynaecologists
ADA	-	American Diabetes Association
BMI	-	Body mass index
CC	-	Carpenter and Coustan
CDA	-	Canadian Diabetes Association
DIAPA	-	Diabetes in Pregnancy Awareness and Prevention
DIPSI	-	Diabetes in pregnancy study group of India
DM	-	Diabetes mellitus
e.g.,	-	For example
ESRD	-	End-stage renal disease
FPG	-	Fasting plasma glucose
GCT	-	Glucose challenge test
GDM	-	Gestational diabetes mellitus
GIGT	-	Gestational impaired glucose tolerance
gms	-	Grams
GOD	-	Glucose oxidase peroxidase
h	-	Hour
HAPO	-	Hyperglycemia and Adverse Pregnancy Outcome
HbA1c	-	Glycated haemoglobin
HELLP	-	Hemolysis, elevated liver enzymes, and low platelet count
hPL	-	Human placental lactogen
i.e.	-	That is,

IADPSG	-	International Association of Diabetes in Pregnancy Study Groups
IDMs	-	Infants of diabetic mothers
IFG	-	Impaired fasting glucose
IGT	-	Impaired glucose tolerance
IOM's	-	Institute of Medicine's
IQWiG	-	Institute for Quality and Efficiency in Health Care
kg	-	Kilogram
LGA	-	Large for gestational age
m^2	-	Square meter
mg	-	Milligram
mg/dL	-	Milligram per deciliter
mL	-	Millilitre
mmol/L	-	Millimole per liter
n	-	Total number
NDDG	-	National Diabetes Data Group
NGT	-	Normal glucose tolerance
ob	-	Obesity
OGTT	-	Oral glucose tolerance test
p	-	Probability
PCOS	-	PolysCystic Ovarian Syndrome
PG	-	Plasma glucose
RDS	-	Respiratory distress syndrome
SD	-	Standard deviation
T2DM	-	Type 2 diabetes mellitus

USPSTF	-	United States Preventive Services Task Force
vs	-	Versus
WHO	-	World Health Organization

ABSTRACT

Background and objective

Universal screening and care of women with GDM is of paramount public health priority in high risk population for GDM and diabetes. This study was aimed to find the concordance between the present practice of diagnosing GDM by DIPSI criterion of 2-h PG ≥ 140 mg/dL and IADPSG recommendation.

Methodology

This cross sectional study was done at Antenatal Clinic, Department of Obstetrics and Gynaecology, KLES Dr Prabhakar Kore Hospital and Medical Research Center, Belgaum from January 2013 to December 2013. A total of 225 pregnant women between 24 to 28 weeks gestations were studied. Diagnosis and the prevalence of GDM were assessed by applying both DIPSI and IADPSG criteria.

Results

In the present study most of the women (58.11%) were age between 22 to 25 years and the mean age was 23.78±3.38 years. Most of the women reported gravida two (46.22%) The gestational age was 26 weeks in 26.67% of the women and mean gestational age was 26.25±2.70 weeks. Majority of the women (74.22%) had body mass index between 19.8 to 25.99 Kg/m^2 and mean body mass index was 22.83±3.75 kg/m^2. The fasting, one hour and two hours plasma glucose levels were ≥92, ≥180 and ≥153 mg/dL in 9.33%, 8.99% and 7.56% of the women respectively and at same intervals the mean fasting plasma glucose levels were 80.35±17.37, 122.90±31.96 and 107.76±29.51 mg/dL respectively.

Conclusion

Based on the IADPSG criteria, the prevalence of GDM was 19.11% and by applying DIPSI criteria that is ≥140 mg/dL plasma glucose levels at two hours, prevalence of GDM was 16.89%. The difference in diagnostic capability between IADPSG and DIPSI was found to be 2.8% and the kappa statistics showed good strength of agreement between the two tests ($p>0.302$; Kappa= 0.774). Hence it may be concluded that, the diagnosis GDM based on DIPSI is as effective as IADPSG criteria.

Keywords:

Diabetes in Pregnancy Study Group of India criteria (DIPSI); Gestational diabetes mellitus; International Association of Diabetes in Pregnancy Study Groups criteria (IADPSG);

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INTRODUCTION

In most Asian countries, the economic prosperity is increasing. This has implications for the way people live, what they eat and patterns of disease they experience. China, India and several South East Asian nations are experiencing a rise in obesity and diet related non-communicable diseases. Type 2 diabetes mellitus has been documented to be increasing in Asia,[1] although the increase seems to be greater in South Asia compared to East and South East Asia.[2] It was estimated that globally in 2011, 366 million people were living with diabetes. This is predicted to rise to 552 million people by 2030 with half of these living in Asia.[3]

Gestational diabetes mellitus (GDM) is defined as glucose intolerance first discovered in pregnancy. It is carbohydrate intolerance with onset or recognition during pregnancy.[4]

Depending on the diagnostic criteria used and the population screened, the prevalence of GDM ranges from 1.1 to 25.5% of pregnancies in the United States.[5-9] In 2009 the Centers for Disease Control and Prevention reported a prevalence of 4.8% of diabetes in pregnancy. An estimated 0.5% of these cases likely represented women with pregestational diabetes. Data from the International Hyperglycemia and Adverse Pregnancy Outcome (HAPO) Study[7] indicate that 6.7% of the women met a fasting plasma glucose threshold of 95 mg/dL (5.3 mmol/L), which is in keeping with the Carpenter and Coustan[9] (CC) criteria that are in common practice in North America. In contrast, 17.8% of women were diagnosed with GDM using the International Association of Diabetes in Pregnancy Study Groups (IADPSG) criteria[10] in which lower glucose thresholds are proposed to diagnose GDM.[5] The

prevalence of GDM in India varies from 3.8 to 21% in different parts of the country, depending on geographical locations and diagnostic methods used.[11-14]

Gestational diabetes mellitus occur in women in whom beta cell function is not able to overcome the antagonism created by anti insulin hormones of pregnancy and the increased fuel consumption required to provide for the growing feto maternal unit. Insulin is detectable in fetal pancreas as early as nine weeks after conception.[15] An increase in beta cell mass and insulin secretion in the fetus occurs by the 16th week of gestation, in response to maternal hyperglycemia.[16] The priming of the fetal beta cells may account for the persistence of fetal hyperinsulinaemia throughout pregnancy and risk of accelerated fetal growth,[17] even when the mother enjoys good metabolic control in later pregnancy.[18] This necessitates performing the test procedures to diagnose GDM in the first trimester itself. Further, early detection and care results in a better fetal outcome.[19]

Early diagnosis and treatment of GDM can reduce adverse pregnancy outcomes, including stillbirth, neonatal macrosomia, neonatal hypoglycaemia, birth trauma and neonatal respiratory distress syndrome as well as decrease the risk of preeclampsia in the mother.[3]

Studies conducted in different populations and with different methodologies, consistently reported an increase in GDM in all race/ethnicity groups, suggesting that there is an increase in GDM prevalence. A true increase in the prevalence of GDM aside from its adverse consequences for the infant in the newborn period might reflect or contribute to the ongoing pattern of increasing diabetes and obesity. This implies that Universal screening and care of women with GDM is of paramount

public health priority in high risk population for GDM and diabetes like Asian Indians, rather than risk factor screening. In this aspect, except the existing diagnostic criterion of World Health Organization (WHO) 2-h plasma glucose (PG) ≥ 140 mg/dL with 75g oral glucose load, other diagnostic criteria are country specific or recommended by various associations.[20]

Currently Diabetes in Pregnancy Study Group of India (DIPSI) criteria[21] is being used commonly in the community as it is difficult to get the patients in fasting state but since in a tertiary care hospital where patients come for regular antenatal follow up it is possible for them to report back during 24 to 28 weeks in a fasting state therefore this criteria can also be adopted to find the prevalence of GDM.

Recently, based on the Hyperglycemia and Adverse Pregnancy Outcome (HAPO) study, the International Association of Diabetes and Pregnancy Study[7] Groups (IADPSG) consensus panel recommended that GDM can be diagnosed, if any one value of fasting plasma glucose (FPG), 1-h and 2-h plasma glucose (PG) concentrations meet or exceed 92 mg/dL, 180 mg/dL and 153 mg/dL respectively, with 75g oral glucose tolerance test (OGTT).[10] India one of the most populous countries in the world was not a part of the HAPO study.

The HAPO (Hyperglycemia and adverse pregnancy outcome) study[7] was performed in response to the need for internationally agreed upon diagnostic criteria for GDM, based on their predictive value for adverse pregnancy outcome increases with each of the three values on the 75 gms, 2 hour oral glucose tolerance test are associated with increase in the likelihood of pregnancy outcomes such as large for gestational age, cesarean section, fetal insulin levels and neonatal fat content.

The IADPSG (International association of diabetes in pregnancy study groups) recommends that the diagnosis of GDM be made when any of the following 75 gram of oral glucose, fasting, 1 hour, 2 hour oral glucose tolerance test (OGTT) thresholds are met or exceeded; fasting 92 mg/dL, 1 hour 180 mg/dL, or 2 hours 153 mg/dL.[10,22]

Considering the magnitude of adverse pregnancy outcomes related to gestational diabetes the present study was undertaken to find out the prevalence of gestational diabetes mellitus using the IADPSG and DIPSI criteria[10,22] to ascertain whether the present practice of diagnosing GDM by the guidelines recommended by Diabetes In Pregnancy Study Group India (DIPSI)[21] based on WHO criterion of 2-h PG ≥ 140 mg/dL can still be followed in our settings or adopt IADPSG recommendation.

OBJECTIVES

The objective of the present study was to find out the prevalence of Gestational Diabetes Mellitus using the International Association of Diabetes in Pregnancy Study Groups and Diabetes in Pregnancy Study Group of India criteria in KLES Dr. Prabhakar Kore Hospital and Medical Research Center, Belgaum.

REVIEW OF LITERATURE

Diabetes mellitus refer to a group of common metabolic disorder that shares the phenotype of hyperglycemia. Distinct types of DM exist and are caused by a complex interaction of genetics and environmental factors. Depending on the etiology of the DM, factors contributing to hyperglycemia include reduce insulin secretion, decreased glucose utilization, and increased glucose production.[23]

The metabolic dysregulation associated with DM causes secondary pathophysiologic changes in multiple organ systems that impose burden on the individual with diabetes and on the health care system. In the United States, DM is the leading cause of end-stage renal disease (ESRD), non traumatic lower extremity amputations, and adult blindness. It also predisposes to cardiovascular diseases. With an increasing incidence worldwide, DM will be leading cause of morbidity and mortality for the future.[23]

Classification of diabetes mellitus

DM is classified on the basis of the pathogenic process that leads to hyperglycemia, as opposed to earlier criteria such as age of onset or type of therapy. The two broad categories of DM are designated as[24]

- Type 1
- Type 2

Both types of diabetes are preceded by a phase of abnormal glucose homeostasis as the pathogenic processes progresses. Type 1 diabetes is the result of

complete or near-total insulin deficiency. Type 2 DM is a heterogeneous group of disorders characterized by variable degrees of insulin resistance, impaired insulin secretion, and increased glucose production. Distinct genetic and metabolic defects in insulin action and/or secretion give rise to the common phenotype of hyperglycemia in type 2 DM and have important potential therapeutic implications now that pharmacologic agents are available to target specific metabolic derangements. Type 2 DM is preceded by a period of abnormal glucose homeostasis classified as impaired fasting glucose (IFG) or impaired glucose tolerance (IGT).[24]

Spectrum of glucose homeostasis and diabetes mellitus[25]

Type of diabetes	**Normal glucose tolerance (NGT)**	**Impaired fasting glucose or impaired glucose tolerance**	**Hyperglycemia**		
			Diabetes mellitus		
			Not insulin required	**Insulin required for control**	**Insulin required for survival**
Type 1					
Type 2					
Other Specific types					
Gestational diabetes					
Time (years)					
FPG (mg/dl)	< 100	100-125		≥ 126	
2-h plasma glucose (mg/dl)	< 140	140 – 199		≥ 200	

Etiologic classification of diabetes mellitus[24]

I. Type 1 diabetes (-cell destruction, usually leading to absolute insulin deficiency)

A. Immune-mediated

B. Idiopathic

II. Type 2 diabetes (may range from predominantly insulin resistance with relative insulin deficiency to a predominantly insulin secretory defect with insulin resistance)

III. Other specific types of diabetes

IV. Gestational diabetes mellitus (GDM)

GESTATIONAL DIABETES MELLITUS

History

Gestational diabetes (GD) as a clinical entity officially began in 1979 when the National Diabetes Data Group (NDDG) issued an updated classification of diabetes types, including one that was present only during pregnancy. In 1979, the First International Workshop-Conference on GDM also met, essentially declared GD a disease, finding it a significant health risk that needed treatment. Instead of the more neutral "Carbohydrate Intolerance of Pregnancy", the term "Gestational Diabetes Mellitus" was used .[24]

Hadden (1998) reports incidents in the medical literature appearing as early as 1823 where diabetic-like conditions presented during pregnancy but seemed to

disappear afterwards. However, greater attention to the concept that lesser degrees of hyperglycemia might negatively affect a pregnancy began to appear in the 1940s and 1950s. In these studies, it was found that there is increased perinatal mortality among the babies of women who developed diabetes years later, leading to the coining of the term "prediabetes in pregnancy."[26]

The first major prospective study was established in Boston in 1954, and the one hour 50 gm glucose screening test was first used there. However, the emphasis was on criteria that established risk for future diabetes, not on risk to the fetus. The results from this Boston study were presented by O'Sullivan and Mahan in 1964, and showed that higher blood glucose values in pregnancy correlated with the development of diabetes later in life.[26]

Definition

It is now defined as "*Carbohydrate intolerance of variable severity with onset or first recognition during the present pregnancy. The definition applies whether insulin is used for treatment or the condition persists after pregnancy but does not exclude the possibility that the glucose intolerance may have antedated the pregnancy"*.[27]

According to WHO, GDM is defined as plasma glucose concentration of greater than 140 mg/dL at two hours after an 75 gm of oral glucose tolerance test similar to that of impaired glucose tolerance test in a non pregnant state.[28]

Epidemiology

Prevalence

Global statistics

GDM is the most common metabolic disease of pregnancy. GDM affects an estimated 1,70,000 (1-14%) pregnancies each year in the United States, depending on the diagnostic criteria used and characteristics of the population.[29]

30-50% of women with GDM will have recurrent GDM in a future pregnancy.[30]

Of particular concern, 20-50% of women with GDM will develop type 2 diabetes mellitus (T2DM) in the 5-10 years after delivery.[29]

Recent meta-analysis reports that GDM corresponds to a 7.4 fold increased risk for developing T2DM.[30]

Indian context

The scenario in India today is that there are over 43 million diabetics in the country. This makes India the diabetes capital of the world, with half the diabetic population being women. There are 14 million women in India, in the age group of 20 to 39 years who are considered in the child bearing age. In Indian context the prevalence of GDM is steadily increasing from two percent in 1982 to 12% in 1991 and it has almost doubled to 16.55% in 2002.

