A COMPARATIVE STUDY OF ADA & WHO CRITERIA FOR SCREENING OF GESTATIONAL DIABETES MELLITUS AND FOLLOW UP OF GDM PATIENTS IN SALEM.

Dissertation submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI

In partial fulfillment of the regulations
For the award of the degree of

MASTER OF SURGERY
IN
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DECLARATION BY THE CANDIDATE

I here declare that this dissertation entitled

"**A COMPARATIVE STUDY OF ADA & WHO CRITERIA FOR SCREENING OF GESTATIONAL DIABETES MELLITUS AND FOLLOW UP OF GDM PATIENTS IN SALEM**" is a bonafide and genuine research work carried out by me under the guidance of

Dr.N.GEETHA. MD OG., Associate professor, Department of Obstetrics and Gynecology, GMKMCH,Salem, and

Dr. G.Prakash MD., Dip in diabetes., Associate professor in Diabetology, Government Mohan Kumaramangalam Medical College, Salem. I have not submitted this previously to this university or any other university for the award of any degree or diploma.

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Place : Salem

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ENDORSEMENT BY THE DEAN OF THE INSTITUTION

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It is with great pleasure, I record my deep respects, gratitude and indebtedness to **Dr.B.JAYAMANI**. **MD.,DGO**., Professor and Head of the department, Department of Obstetrics and gynaecology, Govt mohan kumaramangalam medical college, Salem, for her remarkable guidance, encouragement and selfless support which enabled me to pursue the work with

Perseverance and a skillful mind to view and analyze things that appear small to bring forth scientific outcome.

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Office of the Dean,
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Dated:29 .06.2015.

Ethical Committee Meeting held on 18.06.2015 at 10.00 A.M in the Seminar Hall, IInd Floor, Medicine Block, Govt. Mohan Kumaramangalam Medical College Hospital, Salem 01.

The following Members were attended the Meeting.

MEMBERS:

1. Dr. V. Dhandapani, MD., Deputy Chairman, External Social Scientist, ECIRB.
2. Dr. S. Mohamed Musthafa, MD., Vice Principal, Govt. Mohan Kumaramangalam Medical College, Salem.
3. Mr. S. Shanmugam, B.Sc., BL, Advocate, External Legal Expert.
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6. Dr. C. Rajasekaran, MS., Professor and HOD of Surgery, Govt. Mohan Kumaramangalam Medical College Hospital, Salem.
7. Dr. N. Geetha, MD., Associate Professor of Obstetrics & Gynaecology, Govt. Mohan Kumaramangalam Medical College Hospital, Salem.
8. Dr. S. Vijayarangan, MD., Associate Professor of Pharmacology, Govt. Mohan Kumaramangalam Medical College, Salem.

Sl. No.	Name of the Presenter with Address	Title	Name of the Guide and Address	Whether it is Approved or not.
1.	Dr. Y.M. Madhumathi, II Year, Post Graduate Student of MS (O&G), GMKMC, Salem-30.	"A comparative study of ADA & WHO criteria for screening of gestational diabetes mellitus and follow up of GDM patients in Salem".	Dr. N. Geetha, MD., Associate Professor of O & G and Dr. G. Prakash, MD., Associate Professor of Diabetology, GMKMC, Salem-30.	Approved

The Ethical Committee examined the studies in detail and is pleased to accord Ethical Committee approval for the above Post Graduate student of this College to carry out the studies with the following conditions.

1. He should carry out the work without detrimental to regular activities as well as without extra expenditure to the institution to Government.
2. He should inform the institution Ethical Committee in case of any change of study procedure site and investigation or guide.
3. He should not deviate from the area of the work for which applied for Ethical clearance. He should inform the IEC immediately, in case of any adverse events or serious adverse reactions.
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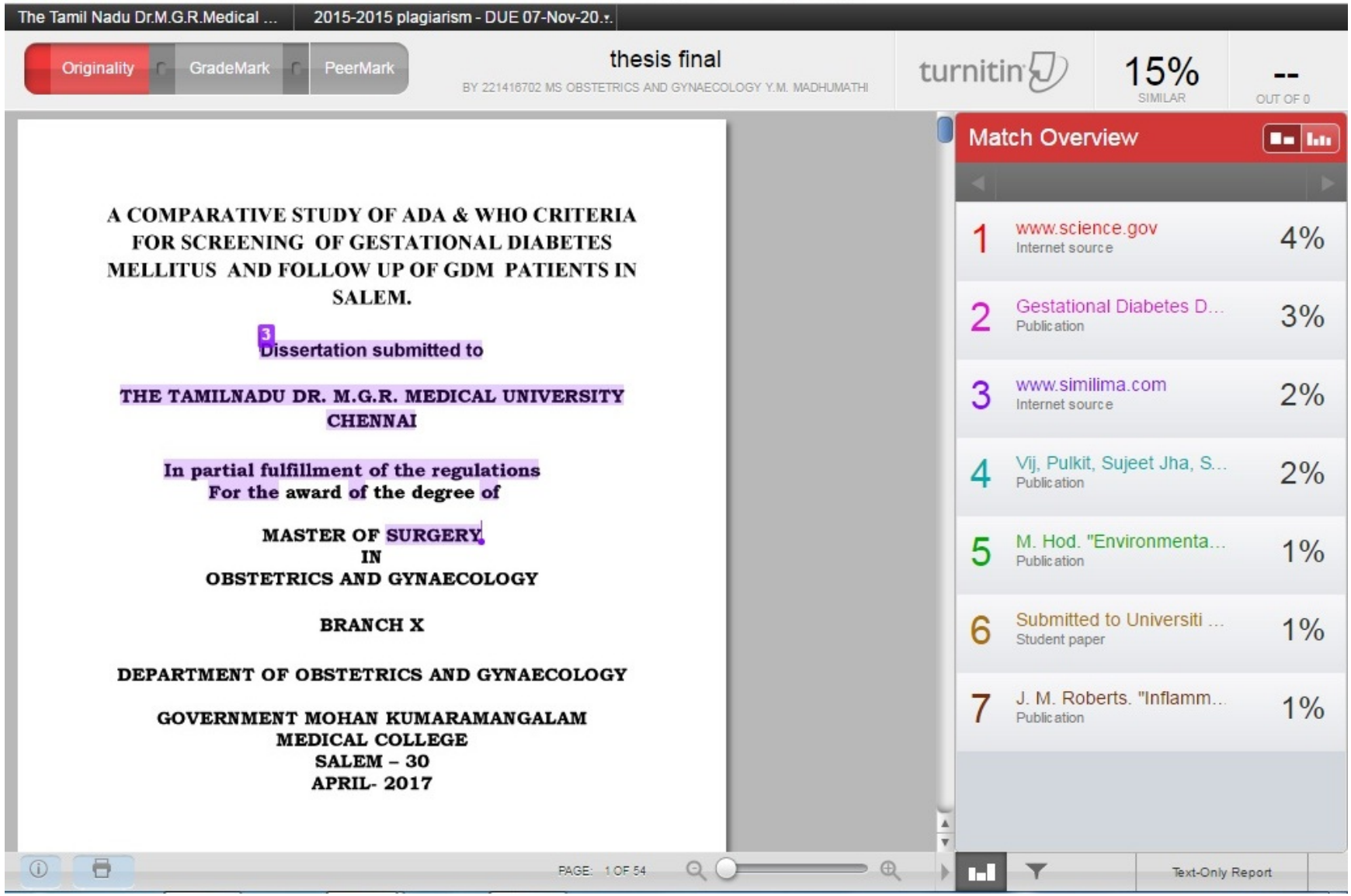
5. He should complete the work within the specific period and if any extension of time is required he should apply for permission again and do the work.
6. He should submit the summary of the work to the Ethical Committee on completion of the work.
7. He should not claim any funds from the institution while doing the work or on completion.
8. He should understand that the members of IEC have the right to monitor the work with prior intimation.

For 29/06/2015

DEAN

LIST OF ABBREVIATIONS:

GDM	-	Gestational diabetes mellitus
IADPSG	-	International Association of Diabetes In pregnancy study group
ADA	-	American Diabetes Association
WHO	-	World Health Organisation
PRAMS	-	Pregnancy Risk Assessment Monitoring System
NDDG	-	National Diabetes Data Group
HAPO	-	Hyperglycemia and Adverse Pregnancy Outcome
RCOG	-	Royal College of Obstetricians and Gynaecologists
NICE	-	National Institute for Health and Care Excellence
OGTT	-	Oral glucose tolerance test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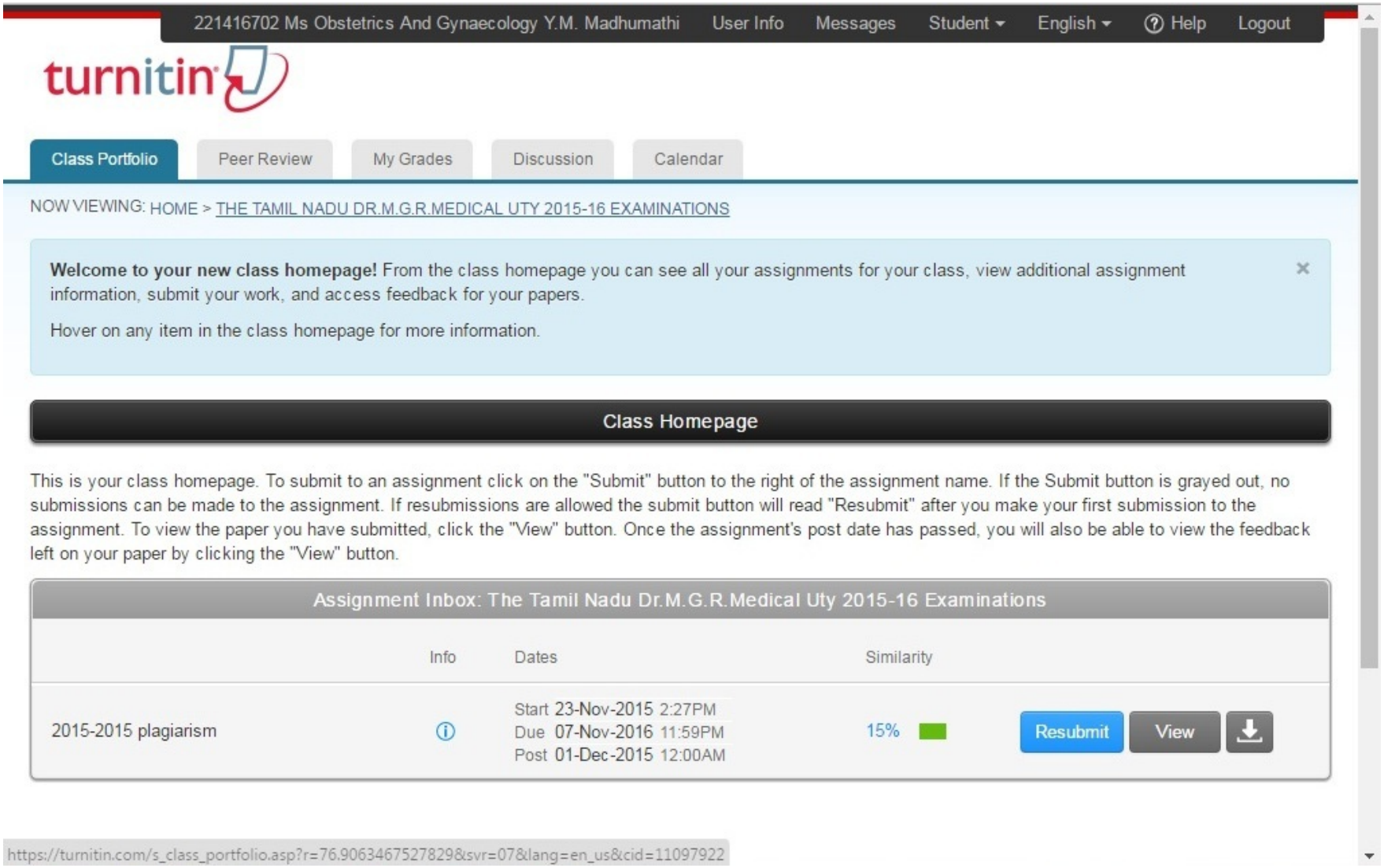
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INDEX

INTRODUCTION

- There is no uniform criteria to be followed for screening of GDM
- Among controversy exist between different associations in screening for GDM,Screening is essential in all Indian pregnant women as there is a increased risk of developing increased glucose intolerance during pregnancy.
- There are both maternal and fetal risk due to uncontrolled hyperglycemia during pregnancy.
- Diabetes in pregnancy increases the risk of obesity and type 2 diabetes mellitus in the offspring as well as the mother in later part of life.Hence glycemic control helps in the prevention of all these complications to the mother and the fetus.
- IADPSG criteria for diagnosis of GDM had been accepted by most associations including ADA. WHO criteria is different that of IADPSG criteria. Hence the study was conducted to compare the two criteria ADA and WHO for diagnosis of GDM in our population.
- There is controversy regarding screening for GDM in AN mothers.
- There is no uniform criteria the world .

AIMS AND OBJECTIVES:

Our study is to compare the ADA & WHO Criteria for screening of Gestational Diabetes Mellitus. Our objective was to study the implications of implementing the ADA guidelines and WHO guidelines for screening and diagnosis of GDM in Salem.

NEED OF THE STUDY:

- The better diagnostic criteria for gestational diabetes mellitus remain controversial.
- It is essential to diagnose GDM early in the pregnancy to avoid GDM related complications.

REVIEW OF LITERATURE

1.1 Definition

1.2 Pathophysiology

1.3 Epidemiology

1.3.1 International scenario

1.3.2 Indian scenario

1.4 Clinical profile and risk factors

1.5 Screening

1.6 Previous studies

1.7 GDM and obesity

1.8 Management of GDM

1.9 Maternal outcomes

1.10 Fetal outcome

1.1 Definition

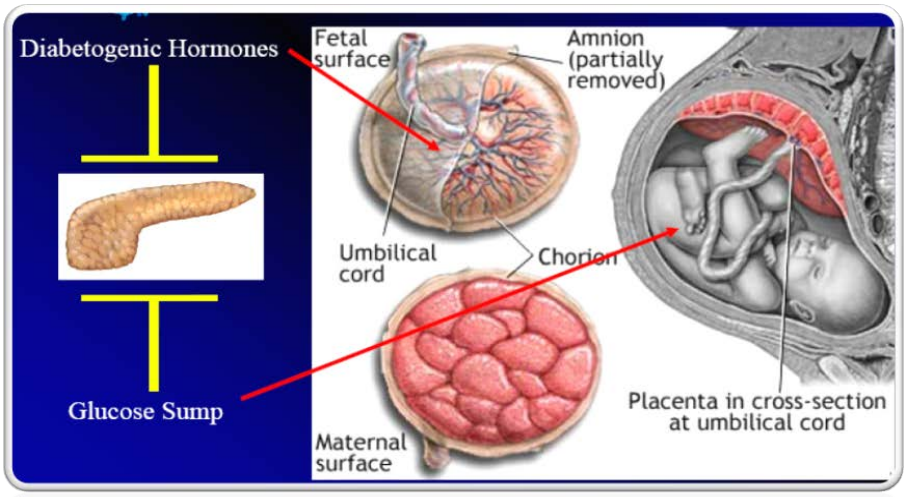
Gestational diabetes mellitus (GDM) is defined as “glucose intolerance first discovered in pregnancy”[6]. The definition applies whether insulin or only diet modification is used for treatment and whether or not the condition persists after pregnancy. It does not exclude the possibility that unrecognized glucose intolerance may have antedated or begun concomitantly with the pregnancy.