In India, a community based study Diabetes in Pregnancy Awareness and Prevention (DIPAP), was performed to ascertain the prevalence of GDM in a cohort

of 12,056 pregnant women living in urban, semi - urban, and rural areas by using WHO criteria. Among them, the overall prevalence of GDM was 13.9%.[31]

To ascertain the consistency of WHO criteria in diagnosing GDM, after determining the desired sample size with the required statistical power, a total of 1246 pregnant women underwent 75gm OGTT. Among them 13.2% were detected to have GDM with a two hour PG ≥ 140 mg/dL. This finding substantiates and validates the previous prevalence data as well as the WHO criteria. Thus 2 hour plasma glucose ≥ 140 mg with 75 gm oral glucose load has been accepted by the Diabetes in pregnancy Study group India (DIPSI) for diagnosing GDM.[31]

The prevalence of GDM in India varied from 3.8 to 21.0% in different parts of the country, depending on the geographical locations and diagnostic methods used.[11-15]

Risk Factors

Risk factors for gestational diabetes vary from study to study, but some remain consistent. These are listed as 'strong' associations solely due to their consistency of appearance in each study, and are put near the top. Others whose associations are less clear are listed towards the bottom.[32]

- Family history of diabetes
- Parity (number of kids, especially 3-4 or more)
- Previous pregnancy with GDM
- Obesity
- Previous child over 4000 g (almost 9 lbs)

- Women whose own birth weights were over 9 lbs.
- Unexplained multiple miscarriages, stillbirths, or birth defects
- Weight gain in early adulthood
- Central fat distribution
- PCOS (PolysCystic Ovarian Syndrome)
- Cigarette smoking
- Multiple Pregnancies
- History of Skin/Urinary Tract/Genital Infections
- Hypertension
- Chronic Steroid Use
- Non-white ethnicity

The risk in *Asians* is less clear. Southeast Asians had increased rates, while Korean women had very low rates. Chinese women clearly had increased rates, especially if they were immigrants, but those in China also had slightly higher rates too. Japanese-Americans also have increased rates.[32]

Women from India had very high rates in some areas, second only to those of Native Americans. However, not all areas of India showed such high rates. *Arabic* women also had slightly increased rates of GDM.[32]

Pathophysiology

Maternal-fetal metabolism in normal pregnancy

With each feeding, the pregnant woman undergoes a complex series of maternal hormonal actions (a rise in blood glucose; the secondary secretion of

pancreatic insulin, glucagon, somatomedins, and adrenal catecholamines). These adjustments ensure that an ample, but not excessive, supply of glucose is available to the mother and fetus.[33]

The key features of this complex interaction include compared to nonpregnant subjects, pregnant women tend to develop hypoglycemia (plasma glucose mean = 65 to 75 mg/dL) between meals and during sleep. This occurs because the fetus continues to draw glucose across the placenta from the maternal bloodstream, even during periods of fasting. Interprandial hypoglycemia becomes increasingly marked as pregnancy progresses and the glucose demand of the fetus increases.[33]

Levels of placental steroid and peptide hormones (estrogens, progesterone, and chorionic somatomammotropin) rise linearly throughout the second and third trimesters. Because these hormones confer increasing tissue insulin resistance as their levels rise, the demand for increased insulin secretion with feeding escalates progressively during pregnancy. Twenty-four–hour mean insulin levels are 50% higher in the third trimester compared to the nonpregnant state.[33]

If the maternal pancreatic insulin response is inadequate, maternal and, then, fetal hyperglycemia results. This typically manifests as recurrent postprandial hyperglycemic episodes. These postprandial episodes are most significantly accountable for the accelerated growth exhibited by the fetus.[33]

Surging maternal and fetal glucose levels are accompanied by episodic fetal hyperinsulinemia. Fetal hyperinsulinemia promotes excess nutrient storage, resulting

in macrosomia. The energy expenditure associated with the conversion of excess glucose into fat causes depletion in fetal oxygen levels.[33]

These episodes of fetal hypoxia are accompanied by surges in adrenal catecholamines, which, in turn, cause hypertension, cardiac remodelling and hypertrophy, stimulation of erythropoietin, red cell hyperplasia, and increased hematocrit. Polycythemia (hematocrit >65%) occurs in 5-10% of newborns of diabetic mothers. This finding appears to be related to the level of glycemic control and is mediated by decreased fetal oxygen tension. High hematocrit values in the neonate lead to vascular sludging, poor circulation, and postnatal hyperbilirubinemia.[33]

During a healthy pregnancy, mean fasting blood sugar levels decline progressively to a remarkably low value of 74 ± 2.7 mg/dL. On the other hand, peak postprandial blood sugar values rarely exceed 120 mg/dL. Meticulous replication of the normal glycemic profile during pregnancy has been demonstrated to reduce the macrosomia rate. Specifically, when two postprandial glucose levels are maintained less than 120 mg/dL, approximately 20% of fetuses demonstrate macrosomia. Conversely, if postprandial levels range up to 160 mg/dL, macrosomia rates rise to 35%.[33]

Pregnancy – The diabetogenic state

The fuel metabolism during normal (non-diabetic) pregnancy is characterized by:

- Facilitated insulin action during the first half of pregnancy.
- Diabetogenic stress during the second half of pregnancy.

Carbohydrate metabolism in early pregnancy (<20 weeks)

Hormonal alteration	Effect	Metabolic change
↑ Estrogen and progesterone ↓ Beta cell hyperplasia ↓ ↑ Insulin secretion	↑ Hepatic glycogen synthesis ↓ ↑ Hepatic glycogenolysis ↓ ↑ Peripheral glucose utilization	Hypernsulinemia ↓ 10% ↓ in fasting plasma glucose value

Carbohydrate metabolism in late pregnancy (>20 weeks)

Hormonal alteration	Effect	Metabolic change
↑ Human placental lactogen (hPL) ↓ ↑ Prolactin ↓ ↑ Bound and free cortisol	↑ Insulin resistance (Post receptor defects) ↓ ↑ Hepatic glycogenolysis ↓ ↑ Hepatic glycogen synthesis	Facilitated anabolism in fed state ↓ Accelerated starvation in fasting state ↓ Ensure glucose and amino acid to fetus

Pathogenesis of glucose intolerance in pregnancy

A. Genetic factors

   Increasing maternal age and obesity

B. Gestational factors

- Autoimmune destruction of islet cells
- Impaired beta cell function
- Antagonistic effect of pregnancy hormones
- Increased insulin degradation
- Impaired insulin – Receptor binding

- Post – Receptor defect in insulin signaling cascade mediated by TNF-α.

C. Role of Leptin

Leptin, a product of obesity (ob) gene, is produced and secreted by the adipose tissue. Its plasma levels are significantly elevated in pregnant than in non pregnant women, indicating pregnancy to be a leptin – resistant state.

Effects of diabetes on fetus

Miscarriages

In all women with preexisting diabetes mellitus, there is a 9-14% rate of miscarriage. Current data suggest a strong association between degree of glycemic control prior to pregnancy and miscarriage rate. Suboptimal glycemic control has been shown to double the miscarriage rate in women with diabetes. A correlation also exists between more advanced diabetes and miscarriage rates. Patients with long-standing (>10 y) and poorly controlled (glycohemoglobin exceeding 11%) diabetes have been shown to have a miscarriage rate of up to 44%. Conversely, reports demonstrate a normalization of miscarriage rate with excellent glycemic control.[34]

Birth defects

Among the general population, major birth defects occur in one to two percent of the population. In women with overt diabetes and suboptimal glycemic control prior to conception, the likelihood of a structural anomaly is increased four to eight fold. Although initial reports demonstrated anomaly rates as high as 18% in women with preexisting diabetes mellitus,[35] more recent reports with more

aggressive preconception and first trimester management report anomaly rates between 5.1 and 9.8%.[36,37] Two-thirds of anomalies involve the cardiovascular and central nervous systems.

Neural tube defects occur 13 to 20 times more frequently in diabetic pregnancy. Genitourinary, gastrointestinal, and skeletal anomalies are also more common. The fact that no increase in birth defects occurs among the offspring of fathers who are diabetic and women who develop gestational diabetes after the first trimester is notable. This suggests that periconceptional glycemic control is the main determinant of abnormal fetal development in diabetic women.

When the frequency of congenital anomalies in patients with normal or high first-trimester maternal glycohemoglobin values was compared to the frequency in healthy patients, the rate of anomalies was only 3.4% with HbA1C of less than 8.5%, whereas patients with poorer glycemic control in the periconceptional period (HbA1C >8.5%) had a 22.4% rate of malformations. An overall malformation rate of 13.3% was reported in 105 patients with diabetes, but the risk of delivering a malformed infant was comparable to a normal population when the HbA1c was less than seven percent.[38] In a review of seven cohort studies, it has been found that patients with a normal glycohemoglobin (0 SD above normal), the absolute risk of an anomaly was two percent. At two SD above normal, this risk was 3%, with an odds ratio of 1.2 (1.1 to 1.4). As the glycohemoglobin increased the risk for malformation increased.[39]

Because birth defects occur during the critical three to six weeks after conception, nutritional and metabolic intervention must be initiated well before

pregnancy begins. It has been demonstrated in various clinical trials of intensive metabolic care that malformation rates similar to those in the nondiabetic population can be achieved with meticulous preconceptional glycemic control.[37] Subsequent trials comparing a preconceptional intensive metabolic program to standard treatment over 15 years duration have demonstrated lowered perinatal mortality (0% vs 7%) and reduced congenital anomaly rate (14% to 2%). In addition, when the preconceptional counseling program was discontinued, the congenital anomaly rate increased by over 50%.[40]

Growth restriction

Although most fetuses of diabetic mothers exhibit growth acceleration, growth restriction occurs with significant frequency in pregnancies in women with preexisting type 1 diabetes. The most important predictor of fetal growth restriction is underlying maternal vascular disease. Pregnant patients with diabetes-associated retinal or renal vasculopathies and/or chronic hypertension are most at risk for growth restriction.

Growth acceleration

Excessive body fat stores, stimulated by excessive glucose delivery during diabetic pregnancy, often extends into childhood and adult life.[41]

Approximately 30% of fetuses of women with diabetes mellitus in pregnancy are large for gestational age (LGA). In preexisting diabetes mellitus this incidence appears slightly higher, 38%. Maternal obesity, common in type 2 diabetes, appears to significantly accelerate the risk of infants being LGA.[41]

A study of the effects of weight gain in women with gestational diabetes found that women with the condition whose gestational weight gain was greater than that in the Institute of Medicine's (IOM's) weight-gain guidelines had an increased risk of preterm delivery, of having a newborn who was LGA, and of requiring a cesarean delivery.[42] The chance that a newborn would be small for gestational age was greater among women with gestational diabetes whose weight gain was below the IOM guidelines.

Fetal obesity

Macrosomia is typically defined as a birth weight above the 90th percentile for gestational age or greater than 4000 grams. In pregnant diabetic women, macrosomia occurs in 15 to 45% of cases, a three fold increase from normoglycemic controls.[43]

Newborns with macrosomia experience excessive rates of neonatal morbidity, as illustrated by a study by Hunter et al in 1993, which compared the neonatal morbidity among infants of 230 women with insulin-dependent diabetes and infants of 460 women without diabetes. The infants of diabetic mothers (IDMs) had five fold higher rates of severe hypoglycemia, a four fold increase in macrosomia, and a doubled increase in neonatal jaundice.[44]

Birth injury, including shoulder dystocia and brachial plexus trauma, are more common among infants of diabetic mothers, and macrosomic fetuses are at the highest risk.

The macrosomic fetus in diabetic pregnancy develops a unique pattern of overgrowth, involving central deposition of subcutaneous fat in the abdominal and

interscapular areas.[45] Skeletal growth is largely unaffected. Neonates of diabetic mothers have a larger shoulder and extremity circumference, a decreased head-to-shoulder ratio, significantly higher body fat, and thicker upper extremity skin folds compared to nondiabetic control infants of similar weights. Since fetal head size is not increased during poorly controlled diabetic pregnancy but shoulder and abdominal girth can be markedly augmented, the risk of injury to the fetus after delivery of the head (eg Erb palsy) is significantly increased.

When serial ultrasonographic examination findings from diabetic fetuses are plotted, the growth velocity of the abdominal circumference is often well above the growth centiles seen in nondiabetic fetuses and is higher than the fetal head and femur centiles. The accelerated growth of the abdominal circumference begins to rise significantly above normal after 24 weeks.

Metabolic syndrome

The adverse effects of abnormal maternal metabolism on the offspring have been documented well into puberty. Glucose intolerance and higher serum insulin levels are more frequent in children of diabetic mothers as compared to normal controls. By age 10 to 16 years, offspring of diabetic pregnancy have a 19.3% rate of impaired glucose tolerance.[46]

The childhood metabolic syndrome includes childhood obesity, hypertension, dyslipidemia, and glucose intolerance. A growing body of literature supports a relationship between intrauterine exposure to maternal diabetes and risk of a metabolic syndrome later in life.[46,47] Fetuses of diabetic women that are born large for gestational age appear to be at the greatest risk.[49]

Perinatal morbidity and birth injury

Perinatal mortality

In diabetic pregnancy, perinatal mortality has decreased 30-fold since the discovery of insulin in 1922 and intensive obstetrical and infant care in the 1970s. However, the current perinatal mortality rates among women who are diabetic remain approximately twice those observed in the nondiabetic population.[50]

Congenital malformations, respiratory distress syndrome (RDS), and extreme prematurity account for most perinatal deaths in contemporary diabetic pregnancies.[50]

Birth injury

Injuries of birth, including shoulder dystocia and brachial plexus trauma, are more common among infants of diabetic mothers, and macrosomic fetuses are at the highest risk.[34]

Most of the birth injuries occurring to infants of diabetic mothers are associated with difficult vaginal delivery and shoulder dystocia. While shoulder dystocia occurs in 0.3-0.5% of vaginal deliveries among healthy pregnant women, the incidence is two to four fold higher in women with diabetes. With strict glycemic control, the birth injury rate has been shown to be only slightly higher than controls (3.2 vs 2.5%).[34]

Polycythemia

A central venous hemoglobin concentration greater than 20 gm/dL or a hematocrit value greater than 65% (polycythemia) is comman in infants of diabetic mothers and is related to glycemic control. Hyperglycemia is a powerful stimulus to fetal erythropoietin production mediated by decreased fetal oxygen tension. Untreated neonatal polycythemia may promote vascular sludging, ischemia, and infarction of vital tissues, including the kidneys and central nervous system.[34]

Hypoglycemia

Approximately 15-25% of neonates delivered from women with diabetes during gestation develop hypoglycemia during the immediate newborn period.[51] Neonatal hypoglycemia is less frequent when tight glycemic control is maintained during pregnancy[52] and in labor. Unrecognized postnatal hypoglycemia may lead to neonatal seizures, coma, and brain damage.

Neonatal hypocalcemia

Up to 50% of infants of diabetic mothers have low levels of serum calcium (<7 mg/100 mL). With better management of diabetes in pregnancy, this occurrence has been reduced to 5% or less. These changes in calcium appear to be attributable to a functional hypoparathyroidism, though the exact pathophysiology is not well understood.[34]

Postnatal hyperbilirubinemia

Hyperbilirubinemia occurs in approximately 25% of infants of diabetic mothers, a rate approximately double that in a healthy population. The causes of hyperbilirubinemia in infants of diabetic mothers are many, but prematurity and

polycythemia are the major contributing factors. Increased destruction of red blood cells contributes to the risk of jaundice and kernicterus. Treatment of this complication is usually by phototherapy, but exchange transfusions may be necessary if bilirubin levels are markedly elevated.[34]

Respiratory problems

Neonatal RDS was the most common and serious morbidity in infants of diabetic mothers. In the 1970s, improved prenatal maternal management for diabetes and new techniques in obstetrics for timing and mode of delivery resulted in a decrease in its incidence from 31% to 3%.[53] However, respiratory distress syndrome continues to be a relatively preventable complication.

The majority of the literature indicates a significant biochemical and physiological delay in infants of diabetic mothers. Tyden[54] and Landon[55] and colleagues reported that fetal lung maturity occurred later in pregnancies with poor glycemic control regardless of class of diabetes when infants were stratified by maternal plasma glucose levels.

The nondiabetic fetus achieves pulmonary maturity at a mean gestational age of 34-35 weeks. By 37 weeks' gestation, more than 99% of healthy newborn infants have mature lung profiles as assessed by phospholipid assays. However, in a diabetic pregnancy, presuming that the risk of respiratory distress has passed is unwise until after 38.5 gestational weeks have been completed.

Prior to contemplating any delivery before 38.5 weeks for other than the most urgent fetal and maternal indications, perform an amniocentesis to document pulmonary maturity.

Effects of diabetes on mother

Diabetic retinopathy

It is the leading cause of blindness in women aged 24-64 years. Some form of retinopathy is present in virtually 100% of women who have had type 1 diabetes for 25 years or more; of these women, approximately 1 in 5 is legally blind. A prospective study showed that while half the patients with preexisting retinopathy experienced deterioration during pregnancy, all the patients had partial regression following delivery and returned to their prepregnant state by 6 months postpartum.[34]

Studies have suggested that rapid induction of glycemic control in early pregnancy stimulates retinal vascular proliferation.[56] However, when the total effect of pregnancy on ophthalmologic status was considered, women with pregnancies had a slower progression of retinopathy than nonpregnant women, probably because the modest deterioration in retinal status during rapid improvement in control is offset by the excellent control during the remainder of the pregnancy.

Current management recommendations include baseline ophthalmology referral for pregnant patients with diabetes, with follow-up according to degree of retinopathy.

Renal function

Patients with underlying nephropathy can expect varying degrees of deterioration of renal function during a pregnancy. As renal blood flow and glomerular filtration rate increase 30-50% during pregnancy, the degree of proteinuria will also increase.[34]

The most recent studies indicate that pregnancy does not measurably alter the time course of diabetic renal disease, nor does it increase the likelihood of progression to end stage renal disease. The progression to renal disease in diabetic patients appears to be related to duration of diabetes and degree of glycemic control.[34]

Perinatal complications are increased in patients with diabetic nephropathy. Preterm birth, intrauterine growth restriction, and preeclampsia are all significantly more common in women with diabetic nephropathy during pregnancy.[34]

Chronic hypertension

This complicates approximately 1 in 10 diabetic pregnancies overall. Patients with underlying renal or retinal vascular disease are at a substantially higher risk, with 40% having chronic hypertension.[57] Patients with chronic hypertension and diabetes are at increased risk of intrauterine growth restriction, superimposed preeclampsia, abruptio placentae, and maternal stroke.

Baseline renal function determination is recommended in all patients with preexisting diabetes. Renal function tests in each trimester should be performed in those with overt vascular disease or who have had diabetes for more than 10 years.[34]

Preeclampsia

Consists of abrupt rise in blood pressure, significant proteinuria, plasma uric acid levels greater than 6 mg/dL or evidence of hemolysis, elevated liver enzymes, and low platelet count (HELLP) syndrome. Preeclampsia is more frequent among women with diabetes, occurring in approximately 12% as compared to 8% of the

nondiabetic population. The risk of preeclampsia is also related to maternal age and the duration of preexisting diabetes. In patients who have chronic hypertension coexisting with diabetes, preeclampsia may be difficult to distinguish from near-term blood pressure elevations.[34]

The rate of preeclampsia has been found to be related to the level of glycemic control, with FPG less than 105 mg/dL, the rate of preeclampsia was 7.8%, if FPG was greater than 105 mg/dL, the rate of preeclampsia was 13.8%.[78] In this same study, pregravida body mass index (BMI) was also significantly related to the development of preeclampsia.

Screening and diagnosis

Screening tests[58]

The different screening tests used are

Fasting blood glucose

An easier screening procedure with cut-off of 95 mg/dL but it is insufficient as sole marker of GDM as most cases have fasting blood glucose below putative threshold. False positive rates are as high as 30 to 57%. Fasting blood glucose level of > 125 mg/dL is diagnostic of overt diabetes during pregnancy.[59,60]

Random blood glucose

Random blood sugar value greater than 200 mg/dL is diagnostic of diabetes during pregnancy and precludes the need for any glucose challenge test. The

diagnosis must be confirmed on a subsequent day in the absence of unequivocal hyperglycemia.[61]

Glucose challenge test (GCT)

This test is performed as routine out-patient procedure without regard to last meal time. Capillary blood glucose estimation is done 1 hour after giving 50 gm of glucose to the pregnant women between 24 to 28 weeks of gestation. Cut off value of 130 mg/dL has 90% detection rate for GDM whereas cut off value of 140 mg/dL has 80% detection rate. GCT has test sensitivity of 79% and specificity of 87%.

American College of Obstetricians and Gynaecologists (ACOG) and ADA state the usage of either threshold. This test needs confirmation by a diagnostic and confirmatory oral glucose tolerance test and forms a part of two step technique for GDM screening.[62,63]

Oral glucose tolerance test with 75/100 gm glucose (one step technique)

This test is both screening and diagnostic test and forms an effective part of one step procedure to screen for GDM. This approach may be cost-effective in high risk populations. It should be done in the morning after an overnight fast of more than 8 hours and after at least 3 days of unrestricted diet, consuming more than or equal to 150 gm of carbohydrate per day. Patients should not smoke before the test and should remain seated during the test. A fasting blood glucose sample is drawn. The pregnant woman is given 75/100 gm of glucose in juice and the samples drawn at 1, 2 and 3 hours respectively.[64]

Diagnosis of GDM is made if two or more values are abnormal on 75/100 g oral glucose tolerance test during pregnancy. All values mentioned in Table 1 indicate plasma blood sugar levels except O'Sullivan and Mahan which mentions venous whole blood. Measurement of blood glucose level in capillary blood by glucometer has made screening test easy and simple as it can be done in office setting and does not require elaborate laboratory facilities. It is important to know that capillary blood glucose levels are comparable to venous blood glucose levels during fasting state but are higher after meals.[65]

In the 4th International workshop conference on GDM in 1997 a decision was made on replacing NDDG criteria by Carpenter and Couston (C&C) criteria which has lower threshold values for the diagnosis of GDM so as to diagnose more cases of GDM.[34] This one stage procedure is preferred over one step approach as there are less follow-up losses, earlier detection and treatment.[66]

Glycosylated haemoglobin (A1c) and serum fructosamine

These tests are time consuming, and are expensive with low sensitivity. International expert committee and ADA now recommends the estimation of HbA1C (>6.5%) in the diagnosis of diabetes mellitus in general population but for the screening of GDM, studies are underway. Serum fructosamine levels indicate glycemic control over a shorter period, but are not indicated for diagnosis of GDM.[61,67]

Glycosuria

This test is affected by numerous physiological factors and has only 30% sensitivity.[68]

Comparison of methods of screening[69]

Of the various screening tests, OGTT is the most acceptable screening as well as diagnostic test. A number of screening procedures and diagnostic criteria (ADA, WHO, Canadian Diabetes Association [CDA], NDDG and Australasian criteria) are being followed in the same as well as in different countries. American Diabetes association recommends screening for selective (high risk) population. But compared to selective screening, universal screening for GDM detects more cases and improves maternal and neonatal prognosis. Hence universal screening for GDM is essential, as it is generally accepted that women of Asian origin and especially ethnic Indians, are at a higher risk of developing GDM and subsequent type 2 diabetes.

ADA procedure

ADA recommends selective screening with two step procedures.

Step 1: A 50 gm glucose challenge test (GCT) is used for screening without regard to the time of last meal or time of the day.

Step 2: If one hour GCT value is more than 140 mg/dL, 100 gm oral glucose tolerance test is recommended and plasma glucose is estimated at 0, 1, 2 and 3 hours. Gestational Diabetes Mellitus is diagnosed if any two values meet or exceed FPG >

95 mg/dL, one hr PG > 180 mg/dL, two hr PG > 155 mg/dL and three hr PG > 140 mg/dL. But major drawback of this criterion is that, the glycaemic cut off was validated against the future risk of these women developing diabetes and not on the fetal outcome. And method is cumbersome as it involves screening and then diagnostic test.

World Health Organization procedure

World Health Organization recommends universal screening with a two hour 75 gm OGTT with a threshold plasma glucose concentration of greater than or equal to 140 mg/dL at two hours similar to that of IGT, outside pregnancy. Carpenter himself now recommends a two hour 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 gm, two 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 75gm, two hour OGTT is in use during pregnancy in many countries , typically using the same thresholds as in non-pregnant individuals". Shortcoming with this method is that, the criteria suggested for diagnosis of GDM was also not based on maternal and fetal outcome but probably the criteria was recommended for its easy adaptability in clinical practice.

International Association of Diabetes and Pregnancy Study Groups (IADPSG)[70] based on the Hyperglycemia and Adverse Pregnancy Outcome (HAPO)

study[18] outcome recommends any one or more values of fasting plasma glucose (FPG) ≥ 92 mg/dL, 1 hr plasma glucose (PG) ≥ 180 mg/dL and two hr PG ≥ 153 mg/dL for the diagnosis of GDM. The IADPSG recommendation would result in variation in the prevalence of GDM from one centre to another depending on the choice of cut-off value used, either fasting one hr, two hr, or any two values for diagnosis. This flexibility will compromise the uniformity and likely to pose difficulty in comparing outcome data.

The HAPO study[7] was performed in response to the need for internationally agreed upon diagnostic criteria for gestational diabetes, based upon their predictive value for adverse pregnancy outcome. Increase in each of the 3 values on the 75 gm, 2-hour oral glucose tolerance test are associated with graded increase in the likelihood of pregnancy outcomes such as large for gestational age, cesarean section, fetal insulin levels, and neonatal fat content. Based upon this, the International Association of Diabetes and Pregnancy Study Groups recommends that the diagnosis of gestational diabetes be made when any of the following 75 gm, 2-hour oral glucose tolerance test thresholds are met or exceeded: fasting 92 mg/dL, 1-hour 180 mg/dL, or 2 hours 153 mg/dL. Various authoritative bodies around the world are expected to deliberate the adoption of these criteria.

ADA and WHO criteria for the diagnosis of GDM[70]

	ADA 100 gm OGTT	**ADA 75 gm OGTT**	**WHO 75 gm OGTT**
Fasting (mg/dL)	95	95	126
1 Hr (mg/dL)	180	180	-
2 Hr (mg/dL)	155	155	140
3 Hr (mg/dL)	140	-	-

For the ADA criteria, two or more of the values form either the 100 or 75 gm OGTT must be met or exceeded to make the diagnosis of GDM. For the WHO criteria, one of the two values from the 75 gm OGTT must be met or exceeded to make the diagnosis of GDM.

Timing of screening for GDM

Insulin is detectable in fetal pancreas as early as 9 weeks after conception.[40] An increase in beta cell mass and insulin secretion in the fetus occurs by the 16th week of gestation, in response to maternal hyperglycemia.[71,72]

The priming of the fetal beta cells may account for the persistence of fetal hyperinsulinaemia throughout pregnancy and risk of accelerated fetal growth,[73] even when the mother has good metabolic control in later pregnancy.[74]

This indicates the need for performing the test procedures to diagnose GDM in the first trimester itself. Further, early detection and care results in a better fetal outcome.[75]

By following this usual recommendation for screening between 24 and 28 weeks of gestation, the chance of detecting unrecognised type 2 diabetes before

pregnancy (pre GDM) is likely to be missed. If the two hour PG> 200 mg/dL in the early weeks of pregnancy, she may be a pre- GDM and HbA1c > 6 is confirmatory (normal A1c levels during pregnancy is 5.3-6).

A pregnant woman found to have normal glucose tolerance (NGT) in the first trimester, should be tested for GDM around 24^{th}- 28^{th} weeks and again around 32^{nd}-34^{th} weeks and also later weeks if necessary, particularly when rapid maternal weight gain occurs or fetal macrosomia is suspected.