Approximately 7% of all pregnancies are complicated by GDM, resulting in more than 200,000 cases annually. The prevalence may range from 1 to 14% of all pregnancies, depending on the population studied and the diagnostic tests employed.

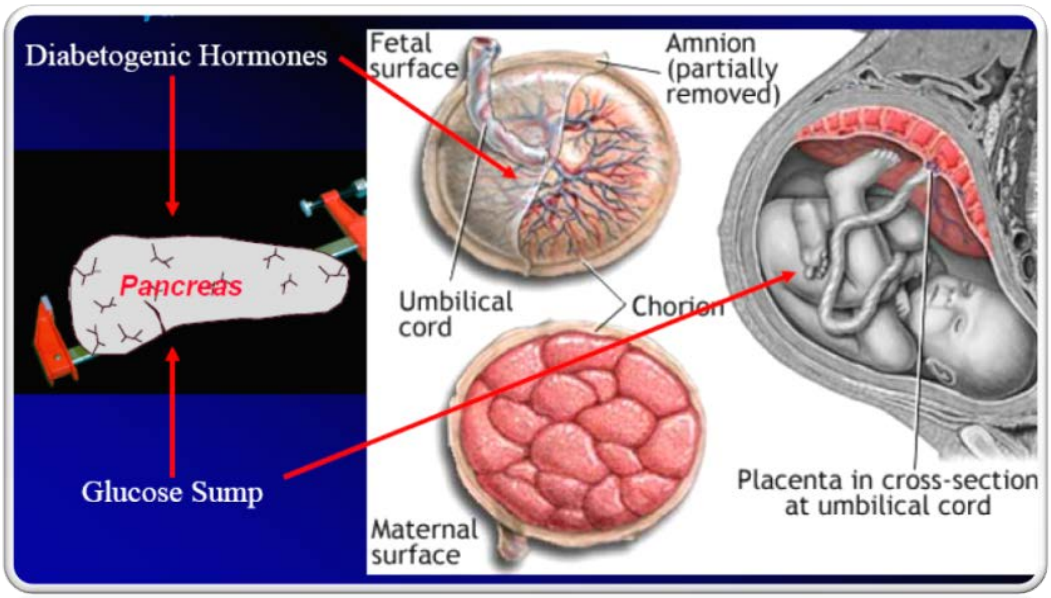
1.2 Pathogenesis

The carbohydrate metabolism undergoes alterations in all normal pregnancy to meet the demand of the fetus. The insulin resistance increases during second trimester which peaks in third trimester. This is due to desensitization of insulin by the placental hormones and weight gain (adiposity) occurring in pregnancy[7]. This is counteracted by raise in insulin secretion in normal pregnancy. Shortly after pregnancy insulin resistance normalises supporting the above said theory.

NORMAL GLUCOSE METABOLISM



ALTERED GLUCOSE METABOLISM

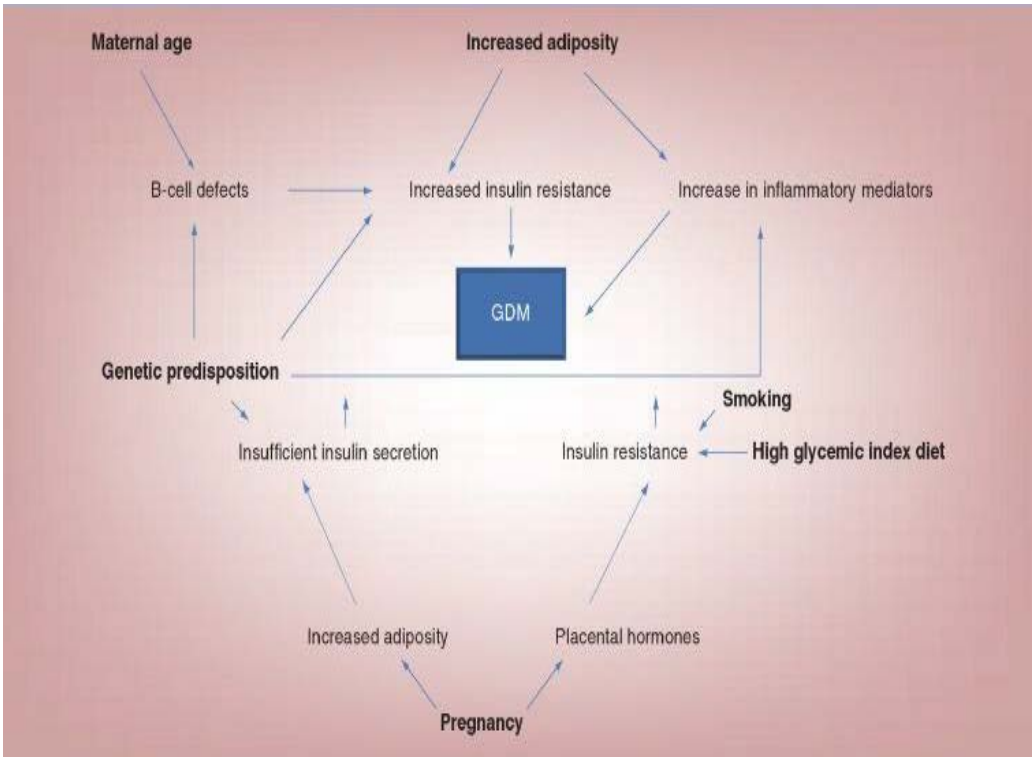


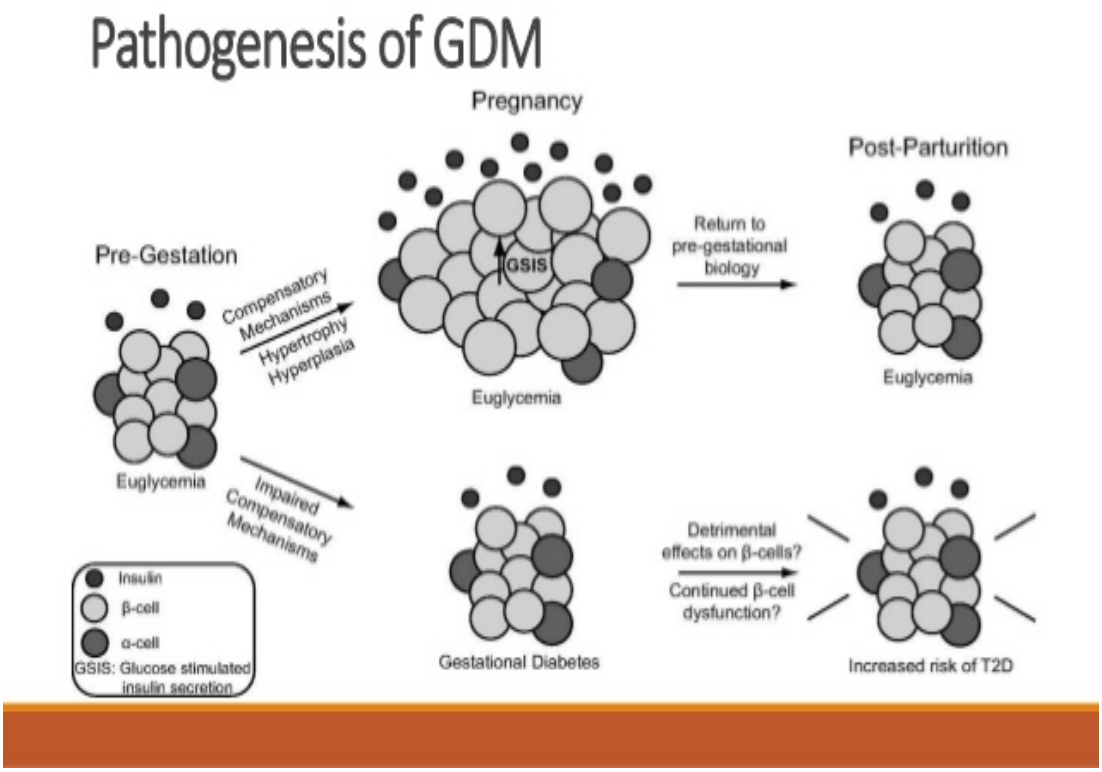
In patients with GDM, there are various mechanisms by which play a role in causation of GDM. The mechanisms include dysfunction of beta cell, chronic insulin resistance and autoimmunity.

Beta cell dysfunction plays a central role that in women with GDM the insulin levels does not rise as expected in a normal pregnant women. Homko et al[8] and Buchanan et al[9] through their studies have proven that chronic beta cell dysfunction underlies the pathogenesis of GDM. They had said that steadily as insulin resistance increases in later part of pregnancy, there is progressive loss of beta cells as it is unable to meet the high insulin demand in GDM women as compared to healthy women. Though the etiology is not clear, some studies have tried to relate it to genetic causes with alleles linked to calpain-10 gene, sulphonyl urea receptor -1 gene() etc..

Chronic insulin resistance also seems to play a role in pathogenesis of GDM. This is due to post receptor signalling defect which is thought to be predate pregnancy and this might be helped by other factors like obesity[10]. This theory is supported by the fact that after delivery the insulin sensitivity normalises in healthy women but not in GDM women.

Pathophysiology





Recently **HLA-G** also been linked to pathogenesis of GDM which is considered to protect the fetus and the antigenic load it renders by down regulation of T cells response to fetal trophoblasts[10]. Other rare causes include autoimmune destruction of pancreas with autoantibodies directed against islet of langerhans (islet cell antibody), beta cell antigens (anti - GAD antibodies). Monogenic causes account for less than 10% of cases and of that MODY- 2, MODY- 3, MODY- 4 have been linked to pathogenesis of GDM.

1.3 Epidemiology

The prevalence varies among different population groups and it depends on the criteria used for gestational diabetes. The data is important that there has been a 16% - 127% increase in prevalence of GDM over past 20 years and it helps in planning health schemes and allocating resources.

1.3.1 International scenario

In 2010, PRAMS (Pregnancy Risk Assessment Monitoring System) in their survey done in United States between 2007-2010 reported a prevalence of 9.2% for gestational diabetes mellitus. Australian institute of health and welfare have reported a prevalence of 4.6% in 2005-2006[11].

1.3.2 Indian scenario

In India the prevalence varies widely among regions. In a study done by Rajput et al in haryana in 2013, the prevalence of GDM was 7.2%[12]. In a study done in Rajasthan by Kalra et al, the prevalence of GDM was 6.6%[13]. In another study done in Tamil Nadu in 2008 by Seshiah et al, GDM was detected in 17.8% women in urban, 13.8% percent women in semi-urban and 9.9% percent women in rural areas[14].

1.4 Risk factors

American diabetes association has divided the patients with risk factors for GDM into high, average and low risk categories. High risk factors are advanced maternal age, obesity, glycosuria, a family history of type 2 diabetes and a history of GDM in previous pregnancy. Low risk factors include age less than 25 years, normal weight prior to pregnancy, ethnic group less prone for GDM, no history of diabetes in first-degree relatives and previous GDM, no bad obstetric history[15].Ethnic groups like asian, african, american and hispanic women are more prone for GDM.

Other risk factors include short maternal stature, polycystic ovarian disease and multiple pregnancies. Some studies have reported western type dietary pattern and physical inactivity as risk factors for GDM.

1.5 Diagnosis of GDM :

1.5.1 Screening

Inspite of many studies and evidences we have on GDM, still there is arguments between universal and selective screening for gestational diabetes. While ADA advices risk factor based screening dividing the patients into high and low risk factor groups, we Indians are at a higher risk for gestational diabetes compared to any other population, compelling the practice of universal screening in our population. In a study by Griffin et al concluded that risk factor based screening missed about half the cases and pointed out incidence doubled from 1.45% to 2.7% by universal screening in the same population [16].

Though universal screening may not be feasible in resource poor settings, definitely universal screening is expected to improve the pregnancy outcomes considering the high prevalence of gestational diabetes in india.

1.5.2 Diagnostic criteria – evolution

Many diagnostic criteria have been proposed for gestational diabetes in the past 50 years and have been debated always. Initially O'Sullivan and Mahan formulated criteria in 1964 which recommended 100 g 3 hour oral glucose tolerance test. The threshold values were framed based on ≥2 SD above the mean of normal population and its reflection to predict the future risk of diabetes. In late 1970 s, the National diabetes data group (NDDG)[17] and carpenter & cousten[18] modified the values and these values came into practice. The National diabetes data group titrated up the blood glucose values by 15% for the difference in way of analysis of blood glucose. Carpenter and cousten criteria had its justifications to alter the threshold values lower than the NDDG values and attributed it to change in techniques to measure glucose from non glucose substances. In these criteria, GDM was diagnosed when 2 out of 4 values were out of range.

In 2007, there was a major breakthrough when results of HAPO study[19] was published. It clarified that adverse pregnancy outcomes showed a strong continuous association as a function of maternal glycemic levels that were considered below the threshold of gestational diabetes.

In 2008, IADPSG consensus panel reviewed the results of the HAPO study and proposed a diagnostic criterion. The threshold values were framed based on the 1.75 times the estimated odds for birth weight >90th percentile, cord C-peptide >90th percentile, and percent body fat >90th percentile[20].This criteria was unique that 75 g glucose was used and recommendation is that if one value was out of range it is enough to label it as gestational diabetes. Other thing was it provided a definition for overt diabetes and it was first of the kind to consider pregnancy outcomes for framing the threshold values. The new criteria can double the prevalence of GDM as it considers one value above range as GDM nevertheless considering the impact of adverse outcomes in GDM, ADA has recommended IADPSG criteria in its guidelines published in 2011.

1.5.3 PREVIOUS STUDIES:

There is always a debate about which criteria is better in diagnosing GDM. There are few studies to find an answer for this.

In a study by Haritha[41] et al for comparison of different criteria for iagnosis of gestational diabetes mellitus, it was concluded that a single 2 h plasma glucose is both easy to perform and economical. Also they suggested for revised WHO criterion using a 2 h threshold of ≥140 mg % to be adopted as a one-step screening and diagnostic procedure for GDM in our country. In the St. Carlos Gestational Diabetes Study by Alejandra Duran et al, it was found that the application of the new IADPSGC was associated with a 3.5-fold increase in GDM prevalence, as well as significant improvements in pregnancy outcomes, and was cost-effective[42]. In another study by Shirazian N et al, they found a higher frequency of occurrence of GDM was 6.1% in a 75-g OGTT based on ADA criteria, and there was fair agreement between ADA and WHO criteria[43].

1.6 GDM and comorbidities

1.6.1 GDM and obesity:

Prepregnancy obesity is independently associated with GDM and both have their roots to common pathophysiology of hyperglycemia, hyperinsulinemia and insulin resistance though obesity also affects pregnancy outcomes with other mechanisms other than insulin resistance. In HAPO study which included 23,316 women reported an incidence of 13.7 % for obesity (BMI > 33 kg/m2) and 25% of diagnosed GDM (IADPSG criteria) mothers were obese[21].

In work done by chu et al which was a meta analysis of 20 studies concluded that the odds ratio were 2.1, 3.5, 8.5 for overweight, obese and severely obese for developing GDM compared to normal pregnancies respectively[22].In other words the risk of developing GDM was two, four and eight fold higher in overweight, obese and severely obese population by the same study.

By HAPO study,the maternal complications which were strongly associated with both GDM and obesity were preeclampsia, primary cesarean delivery and shoulder dystocia. The fetal complications that were associated with GDM and obesity were birth weight > 90th percentile, cord C peptide > 90th percentile and percent body fat > 90th percentile. The mean difference of birth weight between the group with GDM and obesity as compared to group with normal weight and normal glucose tolerance was 339 grams which was significant[21].