Diagnostic criteria for the 100-g 3-hour tolerance test for gestational diabetes mellitus

Status	Plasma or serum glucose level Carpenter and Coustan conversion		Plasma level National Diabetes Data Group Conversion	
	mg/dL	mmol/L	mg/dL	mmol/L
Fasting	95	5.3	105	5.8
1 hour	180	10.0	190	10.6
2 hours	155	8.6	165	9.2
3 hours	140	7.8	145	8.0

Guidelines on GDM

Organisation	Year	Policy	Screening test	Diagnostic test
SOGC[76]	1992	Universal	50 g GCT	100 g OGTT
4th International Workshop-Conference on GDM[77]	1997	Selective	Optional: 2-step 50/100 g GCT/OGTT (threshold 7.2 mmol/L or 7.8 mmol/L) or 1 step 75 g GCT	75 or 100 g OGTT (Carpenter-Coustan conversion)
CDA[78]	1998	Selective	50 g GCT	75 or 100 g OGTT (Carpenter-Coustan conversion)
ADA[79]	1998	Selective	50 g GCT (threshold 7.2 mmol/L)	100 g OGTT (NDG conversion)
ACOG[80]	2001	Universal of selective	50 g GCT (threshold 7.2 mmol/L or 7.8 mmol/L)	100 g OGTT (Either Carpenter-Coustan or NDDG conversion)

Literature review

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."[5]

The 2001 practice guidelines of the American College of Obstetricians and Gynecologists (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 the following criteria: (1) younger than 25 years; (2) not a member of an ethnic group at high risk for development of type 2 diabetes mellitus; (3) BMI of 25 kg/m^2 or less; (4) no history of previous glucose intolerance or adverse pregnancy outcomes associated with GDM; and (5) no first degree relative with known diabetes.[5]

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 testing for GDM in all pregnant women who do not have a diagnosis of pregestational diabetes.[5]

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 receive a subsequent diagnostic test.[81] Typically, a 50 g oral glucose challenge test (OGCT) is initially administered between 24 and 28 weeks' gestation in a nonfasting 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 3 hours. 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.[82]

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. 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.[5]

In 2004, a cross-sectional study reported that universal screening was the most common practice in the United States, with 96% of obstetricians routinely screening for GDM.[83] 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.[84] Since only 10% of pregnant women were categorized as low risk, some argued that selective screening contributed to confusion, with little benefit and potential for harm.[85] Of particular concern was the association between risk factor-based screening and high rates of false negative results.[86] Others have endorsed alternative risk scoring systems for screening.[87]

The IADPSG, an international consensus group with representation from multiple obstetrical and diabetes organizations, recently spearheaded a

reexamination of the definition of GDM in an attempt to bring uniformity to GDM diagnosis. The IADPSG recommended that a one-step 75 g OGTT be given to all pregnant women who do not have a diagnosis of overt diabetes. 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. The diagnostic glucose thresholds recommended by the IADPSG were the maternal glucose values from the HAPO study[7] that identified a 1.75-fold increase in large for gestational age, elevated C-peptide, high neonatal body fat, or in a combination of these factors. 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 pregnancy outcomes, 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 the following criteria: fasting plasma glucose ≥126 mg/dL (7.0 mmol/L), glycated hemoglobin (HbA1c) ≥6.5% or a random plasma glucose ≥200 mg/dL (11.1 mmol/L) confirmed by one of the first two measures.[5]

The Hyperglycemia and Adverse Pregnancy Outcome (HAPO) Study[88] in the year 2009 to clarify whether maternal hyperglycemia less severe than that in diabetes mellitus is associated with increased risks of adverse pregnancy outcomes indicated a continuous association of maternal glucose levels below those diagnostic of diabetes with an adverse outcome, with the strongest risk for increased birth weight and cord blood serum C peptide levels indicating fetal hyperinsulinism.

Additionally an increased risk for maternal complications like preeclampsia was seen.

The current diagnostic criteria for gestational diabetes mellitus are controversial because they lack correlation to maternal and perinatal outcome. The results of the hyperglycemia and adverse pregnancy outcome (HAPO) study[89] demonstrate a linear association between increasing levels of fasting, 1- and 2-h plasma glucose after a 75 g oral glucose tolerance test to several significant outcomes, such as birth weight above the 90th percentile, cord blood serum C-peptide level above the 90th percentile, primary cesarean delivery, clinical neonatal hypoglycemia, premature delivery, shoulder dystocia or birth injury, intensive neonatal care admission, hyperbilirubinemia, and preeclampsia. A consensus report by the IADPSG, based on a vigorous assessment of the HAPO results and other studies, recommended an endorsement of risk-based, internationally accepted criteria for the diagnosis and classification of diabetes in pregnancy.

Recently, Hyperglycemia and Adverse Pregnancy Outcome (HAPO) Study[90] using the new International Association of the Diabetes and Pregnancy Study Groups (IADPSG) criteria[10,22] reported overall frequency of GDM as 17.8% (range 9.3-25.5%). There was substantial center-to-center variation in which glucose measures met diagnostic thresholds. Study concluded that, although the new diagnostic criteria for GDM apply globally, center-to-center differences occur in GDM frequency and relative diagnostic importance of fasting, 1-h, and 2-h glucose levels.

A study[91] to find out whether DIPSI guidelines could still be continued to diagnose GDM in India, enrolled consecutive pregnant women (n=1463) who underwent 75g oral glucose tolerance test (OGTT). The proportion of GDM was computed based on IADPSG and DIPSI criteria and the discordant pair of diagnosing GDM was examined. The prevalence of GDM was 14.6% (N=214) by IADPSG criteria and 13.4% (n=196) by DIPSI criteria. The discordant pair between the two criteria examined by Mc Nemar's test indicated that there was no statistical significance (p=0.210) and thereby implying a close agreement between these two procedures. Study concluded that, DIPSI procedure is cost-effective, without compromising the clinical equipoise and can be continued to diagnose GDM in our country, as well as other less resource countries.

Another study[92] to evaluate characteristics and pregnancy outcomes in women prior classified normal by Carpenter and Coustan criteria (old criteria) and now gestational diabetes (GDM) by the IADPSG criteria studied 6727 pregnancies retrospectively. Using the old criteria, 222 had GDM (old GDM). Using the IADPSG criteria, 382 had GDM of which 160 had a normal glucose tolerance with the old criteria (new GDM). Authors concluded that, using the IADPSG criteria, more women are identified as having GDM, and these women carry an increased risk for adverse gestational outcome compared to women without GDM.

Recently a study[93] from China evaluated the IADPSG criteria versus the ADA criteria for diagnosing GDM in China. Overall, 3083 women with a singleton pregnancy underwent a 75-g, 2-h oral glucose tolerance test between 24 and 28 weeks of pregnancy, and both IADPSG and ADA criteria were used for GDM diagnosis. IADPSG and ADA criteria diagnosed 19.9% and 7.98% of women with

GDM, respectively (P<0.001). IADPSG criteria has a stronger capacity of predicting APOs than ADA criteria (odds ratio (OR)=1.84, 95% confidence interval (CI): 1.52-2.25 for IADPSG, and OR=1.54, 95% CI: 1.16-2.05 for ADA). Study showed that, IADPSG criteria increase GDM diagnosis by almost twofold and GDM diagnosed by IADPSG criteria is more associated with adverse pregnancy outcome, although the economic impact needs further evaluation.

METHODOLOGY

This study was conducted in the Department of Obstetrics and Gynecology, KLE'S Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum.

Study design

The study design was a cross sectional study.

Study duration and period

This one year study was conducted from January 2013 to December 2013.

Place

The present study was done at Department of Obstetrics and Gynaecology, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum a teaching hospital attached to Jawaharlal Nehru Medical College, Belgaum.

Source of data

Pregnant women between 24 to 28 weeks of gestation registered at Antenatal Clinic, Department of Obstetrics and Gynaecology, KLES Dr Prabhakar Kore Hospital and Medical Reasearch Center, Belgaum were studied.

Sample size

A total of 225 pregnant women from 24 to 28 weeks of gestation were included in the study.

Sampling procedure

The sample size was calculated considering the prevalence based on the formula as below.

$n = 4 p q / d^2$

Where,

n = Sample size

p = Prevalence that is, 17%

q = 100 – p = 83%

d = Absolute error = 5% with drop out rate of 20%

Therefore,

$n = 4 \times 17 \times 83 / 5^2$

n = 225

Hence the sample size of 225 was planned.

Selection criteria

Inclusion

- Pregnant women from 24 to 28 weeks of gestation.

Exclusion

- Known diabetic patients.
- Women not consenting to participate in the study.

Ethical clearance

Prior to the commencement, ethical clearance was obtained from the Institutional Ethical committee, Jawaharlal Nehru Medical College, Belgaum.

Informed Consent

The eligible women were explained about the nature of the study and a written informed consent was obtained (Annexure I).

Method of collection of data

After the enrollment demographic data, obstetric history and current pregnancy details were obtained. Further these women were subjected to clinical examination. The body mass index was calculated using the formula as below. \

Body mass index = Weight (kg)/ Height (m^2)

Body mass index in the range of less than 19.8 kg/m^2 was considered as underweight, 19.8 to 26 kg/m^2 as normal, 26 to 29 kg/m^2 as overweight and more than 29 kg/m^2 as obese. These findings were recorded on a predesigned and proforma (Annexure II).

Procedure

Under aseptic precautions, 2 mL of venous blood sample was drawn in the fasting state. Further these women were given 75g oral glucose and their one hour and two hours venous blood samples were drawn. The plasma glucose was estimated in the hospital laboratory by glucose oxidase peroxidase (GOD-POD) method.

Diagnosis of GDM

Diagnosis and the prevalence of GDM were assessed by applying both DIPSI[21] and IADPSG criteria.[10,22]

IADPSG Criteria

Based on IADPSG criteria[10,22] GDM was diagnosed if one or more values equals or exceeds thresholds of;

- Fasting plasma glucose of 5.1mmol/L (92mg/dL).
- One hour plasma glucose level of 10.0mmol/dL (180mg/dL).
- Two hour plasma glucose level of 8.5mmol/L(153mg/dL).

DIPSI Criteria

Using DIPSI criteria[21] GDM was diagnosed if after 75 grams oral glucose two hour plasma glucose value exceeds above 140 mg/dL.

Statistical analysis

The data obtained was coded and entered into Microsoft Excel Worksheet (Annexure III). The categorical data was expressed as rates, ratios and proportions and continuous data was expressed as mean ± standard deviation (SD). Difference in diagnostic capability between IADPSG and DIPSI was expressed in terms of percentage and Kappa statistics was used to evaluate the agreement. A probability value ('p' value) of less than or equal to 0.05 was considered as statistically significant.

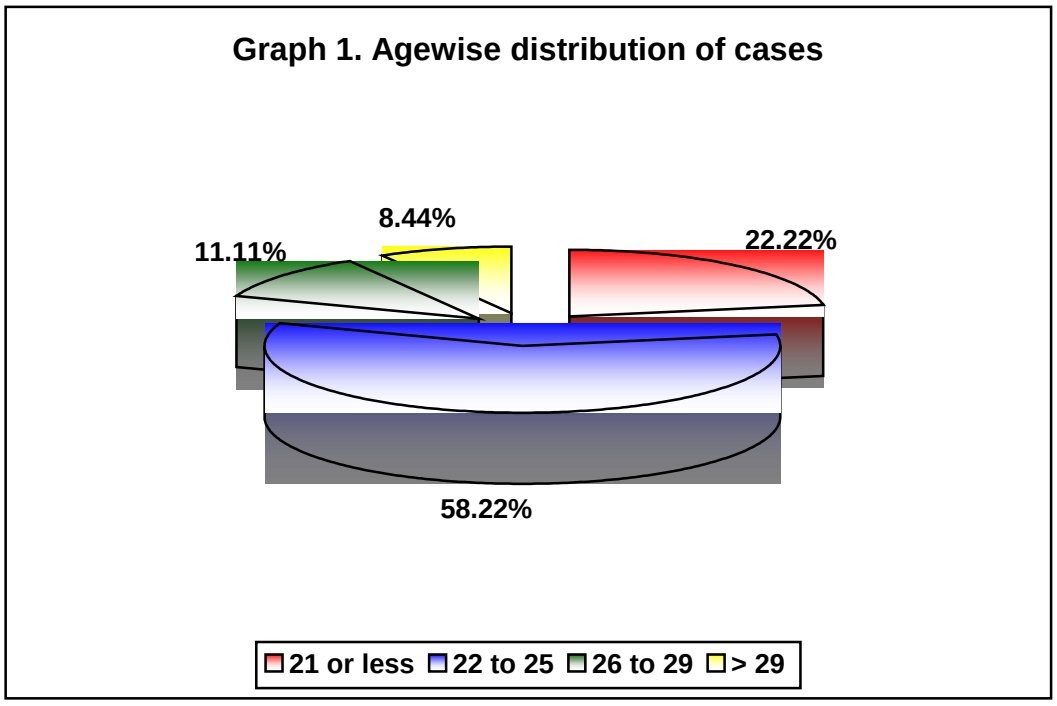
RESULTS

This cross sectional study was conducted from January 2013 to December 2013. A total of 225 pregnant women from 24 to 28 weeks of gestation registered at Antenatal Clinic, Department of Obstetrics and Gynaecology, KLES Dr Prabhakar Kore Hospital and Medical Reasearch Center, Belgaum were included.

The data obtained was coded and entered into Microsoft excel spreadsheet (Annexure III). The data was analysed and the final results and observations were tabulated as below.