The same associations have been proven by randomized controlled trials done by Crowther et al[3] and Landon et al[23] that with management of GDM, maternal weight gain during pregnancy decreased in the treated GDM mothers as compared with the mothers in control group. So controlling the weight gain during pregnancy not only reduces the perinatal complications in present pregnancy but also in future pregnancies through decreased postpartum weight retention.

1.7 Management of GDM

Medical nutrition therapy

Medical nutrition therapy forms the backbone of management of gestational diabetes. ADA recommends individualisation of diet such that the calories are met to satisfy the needs of pregnancy and to maintain a glycemic goal of fasting blood sugar less than 105 mg/dl, 1 hr less than 155 mg/dl and 2 hr less than 130 mg/dl[16]. ADA also mentions restriction of calories to 30 - 33% and restriction of carbohydrate diet to 35 - 40 % in obese women. ACOG recommends a glycemic control which is lower than recommended by ADA i.e fasting blood sugar less than 95 mg/dl, 1 hr less than 140 mg/dl and 2 hr less than 120 mg/dl[23].

In a study, 215 women with GDM were randomized to either MNT or standard care. Fewer subjects in the MNT group required insulin (24.6% vs 31.7%, $p = 0.05$)

Physical activity

Like in diabetes mellitus there is modest benefit from exercise in women with GDM and so it is recommended in GDM light and moderate intensity exercise unless contraindicated till later stages of pregnancy. A randomised trial carried out in 32 women to circuit type exercise three times a week or control, found that resistance training resulted in lower postprandial glucose levels and a delay in the requirement of insulin[24].

Insulin

Insulin is recommended when blood glucose cannot be controlled with medical nutrition therapy alone. Basal bolus regimen is considered better than twice daily regimen with basal dose generally given in night and if needed a dose repeated in morning. The dose of insulin has to be individualized as level of insulin resistance varies among individuals with requirements higher in later part of third trimester of pregnancy and the incidence of hypoglycemia in GDM is lesser compared to type 1 diabetes.

Human insulin is being used for a long time without any adverse effect on the fetus and it does not cross placenta. Newer short acting insulin Lispro and Aspart are also now shown to be as effective as human insulin and without any adverse effects [25][26]. There is not much data available on long acting insulins and their efficacy.

Oral hypoglycemics

Glibenclamide (Glyburide)

Glibenclamide is considered as effective as insulin and in United States it has started replacing insulin. More than half a dozen studies are available and these changes are driven by a study done by Langer et al which was a randomised trial carried out among 404 women. In that trial only 4% in glibenclamide group required additional insulin for control. Glibenclamide was not found in cord blood and the percentage of neonates with large for gestational age and macrosomia was similar. More importantly the incidence of neonatal hypoglycemia and congenital anomalies did not differ between groups[27].

Metformin

Metformin is considered at least as effective as insulin and has been considered as preferred treatment by women with gestational diabetes. Initially the evidences were conflicting until the publication of large randomised Metformin in Gestational diabetes (MiG) study done among 751 women with GDM[4]. It concluded that neonatal complications were similar between metformin and insulin. By that study 42% required supplemental insulin among metformin group in whom the maximum dose was 2500 mg. The numbers of preterm births were slightly higher in metformin group though the congenital anomalies did

not differ among groups. There are no head to head trials available between glyburide and metformin but considering metformin's insulin sensitizing action and decreased gluconeogenesis it theoretically seems to be a better choice.

Postpartum management of GDM

The blood glucose levels are expected to normalise after delivery but GDM women are at higher risk of developing type 2 diabetes later. So women who had GDM should undergo regular screening for diabetes mellitus. ADA recommends screening after 6-12 weeks after delivery for which 75 gm OGTT or fasting blood sugar or HbA1C can be used. A lifelong screening every three years has also been recommended [16].

Apart from screening, the importance of lifestyle modifications has to be stressed to women with GDM. The Finnish Diabetes Prevention Study and Diabetes Prevention Program have underlined the importance of lifestyle modification that 5-7% weight reduction helps in preventing or delaying diabetes in later life[28][29].

1.8 Effects of GDM on Outcomes:

The effect of overt diabetes is known to be associated with adverse maternal and fetal outcome.

The consequences of GDM over the outcomes had been a controversy for a long time until the publication of HAPO study results but the association of varying degrees of glycemia related to adverse outcome is not clear. In addition to this there might be many confounding factors that might be related to adverse outcomes viz., obesity, preeclampsia, advanced age and other comorbidities which are usually associated with GDM. Nevertheless the adverse outcomes can be divided into short and long term effects of GDM on mother and infant.

1.8.1 Maternal outcome - short term

Cesarean section

GDM women are at increased risk for cesarean section which has been evaluated through various studies. But the rates of cesarean section are variable across studies done in and out of india. In HAPO study primary cesarean section was considered a primary outcome. There was a weaker association between primary cesarean section and maternal glycemia (odds ratio 1.1)[20]. But other studies showed a significantly higher rate of cesarean section in GDM women. In a study done by Landon et al[23] on treatment of mild GDM concluded that the cesarean rates were in general higher and it was 26% in treatment group against 33% in routine care group which attained statistical significance. In a study done by Mahalakshmi et al which was a retrospective study done among 1003 GDM women in tamilnadu reported 41% of elective cesarean section and of which 24% were emergency cesarean section[30].

Premature rupture of membranes

The American College of Obstetricians and Gynecologists have classified spontaneous preterm birth into preterm premature rupture of membranes, spontaneous preterm labour and cervical incompetence.

In a study done by Hedderson et al among 46,230 healthy and GDM women, spontaneous preterm birth was seen in 4.2% of total pregnancies. Among GDM women the spontaneous preterm birth was seen in 6.7% of pregnancies. They had found that the rates of spontaneous preterm birth were steadily increasing as a function of blood glucose levels after adjustment for birth weight, pregnancy induced hypertension and polyhydramnios[31].

Shoulder dystocia

Shoulder dystocia is defined as “a vaginal cephalic delivery that requires additional obstetric manoeuvres to deliver the fetus after the head has delivered and gentle traction has failed”[32]. More objective way of defining shoulder dystocia is a delay of more than 60 seconds for head to body delivery.

The overall incidence of shoulder dystocia is variable among vaginal deliveries and reported between 0.5 - 0.7%[33].It is more common in diabetic deliveries and the reported incidence is 2.4%[34].And the risk is

two to four fold higher in infants of diabetic mother compared to infants of non diabetic mothers of same birth weight[35].

Shoulder dystocia has certain risk factors which includes antepartum and intrapartum factors viz previous history of shoulder dystocia, macrosomia, patient with diabetes or gestational diabetes, BMI>30 kg/m2 and intrapartum factors like prolonged first or second stage of labour, assisted vaginal delivery. Though treating physician should be aware of the risk factors, going by the risk factors alone does not prevent shoulder dystocia which was the conclusion made by RCOG.

The management of shoulder dystocia focuses on the mode of delivery. It is against the odds of vaginal delivery which might place the infant at risk of shoulder dystocia and consequent brachial plexus injury against a elective cesarean delivery which has a threefold increased risk of postpartum infection and 11 fold increased risk of wound complication[34].Considering former the reported incidence of brachial plexus injury in macrosomic infants (>4000 g) of diabetic mother allowed for vaginal delivery is 2-5% and of the brachial plexus injury sustained only 6.7% persists according to work done by Rouse and owen[36].It is recommended that elective cesarean has to be performed for pre-existing or gestational diabetes mothers with estimated birth weight of 4.5kg.Nevertheless it is the fetal risk delivered by vaginal delivery against

the maternal morbidity conferred by cesarean delivery. Apart from mode of delivery it is known that induction of labour reduces the incidence of shoulder dystocia in diabetic patients though not in pregnacies not complicated by diabetes with suspected macrosomia.

The maneuvers used for delivery of shoulder dystocia are beyond the scope of this review.

1.8.2 Long term comorbidities

GDM women have a higher risk of developing type 2 diabetes in later life. Around 50 percent of women with GDM go on to develop type 2 diabetes within five to 10 years. The risk of developing GDM depends on many factors which include gestational age at diagnosis, initial

maternal glycemia, subsequent pregnancies, obesity and beta cell dysfunction. Apart from type 2 diabetes the risk of cardiovascular disease and metabolic syndrome is higher in GDM women.

Table 1: Risk factor for post partum diabetes
Ethnicity (e.g. African-American, Latino, Native American, Asian American, Pacific Islander)
Age at delivery ≥ 33-35 years
High Parity
Family history of diabetes
Duration of follow up after pregnancy
Testing modality for diagnosing diabetes (e.g. Oral glucose tolerance test, Fasting plasma glucose, Random plasma glucose or Hemoglobin A1C)
Early Diagnosis of gestational diabetes mellitus (< 22-24 weeks)
Severity of gestational diabetes mellitus:
- Degree of hyperglycemia in pregnancy and immediately postpartum
- Total area under the diagnostic Oral glucose tolerance test
- Number of abnormal Oral glucose tolerance test values
- Level of fasting blood glucose on the Oral glucose tolerance test
- Need for pharmacological therapy to achieve glycemic control
- Elevated fasting glucose level during pregnancy
Lifestyle parameters:
- Limited physical activity
- Consumption of dietary fat
- Smoking
Maternal Weight:
- Pre-pregnancy weight and Body Mass Index
- Gestational weight gain
- Postpartum weight retention

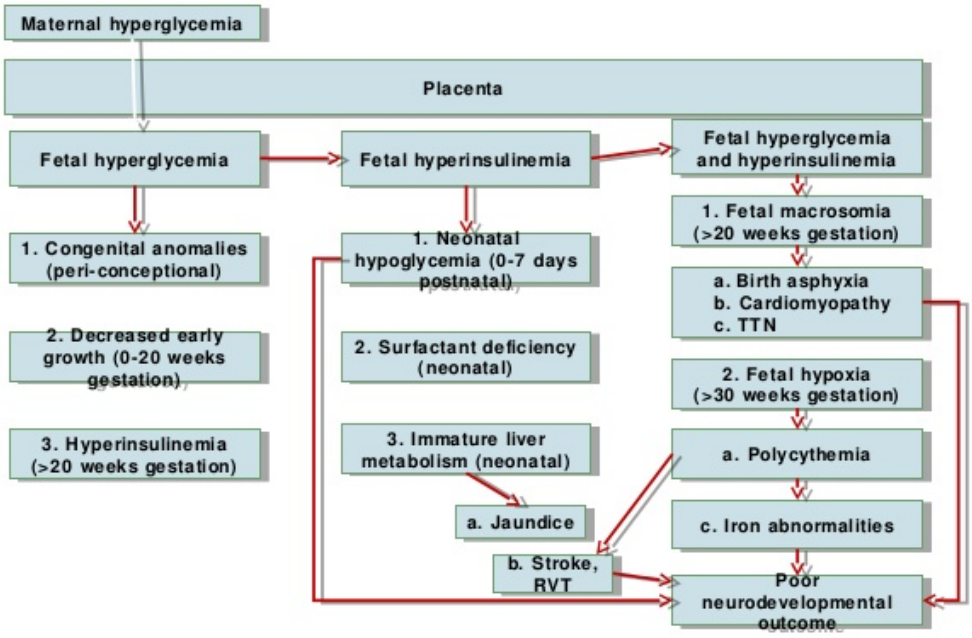
Table 1: Risk factor for post partum diabetes
Metabolic syndrome parameters at early postpartum:
- Waist circumference of 88cm or higher
- High-density lipoprotein cholesterol > 50mg/dL

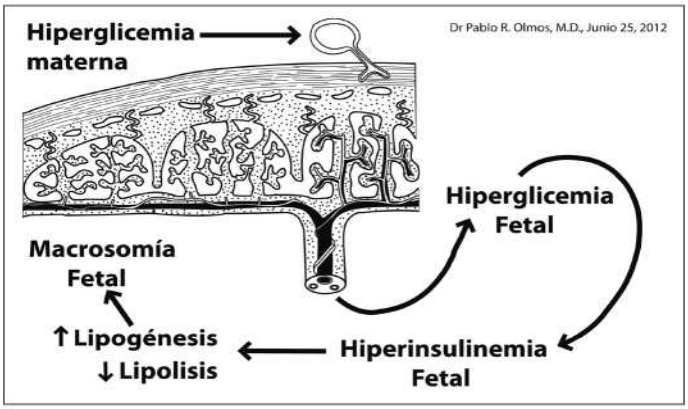
1.8.3 Fetal outcome

The infant of diabetic mother is prone for complication during all the stages viz fetal, neonatal and adulthood.

The Pedersen hypothesis states that fetal hyperglycemia is due to maternal hyperglycemia as glucose readily crosses placenta. Going by the age old hypothesis, the consequences and complications of GDM on fetus can be in part explained by maternal glycemic control. The fetal pancreas start functioning after 20 weeks of gestation and its known fact maternal insulin does not traverse the placental barrier to significant levels. Hyperglycemia in mother causes fetal islet hypertrophy and subsequent hyperinsulinemia. So the adverse outcomes of infant of diabetic mother are due to combined effects of fetal hyperglycemia and hyperinsulinemia.

Pedersen Hypothesis





Growth

In pregnancy complicated by GDM, the fetus is exposed to hyperglycemia from mid - second trimester in most patients, followed by secondary hyperinsulinemia which takes some weeks for fetal islet cells to respond by undergoing islet hypertrophy with an increase in insulin production.

This combination of hyperinsulinemia and hyperglycemia,exerts an anabolic effect resulting in a striking increase in fat stores and protein stores in third trimester. The resultant effects of these changes culminate in weight accretion after 32 weeks of gestation leading to macrosomia. As it is known macrosomia poses a greater threat for birth injury because of cephalopelvic disproportion.

Fetal oxygenation

Chronic fetal hyperglycemia and hyperinsulinemia increases the fetal basal metabolic rate, with secondary change in erythropoiesis and fetal oxygenation. This leads to 30% increase in fetal total body oxygen consumption in a relatively oxygen-limited environment[37].Though the fetus tries to compensate by increasing its rate of oxidation, the placenta has limited ability to increase oxygen delivery to meet the increasing demand. This results in relative hypoxemia and fetus again adapts by

increasing oxygen carrying capacity which is evident in cord blood by elevated cord serum erythropoietin concentration. This results in polycythemia.

Congenital anomalies

The structural anomalies commonly occur during first 2 months of gestation when organogenesis takes place. So congenital anomalies are common in infants born to diabetic mother than in infant of mother with GDM.

Neonatal Complications

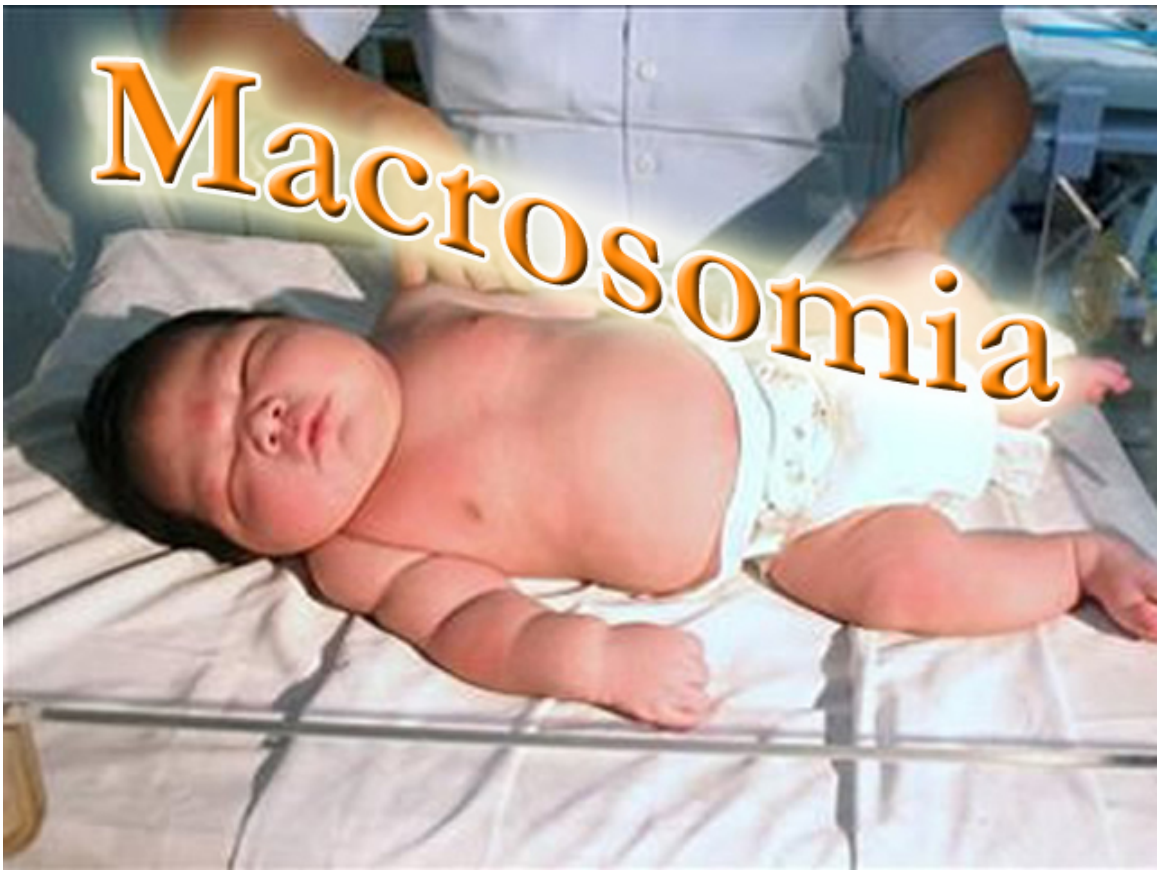
Preterm delivery

Preterm delivery is usually defined as “delivery less than 37 weeks gestation”. In the HAPO study, there were 6.9% of preterm deliveries. Preterm delivery had weaker association with maternal glucose levels with adjusted Odds ratio of 1.05 for fasting glucose levels in the same study[19].

Macrosomia

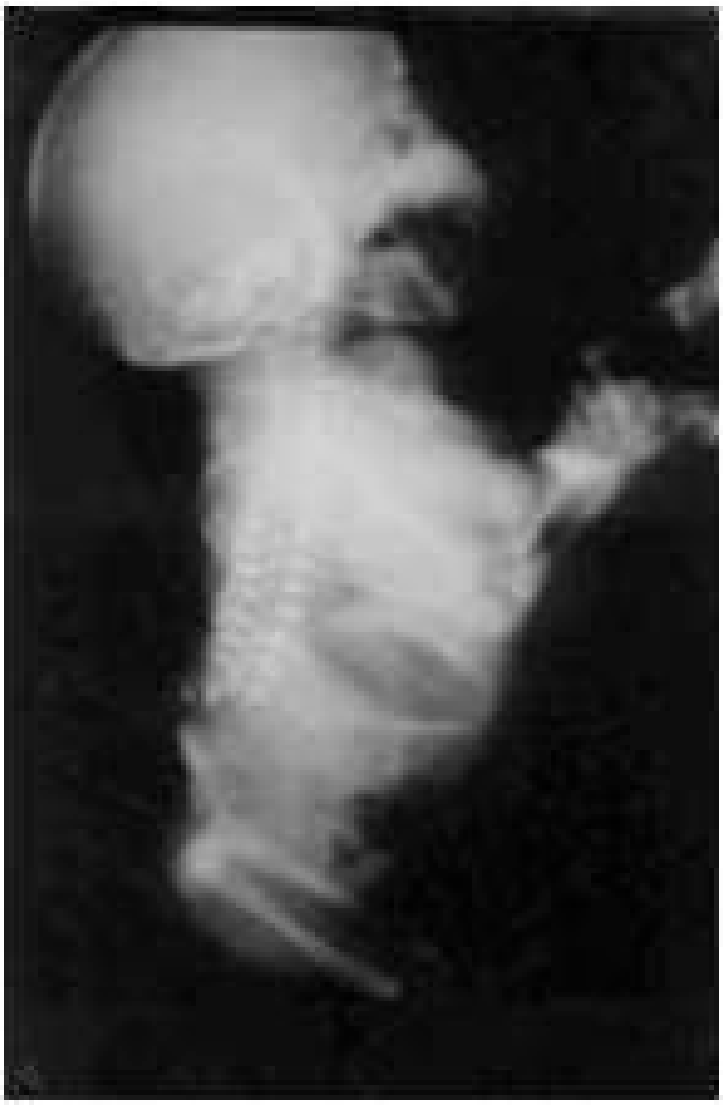
Neonatal macrosomia is directly proportional to poor glycemic control during pregnancy. Macrosomia in GDM is peculiar compared to large for gestational age neonates of normal pregnancy because of the deposition of increased fat mass is not proportionate, having higher weight than length and head circumference percentiles. These differences

in disproportionate growth can be detected through measurements, such as the Ponderal Index or the mid-arm-circumference-to-head-circumference ratio. Macrosomia at birth not only indicates the glycemic control of mother but also is a marker for detecting the subsequent neonatal morbidity, including polycythemia, hypoglycemia, hypocalcemia and intraventricular cardiac septal hypertrophy.



CAUDAL REGRESSION SYNDROME

CAUDAL REGRESSION SYNDROME



In HAPO study, there was a continuous association between increasing levels of fasting, 1-hour, and 2-hour plasma glucose of OGTT and birth weight above the 90th percentile. Among the primary outcomes of HAPO study, odds ratios was highest for birth weight greater than the 90th percentile (OR 1.38 to 1.46) in relation to increasing levels of maternal glycemia[19]. This shows a strong association between birth weight and maternal glycemic control. In ACHIOS trial, the average birth weight of neonates in treatment group were 3335 g as compared to 3482 g in routine care group which shows the treatment effect on fetal growth[3]. In a study done in india by Kale et al, the average birth weight was 2.93 kg compared to 2.80 kg in non-GDM mothers[38].

Glucose metabolism

Hypoglycemia is the frequent metabolic complication and it is common in infants with macrosomia and growth retardation. There is a sudden interruption of free passage of glucose from mother to fetus post delivery, with high neonatal insulin levels. This result in neonatal hypoglycemia in case of macrosomic neonates while the cause of hypoglycemia in growth retarded neonates is due to depleted glycogen stores and the overall incidence seems to be around 50%.The drop in blood glucose usually occurs between 1 and 3 hours of life. The definition of hypoglycemia is

still controversial. But practitioners recommend to treat infants with values less than 40 mg/dL.

The primary objective should be to prevent neonatal hypoglycaemia is by reducing islet cell hyperplasia by maintaining strict glycemic control throughout pregnancy.

Calcium and magnesium metabolism

Hypocalcemia and hypomagnesemia occurs in first 72 hours of birth and the incidence is around 50%.Neonates with respiratory distress or who had asphyxiated are at higher risk.The cause of hypocalcemia might be due to a delay in this postnatal parathyroid hormone response. Hypomagnesemia defined as a serum magnesium level < 1.5 mg/dL. It is associated usually with hypocalcemia.

The signs and symptoms of neonatal hypocalcemia and hypomagnesemia are similar to those of hypoglycemia and include sweating, jitteriness, irritability and seizures except for the little late presentation between 24 to 72 hours. Only IDMs with symptomatic hypocalcemia and symptomatic hypomagnesemia should be treated.

1.8.4 Fetal outcome - long term comorbidities

The long term issues of infant of diabetic mother are obesity, diabetes and neurocognitive impairment in later life. Hillier et al has found higher risk of overweight among children of untreated GDM women. In children of treated and mild GDM women the risk of childhood obesity was not high[39]. Gillman et al in his study among infants of mild GDM women have found that though the macrosomia risk is high at birth, weight of children does not differ at the end of 5 years[40]. The data about the neurocognitive development are limited but it is believed infant of diabetic mother have delayed cognitive development due to metabolic alterations in utero.

METHODOLOGY

STUDY DESIGN:

This is a prospective comparative study.

STUDY POPULATION:

The study population was selected from ante natal out-patient clinic in the department of obstetrics and gynaecology, GMK Medical College & Hospital.

INCLUSION CRITERIA:

All ante-natal mothers of gestational age 24 to 28 weeks

EXCLUSION CRITERIA:

Pre-existing diabetes

SAMPLE SIZE:

200

STUDY PERIOD:

July 2015 to June 2016

Laboratory investigations were performed in the Biochemistry Department,GMKMCH, Salem

ETHICS:

The study was conducted in accordance with the ethical rules of Government Mohan Kumaramangalam Medical College, Salem.

Informed written consent to participate in the study was obtained from all participants.

METHODOLOGY:

All antenatal women attending between 24 and 28 weeks of gestation are subjected to fasting blood glucose measurement followed by an oral glucose tolerance test (OGTT) using 75 g glucose load. Then blood sugar values are collected by venipuncture at the end of 1-h, and 2-h. The ADA and WHO criteria were applied separately for each subject to diagnose GDM.

According to the ADA criteria, presence of any one of either fasting-92 mg/dl (5.1 mmol/L), or 1 h-180 mg/dl (10.0 mmol/L) or 2 h-153 mg/dl (8.5 mmol/L) was used for diagnosis of GDM.

According to the WHO criteria, any one of either fasting-126 mg/dl (7.0 mmol/L) or 2-h value-140 mg/dl (7.8 mmol/L) was used for diagnosis of GDM.

This is a point of care study and no further follow ups were done. The data obtained will be compared to know the usefulness of the 2 criterias in diagnosing GDM.

COLLECTION OF SAMPLE:

Antenatal women with gestational age between 24 to 28 weeks were asked to come in fasting state. Fasting blood sample taken. 75 grams glucose given. 1hour and 2 hour blood samples taken. The samples were sent to laboratory for immediate processing and reporting.

RESULTS:

The study was conducted in department of obstetrics and gynaecology in government mohan kumaramangalam medical college and hospital. A total of 200 patients were included in the study. Results are discussed below

Clinical characteristics:

The average age of patients with GDM at presentation was 25 years. The youngest patient was 19 years and oldest was 40 years. There were nine patients aged 20 years or below, 171 patients in the age group of 20 to 30 and twenty patients were above the age of 30 years. The average gestational age at the time of inclusion in the study was 26.2 weeks. The median gravida of patients with GDM was one. Ninety two patients were primigravida.

Table 1. Patient characteristics:

Patient characteristics	Mean (SD)	Range
Age (in years)	25.0 (5.5)	19-40
Height (in cm)	153.1 (7.7)	133-195
Weight (in kg)	55.5 (10.7)	34-91
Gestational age (in weeks)	26.2 (1.8)	24-36

Table 2. Age category of the study participants:

Age of the participants	Total number of participants N (%)
≤20 years	9 (4.6)
20-30 years	171 (85.3)
>30 years	20 (10.2)
Total	200

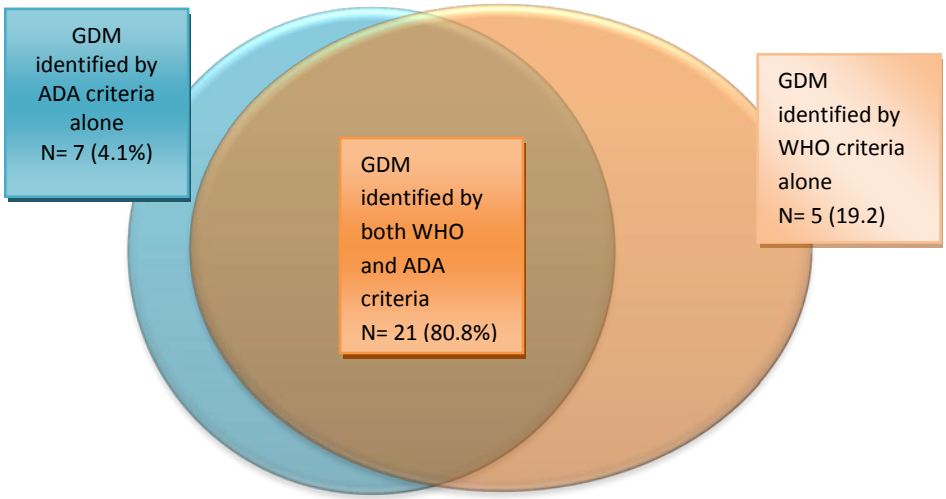
Table 3. Gravida of study participants:

Gravida	Total number of participants N (%)
1	92 (46.7)
2	63 (30.5)
3	33 (16.8)
4	11 (5.6)
5	1 (0.5)
Total	200

Diagnosis of GDM by WHO and ADA criteria:

Among the study population, 28 patients were diagnosed to have GDM applying ADA criteria whereas 26 patients by WHO criteria.

Fig 1. Proportion of GDM patients diagnosed by WHO and ADA criteria:

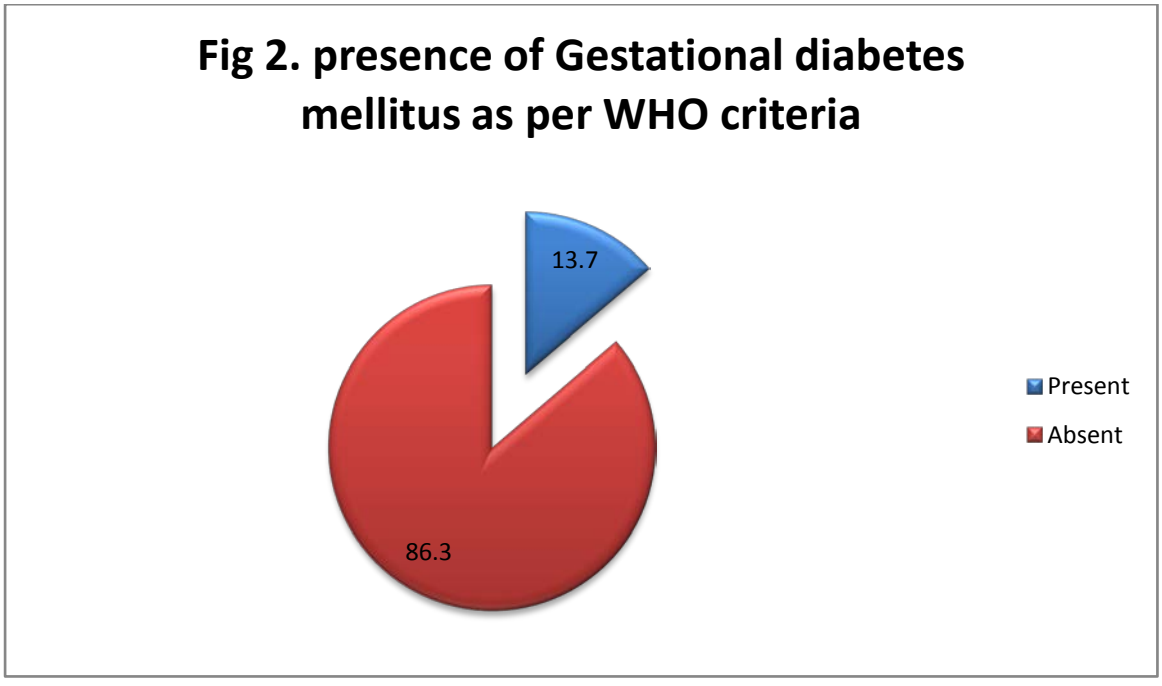


Diagnosis by WHO criteria:

26 patients were diagnosed to have GDM as per WHO criteria.