Table 1. Agewise distribution of cases

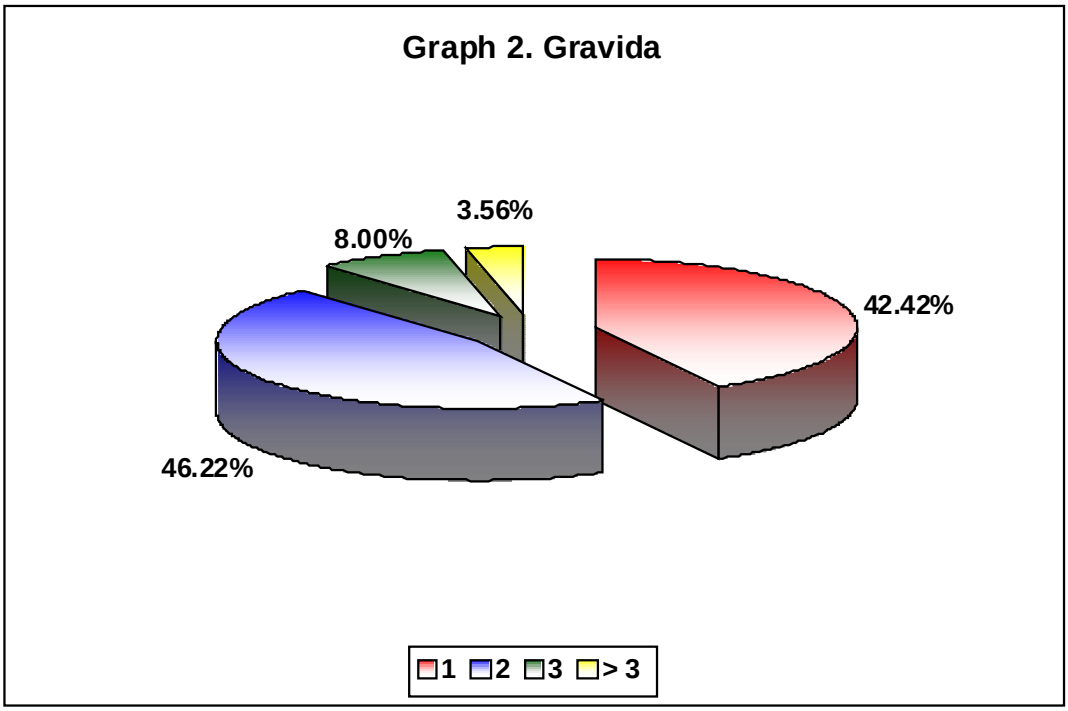
Age group	Distribution (n=225)	
	Frequency	Percent
21 or less	50	22.22
22 to 25	131	58.22
26 to 29	25	11.11
> 29	19	8.44
Total	**225**	**100.00**



In the present study most of the women (58.22%) presented with age between 22 to 25 years followed by less than 21 years (22.22%) The mean age was found to be 23.78 ± 3.38 years.

Table 2. Gravida

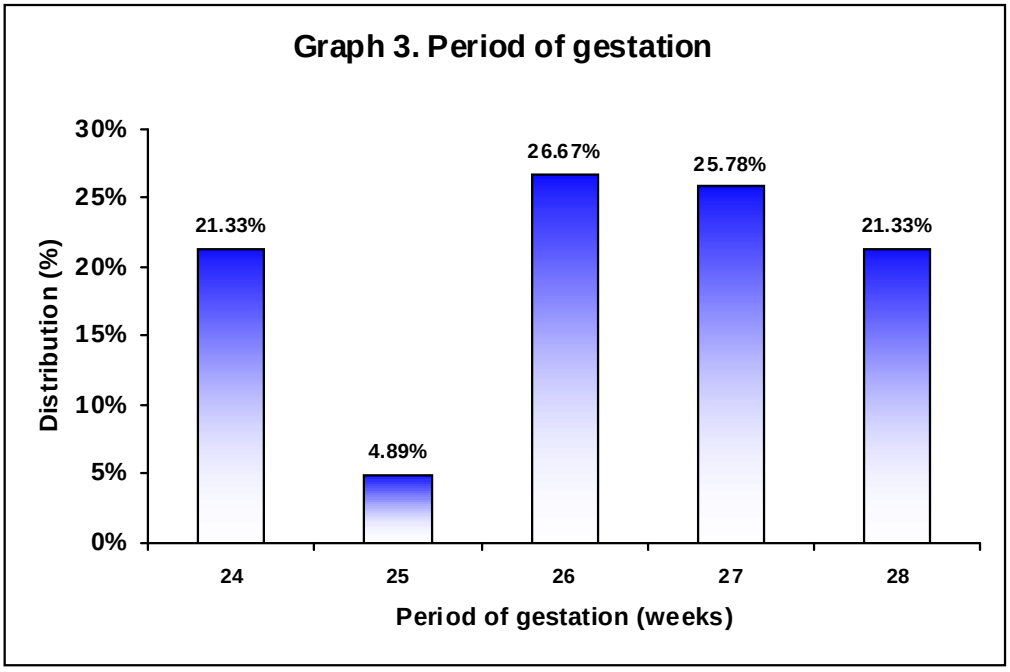
Gravida	Distribution (n=225)	
	Frequency	**Percent**
1	95	42.22
2	104	46.22
3	18	8.00
> 3	8	3.56
Total	**225**	**100.00**



In this study 46.22% of the women who reported were second gravida while 42.42% were primi gravidas.

Table 3. Period of gestation

Period of gestation (weeks)	Distribution (n=225)	
	Frequency	Percent
24	48	21.33
25	11	4.89
26	60	26.67
27	58	25.78
28	48	21.33
Total	**225**	**100.00**

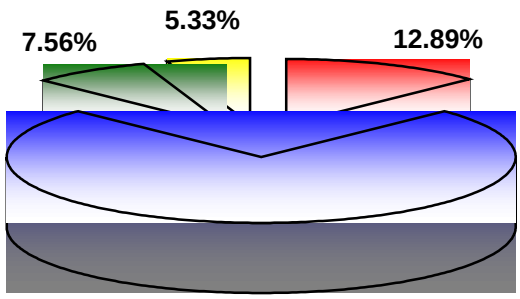


In the present study 26.67%, 25.78% and 21.33% women presented with gestational age of 26, 27 and 28 weeks respectively. The mean gestational age was noted as 26.25 ± 2.70 weeks.

Table 4. Body mass index

Body mass index (Kg/m2)	Distribution (n=225)	
	Frequency	Percent
Less than 19.8	29	12.89
19.8 to 25.99	167	74.22
26.00 to 28.99	17	7.56
29.00 or more	12	5.33
Total	**225**	**100.00**

Graph 4. Body mass index

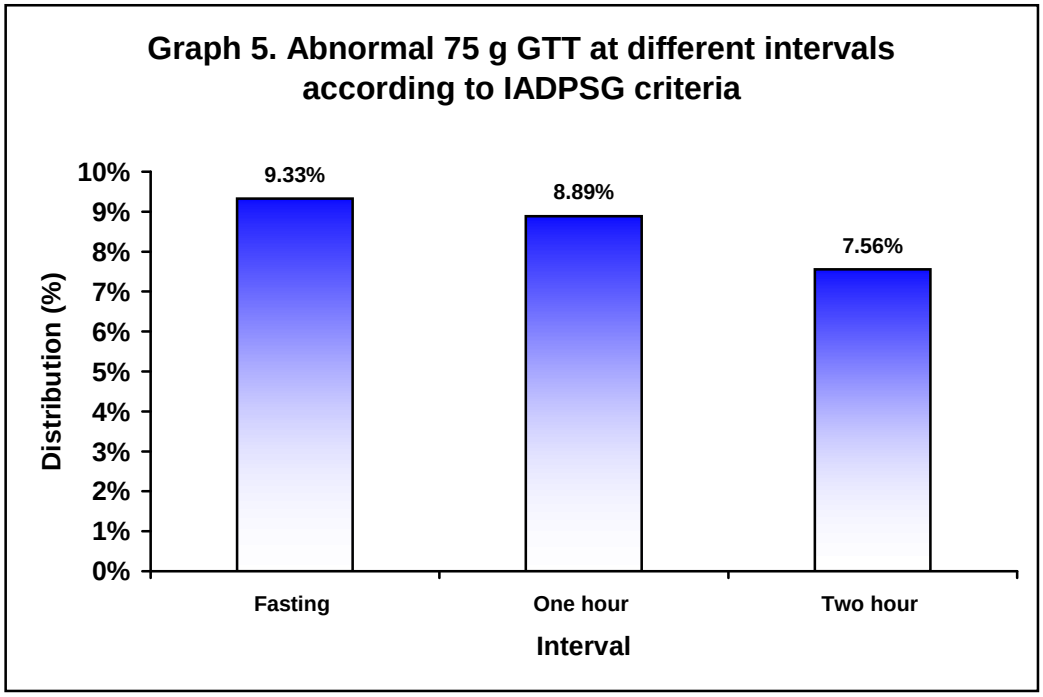


< 19.8 19.8 to 25.99 26.00 to 28.99 29 or more

In this study majority of the pregnant women (74.22%) had body mass index between 19.8 to 25.99 Kg/m^2. The mean body mass index was calculated to be 22.83 ± 3.75 kg/m^2.

Table 5. Abnormal 75 g GTT at different intervals according to IADPSG criteria

GDM (mg/dL)	Distribution (n=225)	
	Frequency	**Percent**
Fasting (≥92)	21	9.33
One hour (≥180)	20	8.89
Two hours (≥153)	17	7.56



In the present study 9.33% of the women had fasting plasma glucose levels of ≥ 92 mg/dL. At one hour and two hours the plasma glucose levels of ≥180 and ≥153 mg/dL were noted in 8.99% and 7.56% respectively.

Table 6. Mean plasma glucose levels at different intervals

Interval	Mean values (n=225) (mg/dL)	
	Mean	**SD**
Fasting	80.35	17.37
One hour	122.9	31.96
Two hours	107.76	29.51

In the present study fasting plasma glucose levels were 80.35 ± 17.37 mg/dL. At one hour and two hours interval the same were found to be 122.90 ± 31.96 mg/dL and 107.76 ± 29.51 mg/dL respectively.

Table 7. Abnormal 75 g GTT at different intervals

Intervals	Distribution (n=225)	
	Frequency	**Percent**
Fasting	13	5.78
1 hour	11	4.89
2 hour	6	2.67
Fasting + 1 hour	2	0.89
Fasting + 2 hours	4	1.78
1 hour + 2 hour	5	2.22
Fasting + 1 hour + 2 hours	2	0.89
Normal	182	80.89
Total	**225**	**100.00**

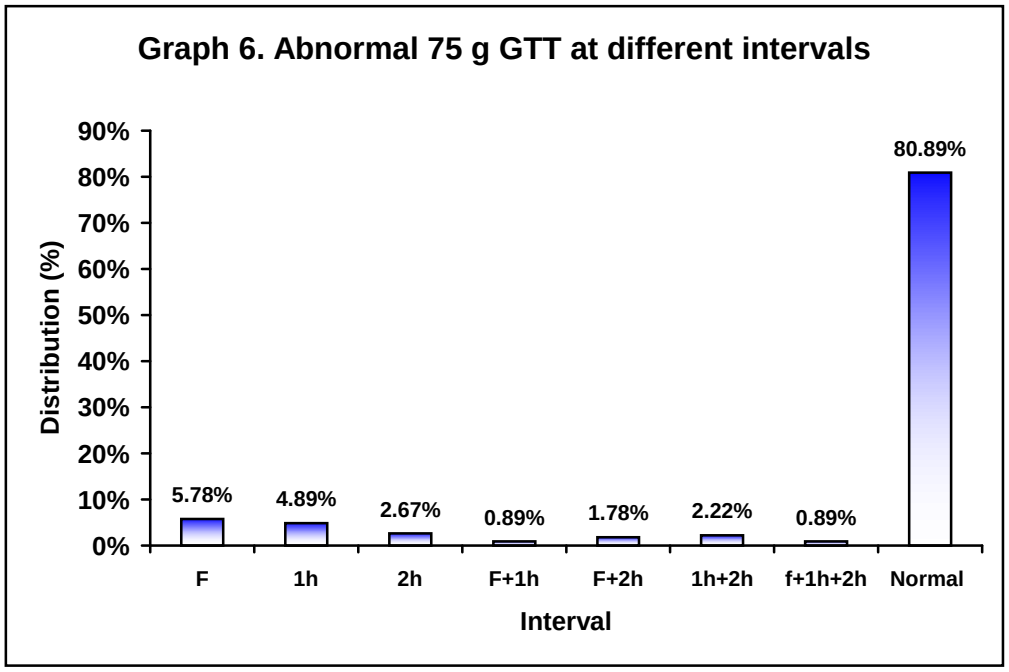
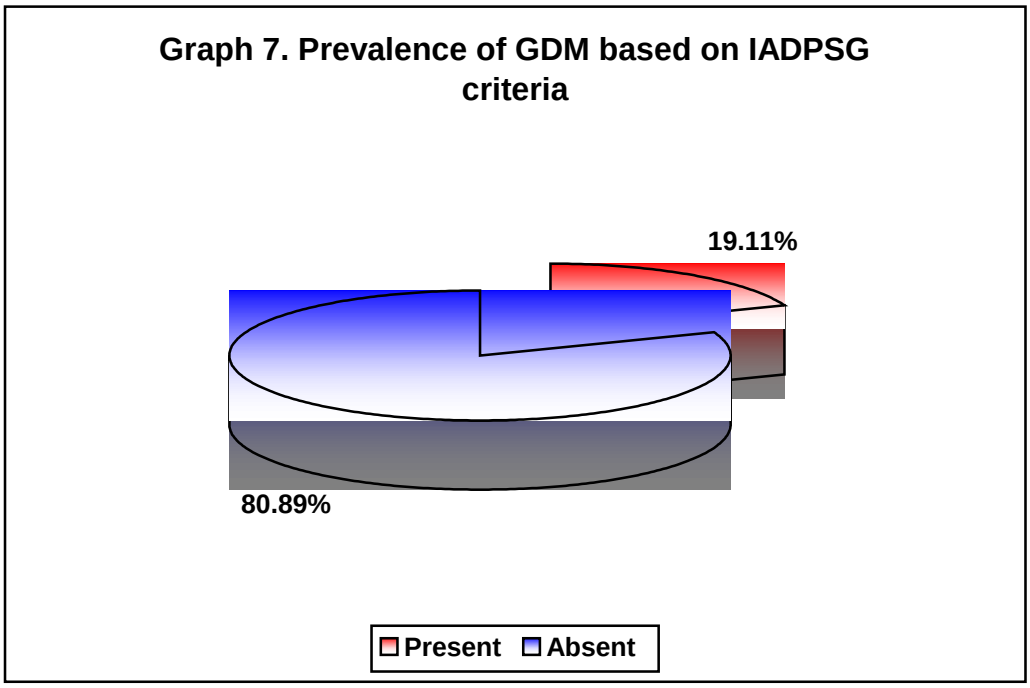


Table 7 and graph 6 shows abnormal 75g GTT at different intervals.