Table 4. Proportion study participants diagnosed with gestational diabetes mellitus as per WHO criteria:

Gestational diabetes mellitus as per WHO criteria	Total number of participants N (%)
Present	26 (13.2)
Absent	174 (86.8)
Total	200



Diagnosis by ADA criteria:

28 patients were diagnosed to have GDM as per ADA criteria.

Table 5. Proportion study participants diagnosed with gestational diabetes mellitus as per ADA criteria:

Gestational diabetes mellitus as per ADA criteria	Total number of participants N (%)
Present	28 (14.2)
Absent	172 (85.8)
Total	200

Out of 28 cases positive for GDM by ADA criteria,

Cases identified by one hour value alone is 1 case, by two hour value alone are 15 cases, both 1 hour and 2 hour are 12 cases. Thus one hour value is statistically not significant compared to two hour value.

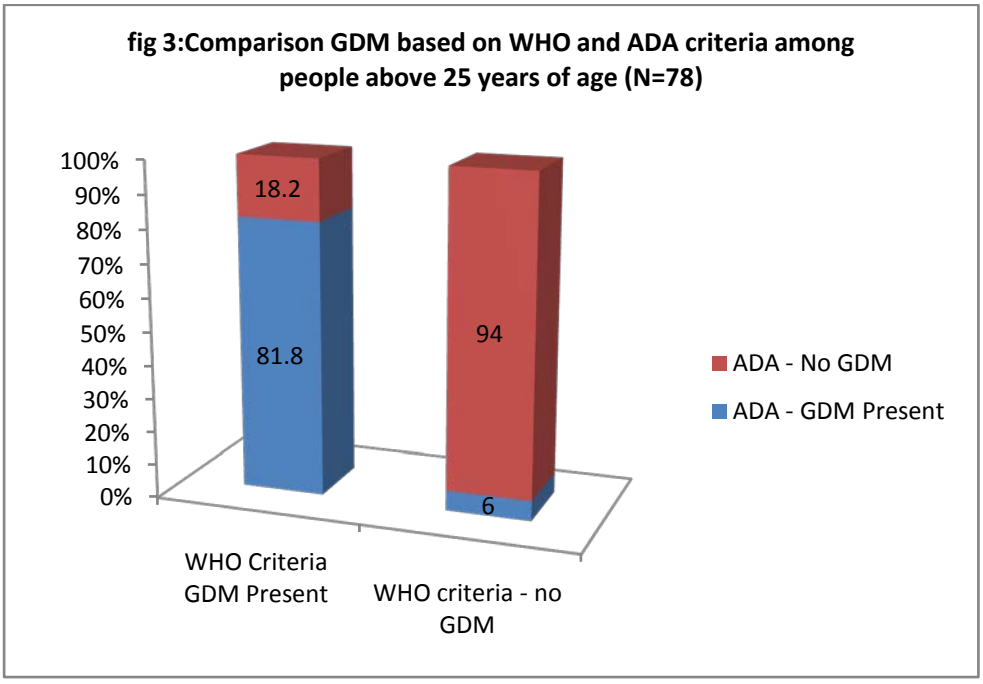
ADA CRITERIA	1 HOUR VALUE ALONE	BOTH 1HOUR AND 2 HOUR	2 HOUR VALUE ALONE
GDM PRESENT	1	12	15

GDM diagnosis by the WHO and ADA criteria in the high risk group(age above 25 years):

There was no significant difference in diagnosis of GDM in patients above the age of 25 years.

Table 6. Comparison GDM based on WHO and ADA criteria among people above 25 years of age (N=78):

ADA Criteria	WHO Criteria GDM Present	WHO criteria - No GDM	Chi square	P value
GDM Present	9 (81.8)	4 (6.0)	0.000	1.000
No GDM	2 (18.2)	63 (94.0)		
Total	11 (14.1)	67 (85.9)		



GDM diagnosis by the WHO and ADA criteria in the high risk group(previous history of GDM):

Of the patients who had conceived previously, 2 patients had history of gestational diabetes

Table 7. previous history of gestational diabetes mellitus:

Previous history of gestational diabetes mellitus	Total number of participants N (%)
Present	2 (1)
Absent	198(99)
Total	200

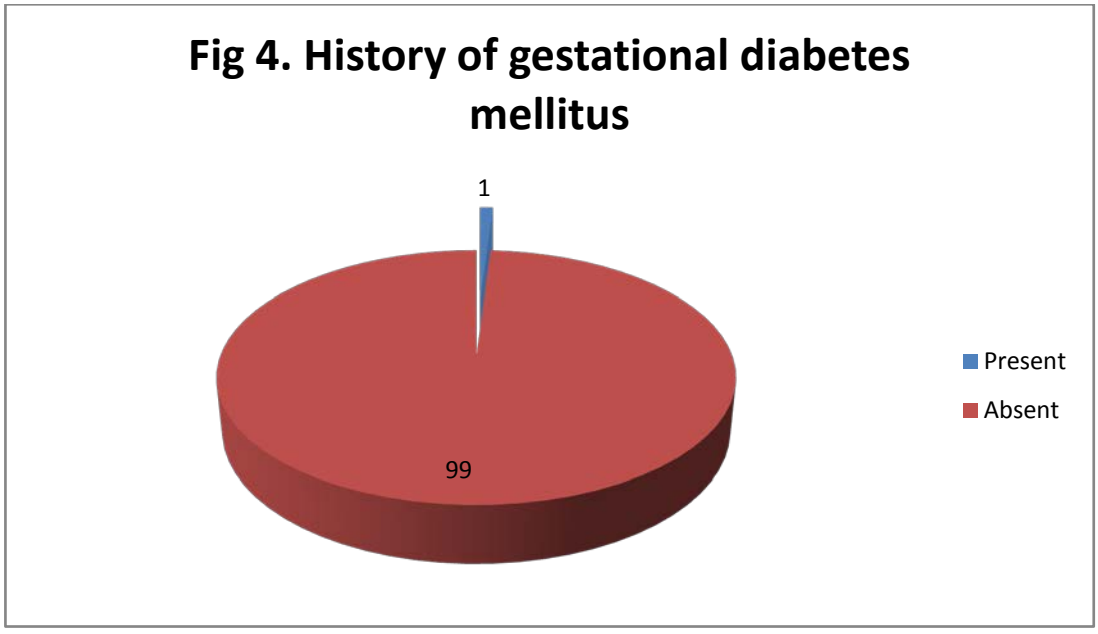
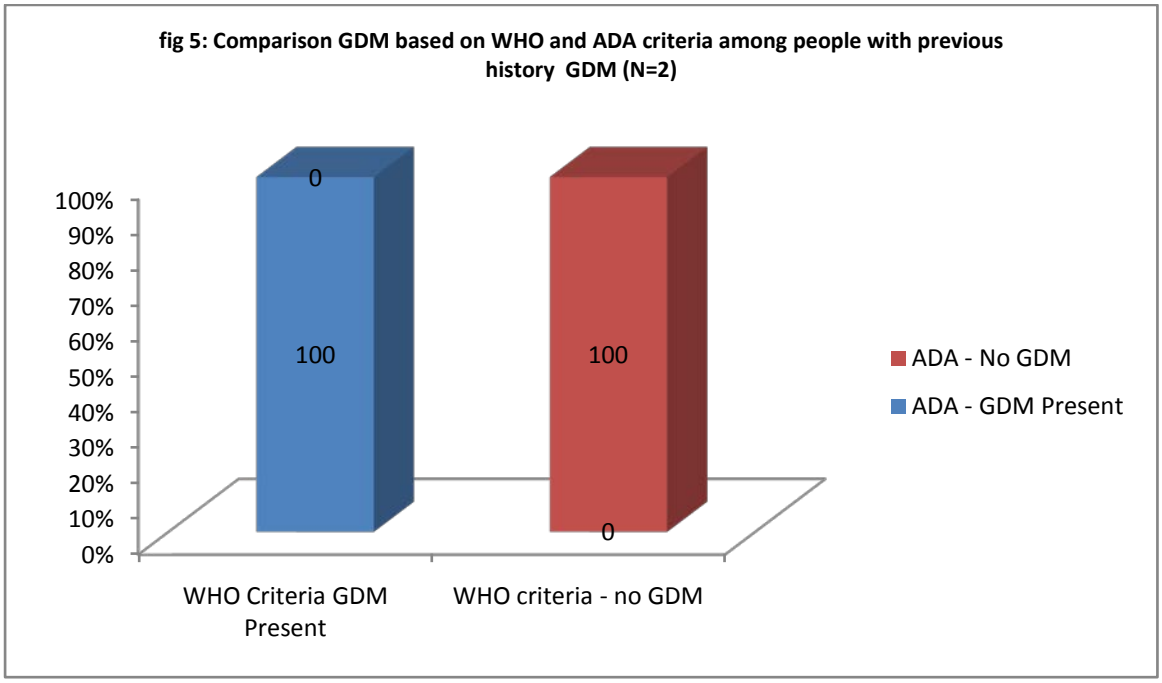


Table 8. Comparison GDM based on WHO and ADA criteria among people with previous history GDM (N=2):

ADA Criteria	WHO Criteria GDM Present	WHO criteria - No GDM	Chi square	P value
GDM Present	1 (100)	0 (0)	0.000	1.000
No GDM	0 (0)	1 (100)		
Total	1 (50)	1 (50)		



GDM diagnosis by the WHO and ADA criteria in the high risk group(thyroid disorder):

Table 9. Thyroid disorders among study participants:

In the study population , 4 people found to have thyroid dysfunction. Of this no patients had GDM.

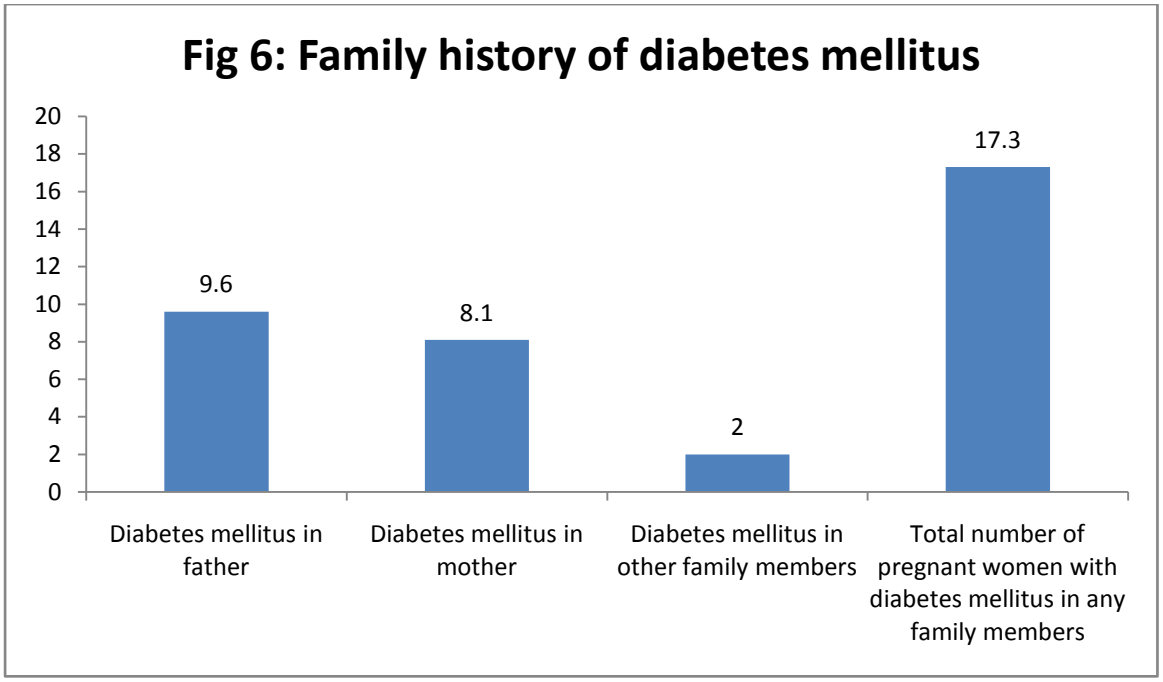
Thyroid disorders	Total number of participants N (%)
Present	4 (2)
Absent	196 (98)
Total	200

GDM diagnosis by the WHO and ADA criteria in the high risk group (family history of diabetes mellitus):

Regarding the family history of diabetes , 30 participants had history of diabetes in first degree relatives and 4 had history of diabetes in other family members.

Table 10. Family history of diabetes mellitus:

Family history of diabetes mellitus	Total number of participants N (%)
Diabetes mellitus in father	19 (9.6)
Diabetes mellitus in mother	16 (8.1)
Diabetes mellitus in other family members	4 (2.0)
Total number of pregnant women with diabetes mellitus in any family members	34 (17.3)



Out of 34 paticipants who had family history of diabetes, 8 patients were diagnosed to have GDM in the current pregnancy. And among those, ADA and WHO criteria picked up seven patients each.

Table 11. Comparison GDM based on WHO and ADA criteria among people with family history of DM (N=34):

ADA Criteria	WHO Criteria GDM Present	WHO criteria - No GDM	Chi square	P value
GDM Present	7 (87.5)	1 (3.8)	23.8	<0.001
No GDM	1 (12.5)	25 (96.2)		
Total	8 (23.5)	26 (76.5)		

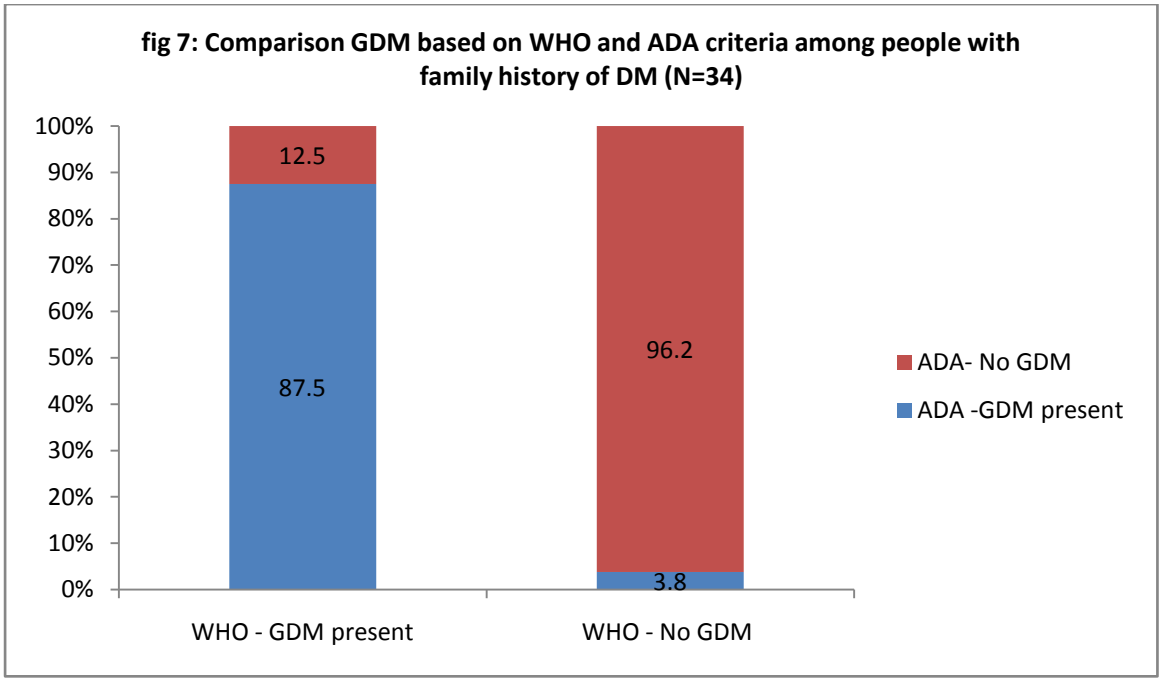


fig 7: Comparison GDM based on WHO and ADA criteria among people with family history of DM (N=34)
100%
90%
80%
70%
60%
50%
40%
30%
20%
10%
0%
12.5
87.5
96.2
3.8
WHO - GDM present
WHO - No GDM
ADA- No GDM
ADA -GDM present

GDM diagnosis by the WHO and ADA criteria in the high risk group(bad obstetric history):

Among the study participants , nine percentage of the study population had bad obstetric history.

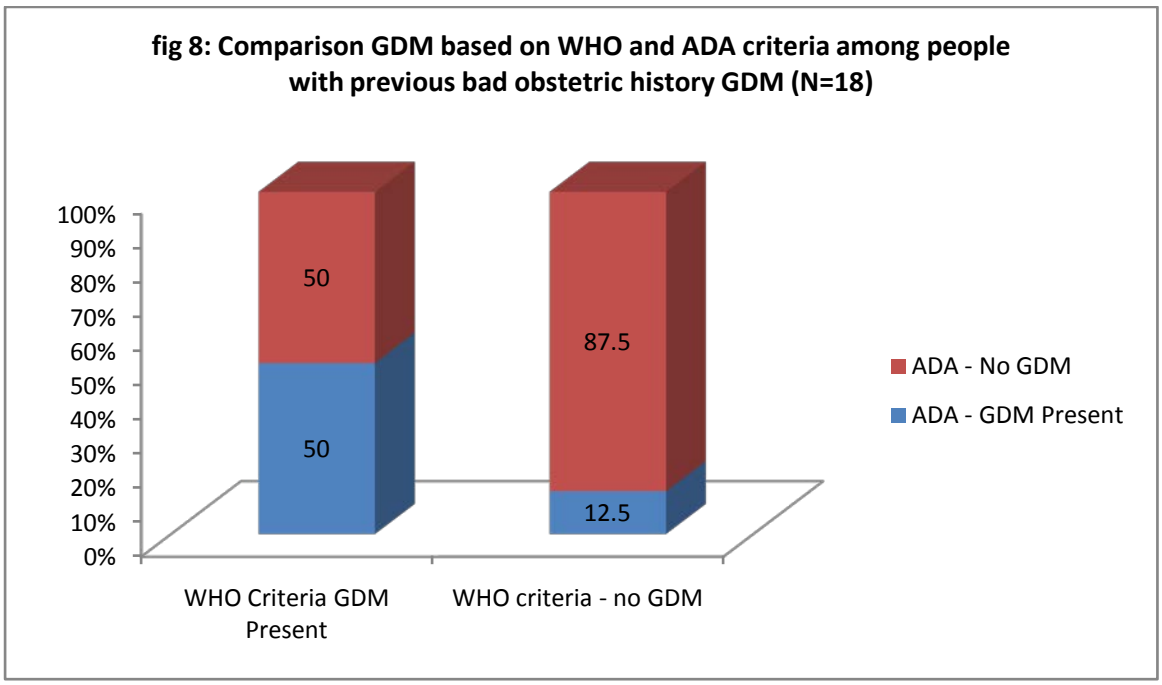
Table 12. Bad obstetric history among study participants:

Bad obstetric history	Total number of participants N (%)
Present	18 (9)
Absent	182 (91)
Total	200

Among the 18 participants who had bad obstetric history, two patients was found to have GDM as per WHO criteria and 3 patients as per ADA criteria in the current pregnancy.

Table 13. Comparison GDM based on WHO and ADA criteria among people with previous bad obstetric history GDM (N=18):

ADA Criteria	WHO Criteria GDM Present	WHO criteria - No GDM	Chi square	P value
GDM Present	1 (50)	2 (12.5)	1.80	0.180
No GDM	1 (50)	14 (87.5)		
Total	2 (11.1)	16 (88.9)		



GDM and the age of the mother:

There was no significant difference found between the age of the mother and occurrence of GDM in this study population

Table 14. Age distribution between GDM and non GDM mothers

Test group	Age	GDM present	No GDM	T test	P value
ADA	Mean (SD)	26.8 (6.1)	24.5 (4.0)	6.62	0.011
WHO	Mean (SD)	27.0 (5.3)	24.7 (5.3)	3.70	0.056

There was no significant difference found between gestational age and the occurrence of GDM.

Table 15. Gestational age distribution between GDM and non GDM mothers

Test group	Gestational age	GDM present	No GDM	T test	P value
ADA	Mean (SD)	26.4 (1.7)	26.1 (3.3)	0.50	0.479
WHO	Mean (SD)	26.4 (1.7)	26.1 (1.8)	0.43	0.513

Table 16. Sensitivity and specificity of the ADA criteria compare to Gold standard WHO criteria for diabetes mellitus:

	GDM as per WHO criteria	Non GDM as per WHO criteria	Total
GDM as per ADA criteria	21 (80.8)	7 (4.1)	28
Non GDM as per ADA criteria	5 (19.2)	167 (95.9)	172
Total	26	174	200

True positives = 26

Test positive = 21

Sensitivity = (Test positive/ True positives)*100 = 21/26=**80.8%**

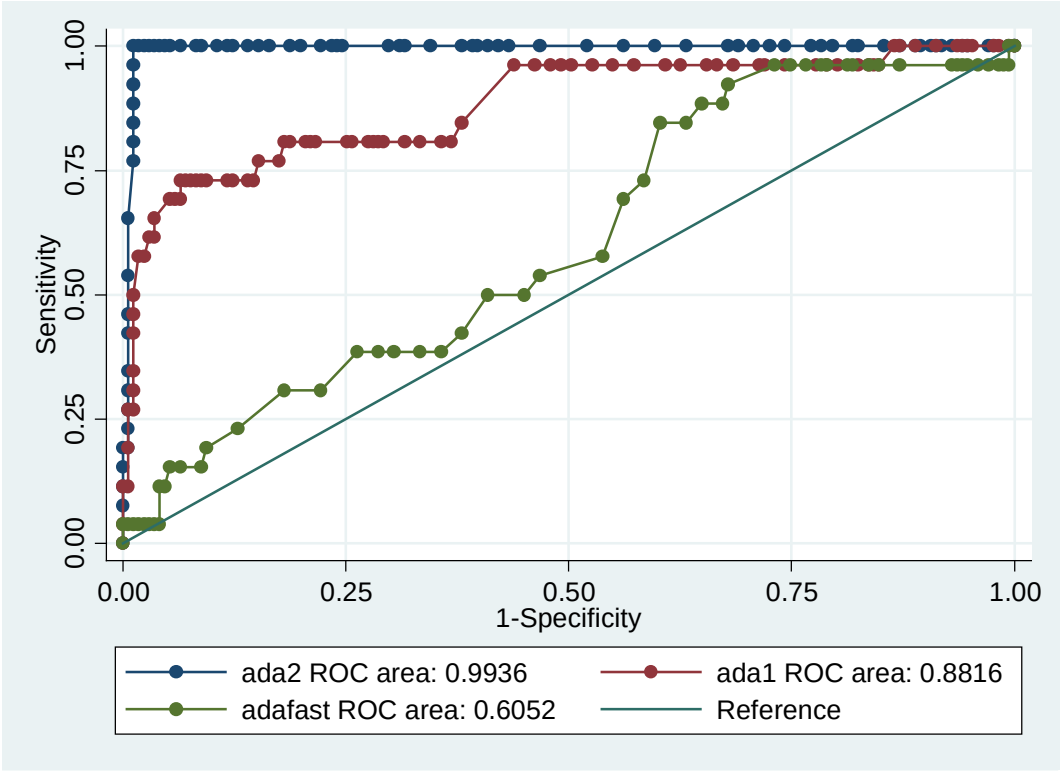
True negatives = 171

Test negatives = 167

Specificity = (Test negatives/ True negatives)*100 =164/174=**94.25**

Fig 9: ROC curve for WHO recommended and ADA recommended blood glucose testing:

When plotted for ROC curve for different cut off points in the two criterias, blood sugar value at the end of two hours had the highest sensitivity and specificity whereas the fasting blood sugar value had low sensitivity and specificity.



Data entry and analysis:

The data was entered in Microsoft excel and analysed using EpiData analysis and Stata 12.0 software. The continuous variables like age, height, weight and gestational age were expressed with mean and standard deviation. The categorical variables like age category, gravida, para, history of abortion, bad obstetric history, thyroid disorders hypertension, family history of diabetes mellitus and birth weight were expressed as proportions. The association between continuous variables like age, time duration for delivery since admission and induction of labour with method of labour management was tested using independent t test. The association between categorical variables such as age, gestational age and gestational diabetes mellitus was tested using t test. WHO criteria for GDM was used as gold standard to assess the sensitivity and specificity of the ADA criteria in diagnosis of GDM. ROC curve was obtained for 75gram OGTT, ADA fasting, ADA 1 hour and ADA 2 hour blood glucose values. The area under reach curve was compared. The association categorical variables such as WHO and ADA identified GDM and birth weight was compared using Chi square test. The p value of <0.05 was considered for statistical significance.

ADA Vs WHO criteria

The purpose of the study was to find out the better diagnostic criteria for diagnosing GDM in the general and high risk population.

General population:

28 patients were diagnosed to have GDM by either of the criteria. 26 were diagnosed by WHO criteria, whereas 28 by ADA criteria. When applied to general population, both WHO and ADA criterias performed equally in picking up the GDM patients and there was no statistically significant difference between the two.

High risk groups:

Sub-group analysis was made by dividing the patients depending on their risk factors like age above 25 years, bad obstetric history, previous history of GDM, family history of diabetes and associated thyroid disorders.

Age above 25 years:

78 patients were above the age of 25 years and considered high risk for developing GDM. And among these 78 patients, 13 patients were diagnosed to have GDM. Though in this study only 16.5 percent of the patients above the age of 254 were found to have GDM, age above 25

years is definitely a high risk factor for GDM. There was no statistical difference between WHO and ADA criteria in diagnosing GDM in patients above the age 25 years.

Past history of GDM:

Patients who had GDM in the previous pregnancies have a very high risk of getting GDM in the subsequent pregnancies. This should be considered by the women with history of GDM with high regard when planning for the next pregnancy. In our study,2 patients had previous history of GDM and among them one patient was found to have GDM in the current pregnancy also.

Family history of diabetes:

34 patients among the study participants had family history of diabetes mellitus. Among these 34 patients, eight patients had GDM in the current pregnancy. All these 8 patients had first degree relatives as a diabetic. So, any woman with a diabetic first degree relative, should be aware of the risk she inherits and take preventive and precautionary measures right from the day she is planning to conceive to address the other modifiable risk factors. This would bring down the chances of her to get GDM. There was no significant difference between the WHO and ADA criteria in diagnosing GDM in this group.

Bad obstetric history:

Mothers with previous history of abortions should be screened for GDM and considered high risk to avoid any mishaps in the current pregnancy because, literature have shown that patients with bad obstetric history have a higher chances of becoming GDM mothers compared to the general population. In our study nine patients were found to have bad obstetric history. Among these patients 4 were found to have GDM in the current pregnancy which goes by the literature. There was no significant difference between the WHO and ADA criteria in diagnosing GDM in this group.

Gestational age:

Mean age of diagnosis for GDM by both ADA and WHO criteria was found to be 26.1 weeks.

Reliability of ADA criteria:

When compared with the gold standard WHO criteria, ADA criteria had sensitivity of 80.8% and specificity of 94.25%.

When plotted for ROC curve for different cut off points in the two criterias, blood sugar value at the end of two hours had the highest sensitivity and specificity whereas the fasting blood sugar value had low sensitivity and specificity.

From all these observations two points stand out from the others.

1. **Need for universal screening – must:**

   From the data presented above, it is very clear that at least half the patients would have been missed from the diagnosis of GDM, if only the patients with high risk were to be screened. By universal screening as followed in this study the chances of picking up the GDM mothers would be doubled. This is more appropriate for the high risk ethnic group like us, Asians, suggested by literature.

2. **Resource poor setting:**

   It would be cumbersome in resource poor settings to take two sugar levels as per WHO criteria and 3 sugar levels as per ADA criteria in resource poor settings like remote and underdeveloped areas. This study has shown that two hour sugar value has maximum sensitivity and specificity in diagnosing GDM and would pick up majority of the cases when performed between 24 and 28 weeks of gestation. Though this can't replace the gold standard criteria, this can be seriously considered as the investigation of choice in resource poor settings. Similar results have been quoted in another study by Haritha Sagili et al[41]

3. Two hour glucose level:

As per WHO criteria the cut off at the end of two hours for diagnosing GDM is 140mg/dl and that by ADA criteria is 153mg/dl. When this single value is used for diagnosing GDM in our study, 28 patients would have been labeled as GDM as per WHO criteria against 23 patients as per ADA criteria. All these 28 patients are labeled as GDM by either standard ADA or WHO criteria definitions. So, when a single value of two hour blood glucose level is to be decided, it would be desirable to have a cut off as 140mg/dl.

CONCLUSION:

- There is no difference among the two recommendations both ADA and WHO in the diagnosis of GDM.
- The 2 hour value done has the statistical significance in diagnosis of GDM.
- The one hour value done in ADA criteria doesnot have statistical significance when compared to two hour value.
- Risk factor analysis doenot have any statistical significance in diagnosis of GDM.
- Hence universal screening for GDM is necessary to diagnose GDM.

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PATIENT CONSENT FORM

STUDY TITLE : A COMPARATIVE STUDY OF ADA & WHO CRITERIA FOR SCREENING OF GESTATIONAL DIABETES MELLITUS AND FOLLOW UP OF GDM PATIENTS IN SALEM

STUDY CENTRE : Department of Obstetrics and Gynaecology, GMKMCH Salem

PARTICIPANT NAME : AGE : SEX:

I.D. NO :

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

I have been explained about the possible complications that may occur during procedure. I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving any reason.

I understand that investigator, regulatory authorities and the ethics committee will not need my permission to look at my health records both in respect to the current study and any further research that may be conducted in relation to it, even if I withdraw from the study. I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law.

I agree not to restrict the use of any data or results that arise from the study.

I hereby consent to participate in this study.