Table 8. Prevalence of GDM based on IADPSG criteria

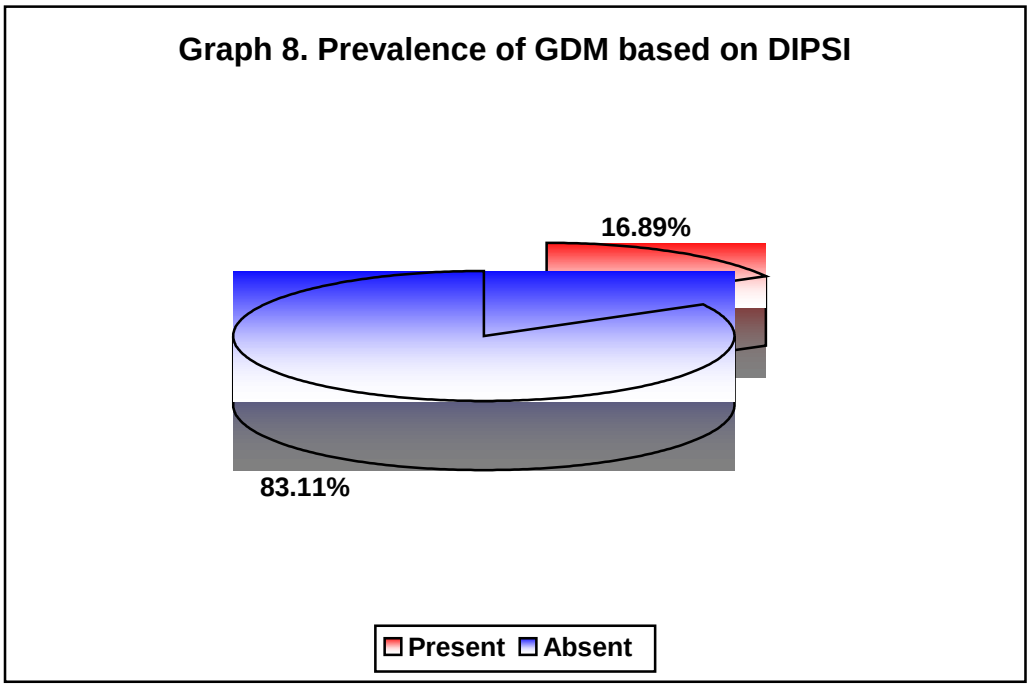
GDM	Distribution (n=225)	
	Frequency	**Percent**
Present	43	19.11
Absent	182	80.89
Total	**225**	**100.00**



In the present study the prevalence of GDM based on IADPSG criteria was 19.11%.

Table 9. Prevalence of GDM based on DIPSI

GDM	Distribution (n=225)	
	Frequency	**Percent**
Present	38	16.89
Absent	187	83.11
Total	**225**	**100.00**



In this study the prevalence of GDM based on DIPSI criteria was 16.89%.

Table 10. Difference in diagnostic capability between IADPSG and DIPSI

GDM based on DIPSI	GDM based on IADPSG				Total (n=120)	
	Present		Absent			
	No.	%	No.	%	No.	%
Present	33	86.84	5	13.16	38	16.89
Absent	10	5.35	177	94.65	187	83.11
Total	**43**	**19.11**	**182**	**80.89**	**225**	**100.00**

p=0.302; Kappa= 0.774; SE of kappa = 0.056; 95% CI: From 0.665 to 0.883

In the present study the prevalence of GDM based on IADPSG criteria was 19.11% and the same was 16.89% based on DIPSI criteria but the difference was statistically not significant. The kappa statistics showed good strength of agreement between the tests.

DISCUSSION

Gestational diabetes mellitus, a common medical complication of pregnancy, is defined as “any degree of glucose intolerance with onset or first recognition during pregnancy”. There is consensus that overt diabetes during pregnancy, whether symptomatic or not, is associated with significant risk of adverse perinatal outcome. The risk of adverse perinatal outcome associated with degrees of hyperglycemia less severe than overt diabetes is controversial. Several factors contribute to this longstanding controversy.[10]

Some have attributed risks of adverse outcomes associated with GDM, such as birth weight that is large for gestational age (LGA), excess fetal adiposity, and higher rate of cesarean section, to confounding characteristics, such as obesity, more advanced maternal age, or other medical complications, rather than glucose intolerance. Bias of caregivers toward expectation of adverse outcomes may increase morbidity due to increased intervention. Some suggest that criteria currently in wide use for the diagnosis of GDM are too restrictive and that lesser degrees of hyperglycemia increase risk of adverse perinatal outcomes. Conversely, others believe that systematic efforts to identify GDM should be stopped unless data become available to link significant morbidities to specific degrees of glucose intolerance.[10]

However, lack of international uniformity in the approach to ascertainment and diagnosis of GDM has been a major hurdle. The initial criteria for the diagnosis of GDM were established more than 40 years ago and, with modifications, remain in use today. These criteria were chosen to identify women at high risk for

development of diabetes after pregnancy or were derived from criteria used for nonpregnant individuals and not necessarily to identify pregnancies with increased risk for adverse perinatal outcome.[10]

This implies that Universal screening and care of women with GDM is of paramount public health priority4 in high risk population for GDM and diabetes like Asian Indians, rather than risk factor screening. In this aspect, except the existing diagnostic criterion of World Health Organization (WHO) 2-h plasma glucose (PG) ≥ 140 mg/dL with 75g oral glucose load, other diagnostic criteria are country specific or recommended by various associations.[91]

Recently, based on the Hyperglycemia and Adverse Pregnancy Outcome (HAPO) study,[7] the International Association of Diabetes and Pregnancy Study Groups (IADPSG) consensus panel recommended that GDM can be diagnosed, if any one value of fasting plasma glucose (FPG), 1-h and 2-h PG concentrations meet or exceed 92 mg/dL, 180 mg/dL and 153 mg/dL respectively, with 75g oral glucose tolerance test (OGTT).[10,22] However, India was not included in the HAPO study[7] despite being one of the most populous countries in the world and with higher prevalence of GDM. The prevalence of GDM in India varies from 3.8 to 21.0% in different parts of the country, depending on the geographical locations and diagnostic methods used.[11-14] In India the diagnosis of GDM is made by the guidelines recommended by Diabetes In Pregnancy Study Group India (DIPSI)[21] based on WHO criterion[47] of 2-h PG ≥ 140 mg/dL. The present study was undertaken to find the concordance between the present practice of diagnosing GDM by DIPSI[21] criterion of 2-h PG ≥ 140 mg/dL and IADPSG recommendation[10,22] in our setting.

The present cross sectional study was done from January 2013 to December 2013 on a total of 225 pregnant women registered at Antenatal Clinic, Department of Obstetrics and Gynaecology, KLES Dr Prabhakar Kore Hospital and Medical Research Center, Belgaum.

In the present study the prevalence of GDM was 19.11% based on IADPSG criteria and 16.89% according to DIPSI criteria. There is wide variation in the prevalence of GDM in India.[94] Earlier studies reported prevalence of 2% in 1982[95] which rose to 7.62% in 1991.[96] During 2002-2003, a random survey performed in various cities in India revealed prevalence of GDM as 16.2% in Chennai, 15% in Thiruvananthapuram, 21% in Alwaye, 12% in Bangalore, 18.8% in Erode and 17.5% in Ludhiana.[97] An overall GDM prevalence of 16.55 per cent was observed. Another study done in Tamil Nadu during 2005-2007, GDM was detected in 17.8%, 13.8% and 9.9% of the women in urban, semi-urban and rural areas, respectively.[12] The findings of the present study were consistent with the studies done in south India during the last decade.[12] However the wide variation in the prevalence rates of GDM may be attributed to the use of different criteria for diagnosis, variation in geographical region and life style with lack of physical activity.

In the present study, fasting, one hour and two hours plasma glucose levels were ≥ 92, ≥180 and ≥153 mg/dL in 9.33%, 8.99% and 7.56% of the women respectively and at same intervals the mean fasting plasma glucose levels 80.35 ± 17.37, 122.90 ± 31.96 mg/dL and 107.76 ± 29.51 mg/dL respectively. Based on the IADPSG criteria,[10,22] the prevalence of GDM 19.11% and by applying DIPSI criteria[21] that is ≥140 mg/dL plasma glucose levels at two hours, prevalence of GDM was 16.89%. The difference in diagnostic capability between IADPSG[10,22]

and DIPSI[21] was found to be 2.8%. However the concordance observed was statistically not significant and the kappa statistics showed good strength of agreement between the two tests (p>0.302; p=0.302; Kappa= 0.774; SE of kappa = 0.056; 95% CI: From 0.665 to 0.883). These findings suggest that, to diagnose GDM, DIPSI procedure[21] based on WHO criterion of 2- h PG ≥ 140 mg/dL is as effective as IADPSG criteria.[10,22] Recently a similar study done in Chennai also found prevalence of GDM, as 13.4% and 14.6% using DIPSI[21] and IADPSG criteria[10,22] respectively. The difference in the diagnostic capability between IADPSG[10,22] and DIPSI[21] was 1.2% (P>0.02).

The IADPSG recommendation[10,22] requires estimation of plasma glucose in three blood samples after administrating 75g oral glucose load whereas, DIPSI criterion[21] requires one blood sample drawn at 2-h for estimating the plasma glucose yielding to higher costs. Further DISPI[21] requires little preparation, without requiring the prior interposition of the screening test and hence it could be applied at the community levels. Thus, DIPSI[21] procedure would still serve the purpose of implementing public health program to diagnose GDM in the community.

Surprisingly similar results were found on the comparison of IADPSG[10,22] and the WHO criteria[47] to diagnose GDM. A maiden study[98] compared the IADPSG[10,22] and the WHO criteria[47] to diagnose GDM in Chennai, India. The study reviewed the retrospective data of 1351 pregnant women who underwent screening for GDM at four selected diabetes centers at Chennai (three private and one government). All women underwent an oral glucose tolerance test using 75g glucose load and fasting, 1-h, and 2-h samples were collected. The IADPSG[10,22] and WHO criteria[47] were compared for diagnosis of GDM. A total of 839 women had GDM by

either the IADPSG[10,22] or the WHO criteria,[47] of whom the IADPSG criteria[10,22] identified 699 and the WHO criteria[47] also identified 699 women as having GDM. However, only 599/839 women (66.6%) were identified by both criteria. Thus, 140/839 women (16.7%) were missed by both the IADPSG[10,22] and the WHO criteria.[47] 687/699 (98.2%) of the women with GDM were identified by the WHO criteria.[47] In contrast, each value of IADPSG criteria[10,22] i.e., fasting, 1 h, and 2 h identified only 12.5%, 14%, and 22%, respectively. A single WHO cut-point of 2 h > 140 mg/dl appears to be suitable for large-scale screening for GDM in India and other developing countries.

The WHO first proposed criteria for GDM using a 75 g OGTT in the 1980s.[99,100] In its technical report published in 1994, it defined GDM as DM first recognized during pregnancy, and gestational impaired glucose tolerance (GIGT) as IGT first recognized during pregnancy.[101] In 1998, WHO recommended new criteria.[102] With regard to GDM, pregnant women who met the WHO criteria[47] for DM or IGT were classified as having GDM and, therefore, the term GIGT disappeared. Some studies have been published taking FPG >126 mg/dl as the criteria for GDM.[103] However, the more recent studies have altogether ignored the FPG criteria and have used only the 2-h > 140 mg/dl criteria of the WHO.[104] When the ADA lowered the FPG to 100 mg/dl from the previous 110 mg/dl for diagnosis of impaired fasting glucose in non-pregnant adults, the FPG level of 126 mg/dl in pregnancy started looking too high and most people just chose to ignore the FPG level for the diagnosis of GDM. However, till date, there is no official recommendation from WHO to drop FPG criteria and to follow only the 2-h value of 140 mg/dl.[98]

It appears an anomaly that in the WHO criteria,[47] the fasting cut-off had been set at 126 mg/dl which is diagnostic of diabetes in non-pregnant adults, whereas the 2-h cut-off was set at 140 mg/dl, which is the diagnostic cut-point for IGT in non-pregnant adults. Probably because of this inherent contradiction in the diagnostic criteria, the fasting values in the WHO criteria[47] are not particularly useful to diagnose GDM and this might explain why the WHO 2-h value alone picked up over 98% of all cases diagnosed by both fasting and 2-h WHO criteria[47] in this study. Another point to be noted is that if a pregnant woman has a FPG ≥ 126 mg/dl, it is considered overt diabetes complicating pregnancy, and not as GDM, by the IADPSG criteria.[10,22]

Another issue of concern is whether too many women would get diagnosed as GDM because of the low FPG cut-off in the IADPSG criteria. [10,22] Indeed, of the 88 women who were diagnosed as GDM by virtue of their FPG abnormality alone using IADPSG criteria,[10,22] only 30 (34%) were classified as GDM by the WHO criteria.[47] A similar comparison with those with GDM according to the IADPSG 1-h cut-off value showed that only 47/98 (48%) had GDM by WHO criteria.[47] It is thus possible that by reducing the FPG cut-point to 92 mg/dl, we could be over-diagnosing GDM in normal pregnant women. This could lead to overloading of the health systems in many countries.[98]

Earlier reports have shown that the sensitivity of the 2-h value in the glucose tolerance test (GTT) is much higher than the fasting plasma glucose among non-pregnant Indian adults.[105] Thus, it is reasonable to assume that since the IADPSG has raised the 2-h value in the IADPSG to 153 mg/dl, many cases of GDM could be missed.[98]

The finding of the present study were in agreement with a study done in Chennai[98] despite the methodological differences that is, difference in study design, criteria used to diagnose GDM and sample size.

Overall the present study showed that, DIPSI procedure based on WHO criterion[47] of 2- h PG ≥ 140 mg/dL would be cost-effective without compromising the clinical equipoise. However the limitations of the study were the study did not consider the diagnostic capability in different confounding variable such as maternal age, parity and obesity as it was beyond the scope of this study. However, association between parity and diabetes is strongly linked to obesity and age. Women with higher parity frequently are older and more obese. Obesity is an intermediate outcome in the causal pathway between parity and gestational diabetes mellitus, probably a mediating factor. However, age is a potential confounder in the association between parity and gestational diabetes mellitus.[106] Hence further studies with large sample size considering the age, parity and obesity would enlighten the role of DIPSI in the diagnosis of GDM.