Time :

Date :

Place :

Patient'name: _____

signature / thumb impression of patient

Signature of the investigator:

Name of the investigator : _____

A STUDY ON GDM

Sl. No	Age	Ht (cm)	Wt (Kg)	BMI	Family H/o			Obese	BOH	Delivery	Hyper tension	Thyroid disorde	Previous	LMP	EDD	Gravida	PARA	Abotrions	GA	75 gm OGTT			WHO	ADA
					F	M	O													F	1Hr	2 Hr		
1	25	151	54	23.6831718	Y	x	x		Nil	Nil	Nil	on thyroid	Nil	12.10.16	19.7.16	Primi			25	160	161	162	pos	pos
2	22	149	52	23.4223684	x	x	x	x	Nil	Nil	Nil	Nil	Nil	5.10.15	12.7.16	G2		1	26	90	124	91	neg	neg
3	25	157	55	22.3132784	x	x	x		Nil	Nil	Nil	Nil	Nil	11.8.15	20.6.16	Primi	-	-	28	55	115	130	neg	neg
4	22	154	70	29.5159386	x	x	x		Nil	Nil	Nil	Nil	Nil	7.11.15	14.8.10	Primi	-	-	26	87	118	90	neg	neg
5	25	143	54	26.4071593	x	x	x		present	Nil	Nil	Nil	Nil	10.11.15	17.8.16	2	1	0	24	84	105	92	NEG	NEG
6	25	157	71	28.804414	x	x	x		present	Nil	Nil	Nil	Nil	3.10.15	10.7.16	2	1	0	26	92	90	99	NEG	NEG
7	22	162	53	20.1950922	x	x	x	Nil	Nil	Nil	Nil	Nil	Nil	17.11.15	24.8.16	Primi			26	70	101	112	NEG	NEG
8	26	152	45	19.4771468	x	x	x	-	present	-	Nil	Nil	Nil	22.11.15	19.8.16	G2	1	0	24	75	193	187	neg	POS
9	29	145	46	21.8787158	x	x	x	-	Nil	7 yrs 1st child	Pre	-	-	13.12.15	20.9.16	G3	2	1st child died	24	63	138	107	neg	NEG
10	26	133	74	41.8339081	x	y	x	+	Nil	Nil	Nil	Nil	Nil	19.10.15	26.7.16	G2	1	Nil	28	102	168	127	NEG	NEG
11	22	164	64	23.7953599	x	x	x		LBW child 8th day	Nil	Nil	Nil	Nil	14.10.15	21.7.16	G3	2	Nil	26	60	104	117	neg	NEG
12	25	186	43	12.4291826	x	x	x		Nil	Nil	Nil	Nil	Nil	12.10.15	19.7.16	Primi			24	60	104	117	neg	NEG
13	25	158	69	27.6398013	x	x	sirtim		-	-	Nil	Nil	Nil	5.10.15	12.7.16	G2	1	Nil	25	56	149	132	neg	NEG
14	21	181	59	18.0092183	x	x	x		-	-	Nil	Nil	Nil	12.12.15	19.9.16	G2	1	Nil	26	60	102	118	neg	NEG

15	35	152	79	34.1932133	x	x	x	-	Nil	Yes 3.7 kg 2nd child	Nil	Nil	Nil	24.12.15	31.9.16	G3	2	Nil	26	49	68	98	neg	NEG
16	19	154	52	21.9261258	Nil	Nil	NIl	Nil	Nil	Nil	Nil	Nil	Nil	1.11.15	8.8.15	primi	0	0	24	51	155	127	neg	NEG
17	19	165	80	29.3847567	+	-	-	-	-	-	+	Nil	Nil	30.11.15	6.9.16	Primi			24	68	131	80	neg	NEG
18	27	162	60	22.8623685	Nil	Nil	NIl	Nil	-	-	Nil	Nil	Nil	11.9.15	12.6.16	Primi			28	81	120	131	NEG	NEG
19	21	168	75	26.5731293	Nil	Nil	NIl	Nil	+	1st child 4 kg	Nil	Nil	Nil	19.11.15	26.8.16	G3	1	1	28	52	138	101	NEG	NEG
20	24	152	49	21.2084488	Nil	Nil	NIl	Nil	-		Nil	Nil	Nil	25.11.15	2.9.16	Primi			28	60	120	102	NEG	NEG
21	24	152	47	20.3427978	Nil	Nil	NIl	Nil	Nil	-	-	-	-	8.12.15	15.9.16	2	1	1	26	76	137	150	POS	NEG
22	20	156	44	18.0802104	Nil	Nil	NIl	Nil	Nil	Nil	Nil	Nil	Nil	18.10.15	25.7.16	Primi	-	-	28	71	112	90	neg	neg
23	24	150	57	25.3333333	Nil	Nil	NIl	Nil	Nil	Nil	Nil	Nil	Nil	11.10.18	18.7.16	Primi			28	65	110	98	neg	neg
24	23	147	43	19.8991161	Nil	Nil	NIl	Nil	Nil	Nil	Nil	Nil	Nil	7.10.16	14.7.16	3	2	1	27	54	96	105	neg	neg
25	31	151	64	28.0689443	-	Nil	NIl	Nil	x	Nil	Nil	Nil	Nil	11.12.16	18.9.16	G3	1	1	24	104	123	119	neg	neg
26	20	153	53	22.6408646		nil	nil	nil	-	-	-	-	-	20.10.15	27.7.16	Primi			28	70	92	142	pos	neg
27	25	160	52	20.3125	Nil	x	NIl	-	-	2	-	-	-	19.10.16	26.7.16	G4	3	1	28	72	132	109	neg	neg
28	20	157	48	19.4734066	-	-	-	-	-	Primi	-	-	-	18.11.15	25.8.16	Primi			26	88	110	99	neg	neg
29	20	148	44	20.0876552	-	-	-	-	-	Nil	-	-	-	10.10.15	17.7.16	G2	1	-	26	83	89	68	neg	neg
30	22	148	44	20.0876552	-	-	-	-	-	3.25	-	-	-	10.12.15	17.9.16	G2	1	-	24	70	140	98	neg	neg

31	28	147	46	21.2874265	-	-	-	-	2	-	-	-	-	24.11.15	31.9.16	G4	1	2	24	57	132	72	neg	neg
32	21	158	63	25.2363403	-	-	-	-	2	3.8	-	-	-	8.12.15	15.9.16	G4	1	2	24	55	110	63	neg	neg
33	19	154	52	21.9261258	-	-	-	-	-	-	-	-	-	1.11.15	8.8.16	Primi			26	63	138	107	neg	neg
34	23	150	49	21.7777778	-	-	-	-	-		-	-	-	21.10.15	28.7.16	G2	P1	-	26	84	136	121	neg	neg
35	25	151	57	24.9989036	Y	-	-	-	present	1st child 3.5 kg	-	-	-	4.11.15	11.8.16	G4	P1	A2	24	88	132	117	NEG	NEG
36	21	143	37	18.0937943	Nil	Nil	NIl	Nil	Nil	Nil	-	-	-	22.12.15	28.9.16	Primi	-	-	24	92	170	122	NEG	neg
37	28	155	56	23.3090531		-	-	-	-	-	-	-	-	5.11.15	12.8.16	G3	1	1	26	87	100	120	neg	NEG
38	35	145	52	24.7324614	nil	-	-	-	present	4th child 3.75kg	-	-	-	1.12.15	8.9.16	G5	1	3	24	54	81	90	neg	NEG
39	23	159	58	22.9421305	-	-	-	-	-	3.3kg	-	-	-	16.11.15	23.8.16	G2	1	-	24	78	141	135	neg	NEG
40	28	165	60	22.0385675	-	-	-	-	-	3.7kg 1st chid	-	-	-	21.12.15	28.9.16	G3	1	1	24	70	135	112	neg	NEG
41	40	152	63	27.2680055	-	-	-	+	present[iud]	Nil	-	-	-	3.1.16	30.10.16	G3	2	-	24	81	195	210	POS	POS
42	28	159	50	19.7776987	-	-	-	-	-	-	-	-	-	4.12.15	11.9.16	G4	3	-	24	84	159	139	neg	NEG
43	21	156	55	22.600263	-	-	-	-	nil	1st child 3.5 kg	-	-	-	26.11.15	3.9.16	G3	1	1	28	70	97	74	neg	NEG
44	23	160	52	20.3125	-	-	-	-	-	-	-	-	-	11.11.15	18.8.16	Primi			26	97	103	85	neg	NEG
45	30	148	51	23.2834186	-	-	-	-	-	3.75kg	-	-	-	1.12.15	8.9.16	G2	1	-	26	98	199	159	POS	pos
46	32	155	76	31.6337149	~~	~	~	~	`~	~	-	-	-	21.11.15	28.8.16	G2	1	-	24	62	101	128	neg	NEG

47	25	140	35	17.8571429	-	-	-	-	~	-	-	-	-	15.12.15	22.9.16	G2	0	1	25	60	127	98	neg	NEG
48	27	147	65	30.0800592	-	-	-	-	Nil	-	-	Present	-	1.11.15	8.8.16	G2	1	-	26	70	135	112	neg	NEG
49	26	153	40	17.087445	-	-	-	-	-	-	-	-	-	15.1.16	22.10.16	G2	1		26	73	132	103	neg	NEG
50	31	143	68	33.2534598	-	-	-	+	1	-	-	-	-	14.12.16	21.9.16	3	1	1	24	65	120	77	neg	NEG
51	21	152	59	25.5367036	-	-	-	-	1		-	-	-	22.11.15	29.8.16	3	1	1	24	65	104	81	neg	Neg
52	28	152	53	22.9397507	-	-	-	-	-		-	-	-	17.11.15	24.8.16	3	2	-		80	110	103	NEG	NEG
53	31	158	40	16.0230732	-	-	-	-	-		-	-	-	28.12.15	5.10.16	2	1	-	26	78	109	94	NEG	NEG
54	32	156	75	30.8185404	~	~	~	~	~	4 KG	~	~	~	13.12.15	20.09.16	5	1	3	26	73	172	149	pos	NEG
55	23	153	41	17.5146311	~	~	~	~	~	~	~	ˋ~	~	30.12.15	07.06.16	primi			28	78	116	93	neg	NEG
56	25	155	55	22.89282	~	present	~	ˋ~	~	~	~	~	`	24.12.15	1.10.16	primi			2	76	157	108	neg	NEG
57	38	150	48	21.3333333	~	present	~	~	~	~	~	~	~	1.1.16	8.10.16	primi			28	91	174	169	pos	pos
58	25	160	67	26.171875	present	present	~	~	1	3.6	s	~	~	4.1.16	11.10.16	3	1	1	24	78	122	108	neg	NEG
59	20	163	50	18.8189243	~	~	~	~	~	ˋ~	~	ˋ~	~	19.12.15	26.9.16	primi			24	78	99	73	neg	NEG
60	26	144	37	17.8433642	~	~	~	~	~	3.2	~	~	~	26.12.15	3.10.16	3	2		26	60	108	116	neg	NEG
61	28	148	69	31.5010957									+	12.10.15	19.7.16	2	1		28	74	137	121	neg	NEG
62	72	148	49	22.3703433	~	present	~	~	~	~	~	ˋ~	~	5.2.16	12.11.16	primi			24	72	143	130	neg	NEG

63	29	155	69	28.7200832	`~	present	~	~	~	`~	~	`~	~	8.1.16	15.10.16	primi			24	64	138	108	neg	NEG
64	20	159	44	17.4043748	present	present	present		1					6.1.16	13.10.16	2		1	24	60	105	71	neg	Neg
65	26	136	39	21.0856401	`~	~	~			2.6	~	~	~	15.11.16	27.10.16	2	1		24	79	125	101	neg	Neg
66	23	162	70	26.6727633	~	~	~		+	3.15	~	~	~`	26.12.16	4.10.16	3	1	1	26	89	110	123	neg	Neg
67	21	154	73	30.7809074	~	present	~	~	~	~	~	~	`~	20.11.15	27.8.16	primi			28	60	92	79	neg	Neg
68	21	142	52	25.788534	~	+	~	~	`~	~	~	~	~	not known	scan wise 8.11.16	2	1		28	79	120	134	neg	Neg
69	20	149	56	25.224089	~	~	~	~	~	`~	~	~	`~	2.1.16	9.10.16	primi			28	69	97	87	neg	Neg
70	23	156	46	18.9020381	`~	~	~	~		2.6	~	~	~	not known	2.10.16scan wise	2	1		26	80	128	99	neg	Neg
71	21	150	60	26.6666667	~	+	~	~	~	2.8	~	~	~	12.2.16	19.11.16	2	1		24	66	118	86	neg	Neg
72	23	151	60	26.3146353	~	~	~	~	~	2.7	~	~	~	19.12.15	26.9.16	2	1		24	94	164	86	neg	Neg
73	23	148	56	25.5661066	~	~	~	~	~	3	~	~	~	20.1.16	27.10.16	2	1		26	74	82	96	neg	Neg
74	22	135	48	26.3374486	present	~	~	~	~	~	~	~	~	19.12.15	26.9.16	primi			28	62	77	70	neg	Neg
75	27	133	44	24.8742156	~	~	~	~	~	3	~	~	~	21.1.16	28.10.16	2	1		24	90	121	159	pos	pos
76	24	150	58	25.7777778	present	~	~	+	~	~	~	~	~	30.12.15	.7.10.16	primi			28	95	120	110	neg	Neg
77	23	154	56	23.6127509	present	~	~	~~	~	3.6	~	~	~	24.1.16	1.11.16	3	1	1	26	75	139	155	pos	pos
78	29	150	60	26.6666667	~	~	~	~	~	~	~	~	~	2.12.15	9.9.16	3	1	1	24	68	113	102	neg	Neg

79	22	154	50	21.0828133	~	~	~	~	~	~	~	~	~	10.12.15	17.9.16	primi			24	79	125	101	NEG	NEG
80	23	165	86	31.5886134	+	~	~	+	~	~	~	~	~	9.2.16	16.11.16	primi			26	87	160	128	NEG	NEG
81	25	156	55	22.600263	~	~	~	~	~	~	~	~	~	11.11.15	18.8.16	primi			24	61	104	64	NEG	NEG
82	22	152	49	21.2084488	present	~	~	~	~	4.05	~	~	~	20.12.15	22.8.16	2	1		25	78	119	101	NEG	NEG
83	20	149	59	26.5753795	~	~	~	~	~	~	~	~	~	23.11.15	30.8.16	primi			25	77	109	81	NEG	NEG
84	20	155	58	24.1415193	~	~	~	~	~	~	~	~	~	7.2.16	14.11.16	primi			24	82	143	99	NEG	NEG
85	26	157	58	23.5303663	~	present	~	~	~	~	~	~	~~	23.1.16	30.10.16	primi			26	69	98	112	NEG	NEG
86	26	155	62	25.8064516	~	~	~	~	~	2.45	~	~	~	17.1.16	24.10.16	2	1		28	60	108	116	NEG	NEG
87	19	150	70	31.1111111	~	~	GM	~	~	~	~	~	~	18.11.15	25.8.16	primi			26	78	82	109	NEG	NEG
88	21	149	48	21.6206477	~	~	~	~	~	3.5	~	~	~	23.12.15	30.9.16	2	1		24	70	107	96	NEG	NEG
89	24	149	59	26.5753795	present	present	~	~	~	3.7	~	~	~	18.1.16	25.10.16	2	1		25	59	198	133	NEG	POS
90	27	155	56	23.3090531	~	~	~	~	~	3.75	~	~	~	26.1.16	3.11.16	2	1		24	67	101	118	NEG	NEG
91	36	152	60	25.9695291	present	~	~	~	~	3.75	~	~	~	21.1.16	28.2	2	1		25	75	120	165	POS	POS
92	20	165	48	17.630854	~	~	~	~	~	~	~	~	~			primi			24	78	112	108	NEG	NEG
93	20	160	55	21.484375	~	~	~	~	~	~	~	~	~	21.1.16	27.10.16	primi			24	81	112	96	NEG	NEG
94	27	143	48	23.4730305	~	~	~	~	~	3	~	~	~	22.11.15	28.8.16	3	2		26	80	115	122	NEG	NEG
95	26	152	53	22.9397507	~	~	~	~	~	2.8	~	~	~	18.2.16	25.11.16	3	2		25	74	124	70	NEG	NEG
96	23	150	58	25.7777778	~	~	~	~	~	~	~	~	~	21.2.16	27.10.16				28	72	88	82	NEG	NEG
97	23	151	60	26.3146353	~	~	~	~	~	~	~	~	~`	20.2.16	27.10.16	Primi			28	70	125	73	neg	NEG
98	20	150	62	27.5555556	~	~	~	~	~	~	~	~	`~	25.2.16	2.12.16	primi			26	61	86	64	NEG	NEG
99	26	164	78	29.0005949	~	~	~	~	~	~	~	~	~	31.12.15	1.10.16	2		1	28	76	83	72	neg	neg
100	25	154	68	28.6726261	~	~	~	`~	~	~	~	~	~	16.2.16	23.11.16	primi			24	76	141	96	neg	neg
101	23	165	86	31.5886134	Y	-	-	+	-	-	-	If not on th	-	NK	NK	primi			28	87	160	128	NEG	NEG
102	25	156	55	22.600263	-	-	-	-	-	-	-	-	-	11.11.15	18.8.16	Primi			28	61	104	64	NEG	NEG
103	25	152	40	17.3130194	-	-	-	-	-	-	-	-	-	8.1.16	15.10.16	2	1	-	26	80	120	90	neg	NEG
104	22	152	49	21.2084488	-	-	-	-	-	4.05k	-	-	-	20.12.15	27.9.16	2	1	-	24	78	119	101	NEG	NEG
105	20	149	59	26.5753795	-	-	-	-	-	-	-	-	-	23.11.15	30.8.16	Primi		-	28	90	129	113	NEG	NEG
106	20	155	58	24.1415193	-	-	-	-	-	-	-	-	-	7.2.16	14.11.16	Primi			24	82	143	99	NEG	NEG
107	26	157	58	23.5303663	-	-	-	-	-	-	-	-	-	23.1.16	30.10.16	Primi			28	69	98	112	NEG	NEG
108	26	155	62	25.8064516	-	-	-	-		-	-	-	-	17.1.16	24.10.16	2	1		28	87	113	102	NEG	NEG
109	19	150	70	31.1111111	-	-	Grand mother	-	-	-	-	-	-	18.11.15	25.8.16	Primi			26	95	170	156	POS	POS
110	30	149	50	22.521508	-	-	-	-	-	3.5kg	-	-	-	24.11.15	31.8.16	3	2		28	88	122	109	NEG	NEG
111	22	160	46	17.96875	-	-	-	-	-	-	-	-	-	14.1.16	21.10.16	Primi			28	89	180	160	POS	POS
112	28	156	66	27.1203156	-	-	-	-	-	-	-	-	-	18.1.16	25.10.16	2	1		24	87	160	160	pos	POS