One of the limitations of the study is that with our present data, we cannot conclude whether IADPSG[10,22] or DIPSI criteria[21] is better for Indian pregnant women as we do not have data on the maternal and fetal outcomes which was beyond the scope of this study. In the absence of the outcome data, which however, was beyond the purview of the study, it was not possible to comment on the suitability of diagnosing GDM by either of the two criteria in this population. Nonetheless, this study compared the ease of use of two criteria in the population studied. Future studies should compare the outcomes of the GDM cases diagnosed

by IADPSG[10,22] and WHO criteria[47] as this would provide the final answer as to which criteria is more suitable for Indians.

CONCLUSION

The prevalence of GDM was 19.11% based on IADPSG criteria and 16.89% according to DIPSI criteria. The difference in diagnostic capability between IADPSG and DIPSI was found to be 2.8%. Based on these results it may be concluded that, the diagnosis GDM based on DIPSI is as effective as IADPSG criteria. Further, in resource poor countries like India, DIPSI procedure would be used with an advantage of being less costly and without compromising the clinical equipoise.

SUMMARY

Universal screening and care of women with GDM is of paramount public health priority in high risk population for GDM and diabetes like Asian Indians. This study was aimed to find the concordance between the present practice of diagnosing GDM by DIPSI criterion of 2-h PG ≥ 140 mg/dL and IADPSG recommendation.

This cross sectional study was done at Antenatal Clinic, Department of Obstetrics and Gynaecology, KLES Dr Prabhakar Kore Hospital and Medical Research Center, Belgaum from January 2013 to December 2013. A total of 225 pregnant women studied. Diagnosis and the prevalence of GDM were assessed by applying both DIPSI and IADPSG criteria.

In the present study most of the women (58.11%) were age between 22 to 25 years and the mean age was 23.78 ± 3.38 years. Most of the women reported gravida two (46.22%) The gestational age was 26 weeks in 26.67% of the women and mean gestational age was 26.25 ± 2.70 weeks. Majority of the women (74.22%) had body mass index between 19.8 to 25.99 Kg/m^2 and mean body mass index was 22.83 ± 3.75 kg/m^2. The fasting, one hour and two hours plasma glucose levels were ≥ 92, ≥180 and ≥153 mg/dL in 9.33%, 8.99% and 7.56% of the women respectively and at same intervals the mean fasting plasma glucose levels were 80.35±17.37, 122.90±31.96 and 107.76±29.51 mg/dL respectively.

Based on the IADPSG criteria, the prevalence of was GDM 19.11% and by applying DIPSI criteria that is ≥140 mg/dL plasma glucose levels at two hours,

prevalence of GDM was 16.89%. The difference in diagnostic capability between IADPSG and DIPSI was found to be 2.8% and the kappa statistics showed good strength of agreement between the two tests ($p>0.302$; Kappa= 0.774). Hence it may be concluded that, the diagnosis GDM based on DIPSI is as effective as IADPSG criteria.

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ANNEXURE I – CONSENT FORM

TITLE: PREVALENCE OF GESTATIONAL DIABETES MELLITUS USING IADPSG AND DIPSI CRITERIA – A CROSS SECTIONAL STUDY"

I.D.NO.

The study is conducted by Dr. **** *****Post graduate student in M.S Obstetrics and Gynaecology under guidance of Dr. **** ******, Professor of OBG, J N Medical College, Belgaum.

Respected Sir/Madam, we invite you to participate in our study as, you are eligible for the same. During the study you will be asked some questions in detail regarding your present complaints.

Purpose of the study

The purpose of this study is to determine the prevalence of gestational diabetes mellitus using IADPSG and DIPSI criteriaat KLE'S Dr Prabhakar Kore Hospital and Medical Research Center, Belgaum. All pregnant women between 24-28 wks registered at antenatal clinic dept of OBG, will be requested to participate in this study during the period of one year.

Procedure and treatment

Should you choose to participate, you will be asked to give a detailed history, undergo a physical examination and a glucose tolerance test with 75 grams of oral glucose a fasting, 1hr, 2hr levels will be recorded.

Risks and benefits

You may undergo some amount of discomfort during the process of investigations, which may include slight pain. However all necessary steps and precautions will be taken to ensure your safety. The result of you taking part in this

research would help health care providers towards a better understanding of Gestational diabetes mellitus and the risk factors associated with it, and thus we will be able to provide improved patient care.

Alternatives

If you decide not to participate in this study, you will still be receiving the usual standard care.

Privacy and confidentiality

Your privacy will be respected and all information collected about you during the course of this study will be kept confidential. Your identity will remain undisclosed.

Financial incentives

You shall not be receiving any payment or any financial incentives for participating in this study.

Authorization to publish results

The results of this study may be published for scientific purpose or presented to a scientific group. Your identity, however, will be maintained confidential at all times.

Voluntary participation

Your participation in this study is voluntary. Your decision whether or not to participate will neither affect the care of your current disease, nor your future relations with the doctor or the hospital. In case you need further information regarding your rights as a study participant, you may please contact Dr **** *****, Telephone no.**** ***** contact or Dr **** ******, Telephone no **** *****. In case you need further information regarding your rights as a study participant, you

may please contact Dr. ***** *****, chairman of the ethical committee, J N Medical College, Belgaum on telephone No. ***** ******.

Statement of Consent:

I.D.NO: | | | |

I Mr/Ms/Mrs _____

volunteer and consent to participate in this study. I have read the consent document or it has been read to me in my vernacular language. I accept to participate in the study. All the information regarding this study is provided to me and I have understood the same. I have been given the opportunity to ask questions and obtain appropriate answers.

Participant's name:

Signature or left thumb print of participant:

Witness name:

Signature of witness:

Signature of the investigator:

Date:

If the participants are Minors (under 18), the parents sign the form, rather than the participants.

ANNEXURE II – PROFORMA

TITLE: PREVALENCE OF GESTATIONAL DIABETES MELLITUS USING IADPSG AND DIPSI CRITERIA – A CROSS SECTIONAL STUDY"

Sl no :

Patient number :

Name : Date:

Age:

Address

Informed consent Y N

Inclusion Criteria Y N

Is she an Overt Diabetic ? Y N

Obstetric History

Gravida Para Abortion Living

Menstrual History

LMP: EDD:

POG:

O/E:

Weight: Height: BMI-

Vitals: PR- BP- CVS-

RS-

P/A-

INVESTIGATIONS-

Blood Group:

Haemoglobin:

Urine R/M:

HIV:

HbsAg:

IADPSG CRITERIA

GTT with 75grams glucose:

FASTING:

1hr:

2hr:

DIPSI CRITERIA

GTT with 75grams glucose:

After 2hr:

FINAL IMPRESION

ANNEXURE III – KEY TO MASTER CHART

Cms	-	Centimeters
DIPSI	-	Diabetes in pregnancy study group of India
g	-	Grams
IADPSG	-	International Association of Diabetes in Pregnancy Study Groups
Kg/m^2	-	Kiligrams per square meter
Kgs	-	Kiligrams

ANNEXURE III - MASTER CHART

Serial number	In / Out patient number	Age (Years)	Obstetric history				Period of gestation (Weeks)	Examination			Investigations			
			Gravida	Para	Abortion	Living		Height (Cms)	Weight (Kgs)	Body mass index (Kg/m2)	GTT 75g (IADPSG)			GTT 75g (DIPSI)
											Fasting	One hour	Two hours	
1	2531123	22	1	0	0	0	24.5	154	51	22.7	75	115	95	95
2	2224626	32	2	1	0	1	26.4	154	58	25.8	68	113	103	103
3	2566117	23	2	1	0	1	24.2	156	58	25.8	73	96	85	85
4	2488336	22	2	0	1	o	24	150	62	27.6	91	116	90	90
5	2682409	28	2	1	o	1	26.4	152	54	20.6	63	137	95	95
6	2588532	32	2	1	0	1	24.5	157	56	22.8	83	179	183	183
7	2488677	22	2	1	0	1	24.4	152	57	22.2	88	112	95	95
8	2620797	24	2	0	1	0	27.2	154	57	25.3	79	110	117	117
9	2620253	22	2	1	0	1	27.2	160	78	30.5	88	150	132	132
10	2682409	19	2	1	0	1	26.2	150	53	23.5	63	137	95	95
11	2611654	28	6	2	3	2	26	156	63	21.8	103	203	177	177
12	2688530	22	1	0	0	o	25	153	46	20.4	60	120	85	85
13	25275421	21	1	0	0	0	26.4	152	44	19.5	81	112	111	111
14	25275408	23	2	1	0	1	26	160	51.5	20.1	82	112	86	86
15	2663885	23	2	1	0	0	24.4	162	60	23.4	76	102	100	100
16	2699175	23	1	0	0	0	28	153	57	25.3	90	101	86	86
17	2708975	21	1	o	o	o	27.5	153	54	24	68	81	66	66
18	2699533	23	1	0	o	0	26	154	62.5	27.8	74	133	94	94
19	2539196	21	2	1	0	1	26	164	52	20.3	76	137	94	94
20	2629735	22	1	0	0	0	28	158	46	18.4	64	89	108	108
21	14338352	22	2	2	0	1	24	138	36	18.9	72	130	81	81
22	2685834	25	1	0	0	0	27.4	160	51	20.1	94	108	152	152
23	2547892	26	3	2	0	2	24.4	156	56	24.9	77	130	58	58
24	2708908	24	2	1	0	1	26	150	46.4	20.6	70	89	84	84
25	2718111	23	1	0	0	0	26.6	158	60	25	82	115	113	113
26	2699241	23	2	1	0	0	28	146	57	23.4	84	106	94	94
27	261810	23	1	0	0	0	26	148	39.4	18.4	72	134	111	111
28	2660508	20	1	0	0	0	24.4	147	70	28.3	85	134	107	107
29	2565907	22	2	1	0	1	24	164	58	21.5	79	152	118	118
30	13184351	23	1	0	0	0	28	158	55	22	74	187	175	175
31	2680412	22	1	0	0	0	24	157	50	22.2	79	147	127	127
32	1938446	19	2	1	0	0	26	147	38	16.9	66	126	96	96
33	2739286	20	1	0	0	0	26	158	50	20	78	95	115	115
34	2739524	20	1	0	0	0	28	152	52	22.6	82	105	111	111

ANNEXURE III - MASTER CHART

Serial number	In / Out patient number	Age (Years)	Obstetric history				Period of gestation (Weeks)	Examination			Investigations			
			Gravida	Para	Abortion	Living		Height (Cms)	Weight (Kgs)	Body mass index (Kg/m2)	GTT 75g (IADPSG)			GTT 75g (DIPSI)
											Fasting	One hour	Two hours	
35	26688891	25	2	1	0	1	27.2	154	52	22	71	132	125	125
36	2531123	22	1	0	0	0	27.3	156	56	23	75	115	95	95
37	11441988	25	3	0	0	0	28	148	50	22.8	72	129	88	88
38	2620797	24	2	0	1	0	27.2	155	57.8	24	79	110	117	117
39	2494173	24	1	0	0	0	26	158	50	20.8	72	112	95	95
40	2681865	22	1	0	0	0	28	156	50	20.5	75	98	94	94
41	2566037	20	1	0	0	0	24	158	51	23.04	78	88	91	91
42	2728273	24	2	1	1	0	25.4	158	60	25	97	137	124	124
43	2543134	19	1	0	0	0	27.6	152	45	19.5	75	125	120	120
44	2510660	30	2	1	0	1	26.3	153	46.8	20	75	112	96	96
45	2681863	25	1	0	0	0	27	150	54	22.7	69	142	118	118
46	27213369	24	2	1	0	1	28	156	56	23	90	93	110	110
47	2713075	25	2	1	0	1	24	158	53	22	73	86	91	91
48	1957346	21	2	1	0	1	27.6	160	50.5	20.2	82	76	86	86
49	2542839	30	3	1	1	1	28	155	74	30.8	73	152	109	109
50	2732731	25	2	2	0	2	27.1	141	45.8	22.7	66	155	75	75
51	2533966	26	5	1	3	1	26.2	140	52	21.6	85	142	188	188
52	2708523	22	2	1	0	1	27	150	69	31.3	74	180	117	117
53	2541204	34	2	1	0	1	26	143	45	22.5	72	146	98	98
54	2728196	19	1	0	0	0	26	156	56	23	81	180	153	153
55	2584140	20	1	0	0	0	28	150	47	21.3	75	62	67	67
56	2699549	24	2	1	0	1	26.3	151	47	19.5	65	105	95	95
57	2592958	30	2	1	0	1	24	156	55	24.3	69	113	97	97
58	2766839	24	2	1	0	1	28	152	65	28	82	101	105	105
59	2539163	24	1	0	0	0	28	147	78	21.4	70	139	131	131
60	2682073	23	3	2	0	1	27.2	158	53	22	85	129	78	78
61	2409853	27	2	1	0	1	27	147	69.7	33.1	92	147	132	132
62	2688391	25	2	1	0	1	27.2	155	51	21.2	71	132	125	125
63	2638478	27	1	0	0	0	25	152	49	21.3	75	86	65	65
64	2556780	25	2	0	0	0	27.3	154	48.5	21	64	72	121	121
65	2664282	27	2	1	0	1	25.1	145	40	19	76	106	111	111
66	2699359	24	1	0	0	0	24	151	50	22.2	98	142	145	145
67	2760658	25	1	0	0	0	26.2	156	55	24.3	97	123	158	158
68	2682218	24	2	1	0	1	24	147	51.1	24	77	133	175	175