113	21	149	48	21.6206477	-	-	-	-	-	3.5 kg	-	-	-	23.12.15	30.9.16	2	1		28	70	107	96	NEG	NEG
114	24	149	59	26.5753795	-	-	-	-	-	3.7 kg	-	-	-	18.1.16	25.10.16	2	1		28	78	196	188	POS	POS
115	28	154	63	26.5643447	-	-	-	-	-	4 kg	-	-	-	14.2.16	21.11.16	2	1		28	68	101	90	NEG	NEG
116	20	151	50 kg	#VALUE!	-	-	-	-	-	-	-	-	-	11.1.16	18.10.16	primi			28	81	195	210	pos	POS
117	23	158	44	17.6253805	-	-	-	-	-	-	-	-	-	1.12.2016	7.9.2016	2	1	-	24	88	120	101	NEG	NEG
118	27	195	56	14.7271532	-	-	-	-	-	3.75 kg	-	-	-	26.1.16	3.11.16	2	1	-	26	85	108	97	NEG	NEG
119	28	155	52	21.6441207	-	-	-	-	-	-	-	-	-	24.1.16	31.10.16	Primi	-	-	26	89	143	130	NEG	NEG
120	36	152	60	25.9695291	Y	-	-	-	-	-	-	-	+	21.1.16	28.10.16	2	1	-	28	75	120	165	POS	POS
121	20	165	48	17.630854	-	-	-	-	-	-	-	-	-			Primi			24	78	90	108	NEG	NEG
122	20	160	55	21.484375	-	-	-	-	-	-	-	-	-	4.2.16	11.11.16	Primi	-	-	24	80	89	106	NEG	NEG
123	20	165	69	25.3443526	-	-	-	-	-	-	-	-	-	20.1.16	27.10.16	Primi			26	81	88	96	NEG	NEG
124	27	143	48	23.4730305	-	-	-	-	-	-	-	-	-	21.11.15	28.8.16	3	2	-	26	80	77	122	NEG	NEG
125	21		45	#DIV/0!	-	-	-	-	-	-	-	-	-	16.12.15	23.9.16	2	1	-	26	90	88	97	NEG	NEG
126	27	152	63	27.2680055	-	-	-	-			Y	-	-	7.12.15	14.9.16	2	1	-	28	103	82	109	NEG	NEG
127	20	148	43	19.6311176	-	-	-	-	-	-	-	-	-	20.1.16	27.10.16	Primi		Nil	28	116	125	172	NEG	POS
128	27	153	46	19.6505617	-	-	-	-	-	-	-	-	-	13.1.16	20.10.16	Primi		-	28	114	123	166	NEG	POS
129	26	152	53	22.9397507	-	-	-	-	-	-	-	-	-	18.2.16	25.11.16	3	2		28	74	124	70	NEG	NEG
130	20	158	47	18.827111	-	-	-	-	-	-	-	-	-	18.2.16	25.11.16	Primi			24	47	192	180	POS	POS
131	20	152	45	19.4771468	-	-	-			Primi				10.12.15	17.9.16			-	36	61	99	94	NEG	NEG
132	29	147	40	18.5108057	-	+	-				-	-	-	16.2.16	28.11.16	93	Po	A2	24	70	98	97	NEG	NEG
133	23	150	58	25.7777778	-	-	-				-	-	-	21.02.16	27.10.16			-	26	72	88	82	NEG	NEG
134	26	164	78	29.0005949	Y	-	-	-	-		-	-	-	31.12.15	7.10.16	Primi	-	-	28	101	123	112	NEG	NEG
135	25	152	53	22.9397507	-	-	-	-	-		-	-	-	4.2.16	11.11.16	2	-	-	28	89	111	101	NEG	NEG
136	19	158	53	21.230572	-	-	-	-	-	-	-	-	-	23.1.16	30.10.16	Primi	-	-	26	63	82	78	NEG	NEG
137	19	153	47	20.0777479	-	-	-	-	-	-	-	-	-	24.2.16	1.12.16	Primi	-	-	24	89	120	93	NEG	NEG
138	19	159	50	19.7776987	-	-	-	-	-	-	-	-	-	2.2.16	7.11.16	Primi	-	-	28	73	133	104	NEG	NEG
139	30	147	75	34.7077607										14.2.16	21.11.16	primi			28	77	100	97	neg	neg
140	26	159	57	22.5465765										12.3.16	19.12.16	primi			26	86	111	101	NEG	NEG
141	27	15	62	2755.55556										19.2.16	26.11.16	primi			28	82	102	97	NEG	NEG
142	22	151	43	18.858822										4.4.16	11.11.17	primi			28	83	101	98	NEG	NEG
143	25	140	46	23.4693878										not known	not known	primi			24	91	119	102	NEG	NEG
144	39	144	45	21.7013889										27.1.16	5.10.16	PRIMI			28	82	170	156	POS	POS
145	26	144	46	22.183642										30.1.16	1210.16	PRIMI			28	86	134	111	NEG	NEG
146	24	154	56	23.6127509										19.12.15	26.9.15	PRIMI			26	83	123	104	NEG	NEG
147				#DIV/0!	-	-	-	-	-	-	-	-	-	05.12.15	12.9.16	primi			28	75	163	155	NEG	POS
148	32	144	68	32.7932099	Y	-	-	-	+	-	-	-	-	15.12.15	22.9.16	3	1	1	28	77	120	146	pos	NEG
149	31	141	54	27.1616116										26.1.16	2.11.16	PRIMI			28	79	273	207	pos	POS
150	20	143	43	21.0279231										NK	NK	PRIMI			26	67	116	101	NEG	NEG
151	22	150	54	24										2.11.15	8.8.16	PRIMI			28	87	121	114	NEG	NEG

152	21	156	70	28.7639711										2.2.16	9.11.16	PRIMI			24	77	156	145	pos	NEG
153	39	150	58	25.7777778										17.3.16	24.12.16	PRIMI			24	89	121	117	NEG	NEG
154	22	149	51	22.9719382	Y									25.12.15	1.10.16	PRIMI			24	88	130	99	NEG	NEG
155	22	163	61	22.9590877	Y									25.11.15	2.9.16	3	2		26	90	120	112	NEG	NEG
156	28	151	45	19.7359765										1.3.16	8.12.16	2	1		26	85	152	86	NEG	NEG
157	21	162	53	20.1950922	-	Y	-	-	-	-	-	-	-	10.2.16	17.11.16	Primi	-	-	28	82	92	80	NEG	NEG
158	23	144	40	19.2901235	-	-	-	-	-		-	-	-	Not known	25.11.16	G2	1	-	28	87	175	166	POS	POS
159	27	151	61	26.7532126	-	-	-	-	-		-	-	-	Not known	6.9.16	3	1	1	24	88	111	96	NEG	NEG
160	20	150	50	22.2222222	Y	Y	-	-	-	-	-	-	-	20.12.15	27.9.16	Primi			28	86	113	93	NEG	NEG
161	30	148	56	25.5661066	-	-	-	-	1	-	-	-	-	23.12.15	30.9.16	4	2	1	28	89	144	136	NEG	NEG
162	21	156	70	28.7639711	-	-	-	-	-	-	-	-	-	2.2.16	9.11.16	Primi			26	65	132	78	NEG	NEG
163	24	163	56	21.0771952	-	-	-		Nil	Nil	-	-	-	22.11.15	29.8.16	Primi			26	20	102	98	NEG	NEG
164	28	159	58	22.9421305	-	-	-		+	-	-	-	-	16.2.16	23.11.16	G4	P2	1	28	94	166	153	neg	POS
165	20	154	66	27.8293135	-	-	-			-	-	-	-	1.1.16	8.10.16	Primi		0	24	85	109	96	NEG	NEG
166	27	150	42	18.6666667	-	-	-		+	-	-	-	-	17.1.16	24.10.16	G5	-	4	24	87	118	102	NEG	NEG
167	22			#DIV/0!	-	-	-	-	-	3.8 kg	-	-	-	14.01.16	21.10.16	2	-	-	28	67	130	105	NEG	NEG
168	28	145	65	30.9155767	-	-	-			3.5 kg	-	-	-	2.2.16	9.1.16	2	-	-	26	75	132	112	NEG	NEG
169	28	150	38	16.8888889	-	-	-	-		3.3 kg	-	-	-	5.2.16	12.11.16	2	-	-	24	89	160	156	NEG	POS
170	27		65	#DIV/0!	-	-	-				-	-	-	6.2.16	13.11.16	Primi			26	89	141	119	NEG	NEG
171	22	157	65	26.3702381	-	-	-	-	-	3.5 kg	-	-	-	4.1.16	11.10.16	3	1	1	28	89	196	190	POS	POS
172	25	138	39	20.478891	-	-	-	-	-		-	-	-	3.2.16	10.11.16	2	1	-	28	73	102	111	NEG	NEG
173	27	142	50	24.7966673	-	-	-				-	-	-	19.2.16	26.11.16	Primi	-	-	28	90	87	97	NEG	NEG
174	27	156	62	25.4766601	-	-	-							21.2.16	28.11.16	PRIMI			28	83	101	91	NEG	NEG
175	23	154	91	38.3707202	Y	-	-							22.3.16	29.12.16	PRIMI			28	90	89	87	NEG	NEG
176	27	151	51	22.36744	-	-	-		Y		-	-	-	4.1.16	11.10.16	2	1	2	26	86	112	88	NEG	NEG
177	30	148	55	25.109569	-	-	-	-	-	-	-	-	-	17.2.16	23.11.16	2	1	-	28	77	89	86	NEG	NEG
178	20	151	41	17.9816675	-	-	-	-	-	-	-	-	-	301.16	7.11.16	Primi	-	-	28	78	98	88	NEG	NEG
179	29	152	40	17.3130194	-	-	-	-	-	-	-	-	-	26.12.15	2.10.16	3	2	-	28	81	111	81	NEG	NEG
180	19	155	46	19.1467222	-	-	-	-	-	-	-	-	-	29.2.16	7.12.16	Primi	-	-	24	60	108	129	neg	NEG
181	27		53		-	-	-							13.3.16	20.12.16	PRIMI			26	84	142	131	NEG	NEG
182	23	164	51	18.9619274	-	-	-							11.04.16	18.1.17	PRIMI			28	89	120	92	NEG	NEG
183	23	154	56	23.6127509	-	-	-			2.5 kg				4.1.16	11.10.16	3	1	A1	24	77	164	156	POS	POS
184	28	146	45	21.1109026	-	-	-	-	-		-	-	-	20.5.16	27.2.17	2	1	1	28	78	92	119	neg	NEG
185	38	151	59	25.8760581	Y	-	-	-	-	-	-	-	-	24.2.16	30.11.16	3	2	-	28	81	135	120	neg	NEG
186	32	145	50	23.7812128	-	-	-	-	-	-	-	-	-	2.2.16	9.11.16	4	2	1	28	76	150	138	neg	NEG
187	24	154	50	21.0828133	-	-	-	-	-	-	-	-	-	8.3.16	15.12.16	2	1	-	26	71	175	167	pos	POS
188	31	151	54	23.6831718	-	-	-	-	-	-	-	-	-	17.12.16	24.11.16	3	1	1	28	92	139	82	NEG	NEG
189	21	154	57	24.0344072	-	-	-				-	-	-	8.3.16	15.12.16	primi	-	-	28	52	91	70	NEG	NEG
190	21	161	50	19.2893793	-	-	-	-	-	2.5 kg	-	-	-	20.3.15	27.12.16	2	1	-	26	92	90	99	NEG	NEG

191	24	151	68	29.8232534	-	-	-	-	-	2.75 kg		-	-	6.2.16	13.11.16	2	1	-	24	74	95	82	NEG	NEG
192	21	148	49	22.3703433	-	-	-	-	-	2.3 kg	-	-	-	29.1.16	5.11.16	2	1	-	28	84	105	92	NEG	NEG
193	29	143	43	21.0279231	-	-	-	-	-	2.5 Kg 2.25 KG	-	-	-	19.2.16	26.11.16	3	2	-	28	77	108	80	NEG	NEG
194	26	154	83	34.9974701	-	-	-	+		-	-	+	-	12.1.16	19.10.16	primi			24	80	110	82	NEG	NEG
195	31	155	75	31.2174818	-	-	-	+		-	-	-	-	17.1.16	24.10.16	4	3	2	28	88	119	91	NEG	NEG
196	22	153	40	17.087445	-	-	-	-	-		-	-	-	15.1.16	22.10.16	Primi			26	99	130	108	NEG	NEG
197	22	148	34	15.522279	-	Y	-	-	-	-	-	-	-	11.12.2015	18.9.2016	3	1	1	28	98	199	159	POS	POS
198	24	157	51	20.69049454	-	-	-	-	-	-	-	-	-	17.1.2016	24.10.2016	primi			27	96	99	108	NEG	NEG
199	23	154	54	22.76943835	-	-	-	-	-	-	-	-	-	22.1.2016	29.10.2016	primi			25	95	105	114	NEG	NEG
200	21	168	62	21.96712018	-	-	-	-		-	-	-	-	05.2.2016	12.11.2016	2	1		26	80	112	120	NEG	NEG