ANNEXURE III - MASTER CHART

Serial number	In / Out patient number	Age (Years)	Obstetric history				Period of gestation (Weeks)	Examination			Investigations			
			Gravida	Para	Abortion	Living		Height (Cms)	Weight (Kgs)	Body mass index (Kg/m2)	GTT 75g (IADPSG)			GTT 75g (DIPSI)
											Fasting	One hour	Two hours	
69	2602592	22	1	0	0	1	24	156	54	22.5	70	138	114	114
70	2705675	23	2	0	1	0	24	152	42	18.67	59	114	105	105
71	2699175	23	1	0	0	0	28	153	57	24.70	90	101	86	86
72	2718560	21	2	0	1	0	26.5	156	54	22.5	78	140	127	127
73	2547892	26	2	2	0	2	28	151	60	27	77	130	58	58
74	2682745	23	1	0	0	0	26	153	46	20	71	138	112	112
75	23530935	24	1	o	o	o	28	156	52	23.6	86	120	90	90
76	2728545	27	3	3	0	0	28	160	59	23	86	102	134	134
77	2585687	22	1	0	0	0	28	151	50.5	22.4	137	212	147	147
78	2766796	25	1	o	0	0	26	155	50	22.2	62	74	72	72
79	2638716	26	1	0	0	0	26.1	159	68	26.7	70	78	88	88
80	2656242	24	2	1	0	1	28	153	86	21.9	67	89	77	77
81	2646834	24	2	1	0	1	24.3	152	53	23.1	82	98	97	97
82	1578553	24	2	1	0	1	26.4	159	46	18.18	76	95	73	73
83	2593088	24	2	1	0	1	28	166	62	24.2	89	103	79	79
84	2664161	18	1	0	0	0	27.3	155	53	23.6	75	132	109	109
85	2688212	25	5	2	0	2	27.4	153	54	24	82	188	153	153
86	2742644	25	2	1	0	1	26.3	152	48	21.3	68	96	59	59
87	2810991	21	1	0	0	0	26.2	148	46	23.5	81	99	98	98
88	2663901	24	2	1	0	1	27.3	150	52	23.1	66	98	65	65
89	2742121	23	1	0	0	0	28	138	44	23.1	83	98	81	81
90	2810654	32	5	4	0	4	27.3	155	60	26.7	78	124	84	84
91	2647220	20	1	0	0	0	26	158	47	18.9	70	80	72	72
92	2656525	20	1	0	0	0	27.5	154	58	24.5	91	119	116	116
93	2656527	24	2	1	0	1	24.1	155	60	25	77	105	85	85
94	2801169	26	1	0	0	0	26.6	138	46	24.2	83	111	127	127
95	2798016	20	1	0	0	0	25	153	54	24	82	68	97	97
96	2699334	22	3	2	0	2	24.3	158	74.4	29.9	116	111	111	111
97	2080151	20	2	1	0	1	27.3	152	57	25.3	90	103	91	91
98	503093	22	2	0	0	1	28	151	57.4	25.3	127	157	149	149
99	2663901	24	2	1	0	1	27.1	150	49	21.8	66	98	65	65
100	2782044	35	3	2	1	1	28	158	60	26.7	84	188	150	150
101	2712234	23	2	1	1	0	27.6	158	58	21.3	88	182	148	148
102	2699445	29	2	1	0	1	24.2	152	42	18.7	78	202	149	149

ANNEXURE III - MASTER CHART

Serial number	In / Out patient number	Age (Years)	Obstetric history				Period of gestation (Weeks)	Examination			Investigations			
			Gravida	Para	Abortion	Living		Height (Cms)	Weight (Kgs)	Body mass index (Kg/m2)	GTT 75g (IADPSG)			GTT 75g (DIPSI)
											Fasting	One hour	Two hours	
103	2544786	20	1	0	0	0	28	156	56	21.9	68	126	76	76
104	2751954	30	3	0	1	0	27.2	142	68	34.7	86	186	118	118
105	2760601	20	1	0	0	0	27.5	155	57	25.3	81	131	142	142
106	2751974	20	1	0	0	0	24	160	40	15.6	71	156	147	147
107	25568704	21	2	1	0	1	27.5	133	38	22.5	72	130	81	81
108	2556887	25	3	2	0	0	27	163	48	21.3	186	165	148	148
109	2470732	24	1	0	0	0	25	160	49	19.1	160	174	163	163
110	2775901	35	1	0	0	0	26	158	58	21.3	78	1o5	104	104
111	1929200	25	1	0	0	0	26	150	48	21.3	61	129	115	115
112	2488677	22	1	0	0	0	24.6	155	57.7	25.3	55	86	78	78
113	2544879	23	3	1	1	0	27.5	156	51	22.7	73	77	64	64
114	2718723	23	1	0	0	0	24.5	155	57	25.3	100	176	162	162
115	596733	27	2	1	1	0	28	149	65	28.9	70	130	84	84
116	2699125	22	1	0	0	0	27.4	163	47	18.4	88	153	145	145
117	2505561	19	2	0	1	0	27.5	153	52	23.1	80	81	96	96
118	2783429	19	1	0	0	0	28	155	44	19.6	87	75	91	91
119	3106133	22	1	0	0	0	24.5	154	51	22.7	75	115	95	95
120	3138675	32	2	1	0	1	26.4	154	58	25.8	68	113	103	103
121	3013263	23	2	1	0	1	24.2	156	58	25.8	73	96	85	85
122	3097396	22	2	0	1	o	24	150	62	27.6	91	116	90	90
123	2946628	28	2	1	o	1	26.4	152	54	20.6	63	137	95	95
124	2949499	32	2	1	0	1	24.5	157	56	22.8	83	179	183	183
125	3007547	22	2	1	0	1	24.4	152	57	22.2	88	112	95	95
126	307790	24	2	0	1	0	27.2	154	57	25.3	79	110	117	117
127	2896642	22	2	1	0	1	27.2	160	78	30.5	88	150	132	132
128	2216426	19	2	1	0	1	26.2	150	53	23.5	63	137	95	95
129	1487409	28	6	2	3	2	26	156	63	21.8	103	203	177	177
130	1202184	22	1	0	0	o	25	153	46	20.4	60	120	85	85
131	3059338	21	1	0	0	0	26.4	152	44	19.5	81	112	111	111
132	2472711	23	2	1	0	1	26	160	51.5	20.1	82	112	86	86
133	3043820	23	2	1	0	0	24.4	162	60	23.4	76	102	100	100
134	3138477	23	1	0	0	0	28	153	57	25.3	90	101	86	86
135	3138687	21	1	o	o	o	27.5	153	54	24	68	81	66	66
136	3128889	23	1	0	o	0	26	154	62.5	27.8	74	133	94	94

ANNEXURE III - MASTER CHART

Serial number	In / Out patient number	Age (Years)	Obstetric history				Period of gestation (Weeks)	Examination			Investigations			
			Gravida	Para	Abortion	Living		Height (Cms)	Weight (Kgs)	Body mass index (Kg/m2)	GTT 75g (IADPSG)			GTT 75g (DIPSI)
											Fasting	One hour	Two hours	
137	2998450	21	2	1	0	1	26	164	52	20.3	76	137	94	94
138	2848835	22	1	0	0	0	28	158	46	18.4	64	89	108	108
139	1212893	22	2	2	0	1	24	138	36	18.9	72	130	81	81
140	1848316	25	1	0	0	0	27.4	160	51	20.1	94	108	148	148
141	3016529	26	3	2	0	2	24.4	156	56	24.9	77	130	58	58
142	3096709	24	2	1	0	1	26	150	46.4	20.6	70	89	84	84
143	3043660	23	1	0	0	0	26.6	158	60	25	82	115	113	113
144	2995650	23	2	1	0	0	28	146	57	23.4	84	106	94	94
145	3059504	23	1	0	0	0	26	148	39.4	18.4	72	134	111	111
146	3121612	20	1	0	0	0	24.4	147	70	28.3	85	134	107	107
147	3022269	22	2	1	0	1	24	164	58	21.5	79	152	118	118
148	1686339	23	1	0	0	0	28	158	55	22	74	187	175	175
149	3035024	22	1	0	0	0	24	157	50	22.2	79	147	127	127
150	3008213	19	2	1	0	0	26	147	38	16.9	66	126	96	96
151	3043778	20	1	0	0	0	26	158	50	20	78	95	115	115
152	3026138	20	1	0	0	0	28	152	52	22.6	82	105	111	111
153	2973582	25	2	1	0	1	27.2	154	52	22	71	132	125	125
154	2860293	22	1	0	0	0	27.3	156	56	23	75	115	95	95
155	3022851	25	3	0	0	0	28	148	50	22.8	72	129	88	88
156	3105443	24	2	0	1	0	27.2	155	57.8	24	79	110	117	117
157	3016529	24	1	0	0	0	26	158	50	20.8	72	112	95	95
158	3025963	22	1	0	0	0	28	156	50	20.5	75	98	94	94
159	3062646	20	1	0	0	0	24	158	51	23.04	78	88	91	91
160	3072020	24	2	1	1	0	25.4	158	60	25	97	137	124	124
161	3081038	19	1	0	0	0	27.6	152	45	19.5	75	125	120	120
162	3053407	30	2	1	0	1	26.3	153	46.8	20	75	112	96	96
163	3121184	25	1	0	0	0	27	150	54	22.7	69	142	118	118
164	3053528	24	2	1	0	1	28	156	56	23	90	93	110	110
165	2887356	25	2	1	0	1	24	158	53	22	73	86	91	91
166	3053409	21	2	1	0	1	27.6	160	50.5	20.2	82	76	86	86
167	3053520	30	3	1	1	1	28	155	74	30.8	73	152	109	109
168	3080297	25	2	2	0	2	27.1	141	45.8	22.7	66	155	75	75
169	3106514	26	5	1	3	1	26.2	140	52	21.6	85	142	188	188
170	2826467	22	2	1	0	1	27	150	69	31.3	74	180	117	117

ANNEXURE III - MASTER CHART

Serial number	In / Out patient number	Age (Years)	Obstetric history				Period of gestation (Weeks)	Examination			Investigations			
			Gravida	Para	Abortion	Living		Height (Cms)	Weight (Kgs)	Body mass index (Kg/m2)	GTT 75g (IADPSG)			GTT 75g (DIPSI)
											Fasting	One hour	Two hours	
171	3007842	34	2	1	0	1	26	143	45	22.5	72	146	98	98
172	3129073	19	1	0	0	0	26	156	56	23	81	180	143	143
173	3161774	20	1	0	0	0	28	150	47	21.3	75	62	67	67
174	2899432	24	2	1	0	1	26.3	151	47	19.5	65	105	95	95
175	2929941	30	2	1	0	1	24	156	55	24.3	69	113	97	97
176	3058996	24	2	1	0	1	28	152	65	28	82	101	105	105
177	2910347	24	1	0	0	0	28	147	78	21.4	70	139	131	131
178	2946840	23	3	2	0	1	27.2	158	53	22	85	129	78	78
179	2798134	27	2	1	0	1	27	147	69.7	33.1	92	147	132	132
180	2973343	25	2	1	0	1	27.2	155	51	21.2	71	132	125	125
181	3007716	27	1	0	0	0	25	152	49	21.3	75	86	65	65
182	3007713	25	2	0	0	0	27.3	154	48.5	21	64	72	121	121
183	3097347	27	2	1	0	1	25.1	145	40	19	76	106	111	111
184	2973633	24	1	0	0	0	24	151	50	22.2	98	142	146	146
185	3043661	25	1	0	0	0	26.2	156	55	24.3	97	123	148	148
186	2980407	24	2	1	0	1	24	147	51.1	24	77	133	175	175
187	3062050	22	1	0	0	1	24	156	54	22.5	70	138	114	114
188	2904707	23	2	0	1	0	24	152	42	18.67	59	114	105	105
189	3026306	23	1	0	0	0	28	153	57	24.70	90	101	86	86
190	3135327	21	2	0	1	0	26.5	156	54	22.5	78	140	127	127
191	2860794	26	2	2	0	2	28	151	60	27	77	130	58	58
192	3044192	23	1	0	0	0	26	153	46	20	71	138	112	112
193	3072157	24	1	o	o	o	28	156	52	23.6	86	120	90	90
194	3072172	27	3	3	0	0	28	160	59	23	86	102	134	134
195	2866315	22	1	0	0	0	28	151	50.5	22.4	137	212	147	147
196	3034735	25	1	o	0	0	26	155	50	22.2	62	74	72	72
197	2930199	26	1	0	0	0	26.1	159	68	26.7	70	78	88	88
198	3121536	24	2	1	0	1	28	153	86	21.9	67	89	77	77
199	3129981	24	2	1	0	1	24.3	152	53	23.1	82	98	97	97
200	2893432	24	2	1	0	1	26.4	159	46	18.18	76	95	73	73
201	2887915	24	2	1	0	1	28	166	62	24.2	89	103	79	79
202	2930069	18	1	0	0	0	27.3	155	53	23.6	75	132	109	109
203	3053217	25	5	2	0	2	27.4	153	54	24	82	188	151	151
204	3034357	25	2	1	0	1	26.3	152	48	21.3	68	96	59	59

ANNEXURE III - MASTER CHART

Serial number	In / Out patient number	Age (Years)	Obstetric history				Period of gestation (Weeks)	Examination			Investigations			
			Gravida	Para	Abortion	Living		Height (Cms)	Weight (Kgs)	Body mass index (Kg/m2)	GTT 75g (IADPSG)			GTT 75g (DIPSI)
											Fasting	One hour	Two hours	
205	3053820	21	1	0	0	0	26.2	148	46	23.5	81	99	98	98
206	3077518	24	2	1	0	1	27.3	150	52	23.1	66	98	65	65
207	2896898	23	1	0	0	0	28	138	44	23.1	83	98	81	81
208	3077287	32	5	4	0	4	27.3	155	60	26.7	78	124	84	84
209	2910723	20	1	0	0	0	26	158	47	18.9	70	80	72	72
210	3128806	20	1	0	0	0	27.5	154	58	24.5	91	119	116	116
211	3041031	24	2	1	0	1	24.1	155	60	25	77	105	85	85
212	3135511	26	1	0	0	0	26.6	138	46	24.2	83	111	127	127
213	2956477	20	1	0	0	0	25	153	54	24	82	68	97	97
214	3121643	22	3	2	0	2	24.3	158	74.4	29.9	116	111	111	111
215	3044201	20	2	1	0	1	27.3	152	57	25.3	90	103	91	91
216	2851442	22	2	0	0	1	28	151	57.4	25.3	127	157	162	162
217	2927127	24	2	1	0	1	27.1	150	49	21.8	66	98	65	65
218	2860847	35	3	2	1	1	28	158	60	26.7	84	188	147	147
219	2904678	23	2	1	1	0	27.6	158	58	21.3	88	182	153	153
220	1456720	29	2	1	0	1	24.2	152	42	18.7	78	202	149	149
221	3053131	20	1	0	0	0	28	156	56	21.9	68	126	76	76
222	3105508	30	3	0	1	0	27.2	142	68	34.7	86	186	118	118
223	2177545	20	1	0	0	0	27.5	155	57	25.3	81	131	142	142
224	2980613	20	1	0	0	0	24	160	40	15.6	71	156	147	147
225	3135480	21	2	1	0	1	27.5	133	38	22.5	72	130	81	81