CORRELATION BETWEEN EXCESSIVE EARLY PREGNANCY WEIGHT GAIN & RISK OF GESTATIONAL DIABETES MELLITUS.

Submitted to

THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY

in partial fulfillment of regulations

For award of the degree of

M.S (OBSTETRICS&GYNAECOLOGY)
BRANCH – II

ESIC Medical college & PGIMSR
K.K.Nagar ,Chennai

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

APRIL 2016

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This is to certify that dissertation named **"CORRELATION BETWEEN EXCESSIVE EARLY PREGNANCY WEIGHT GAIN & RISK OF GESTATIONAL DIABETES MELLITUS"** is a bonafide work performed by **Dr.K.VASANTHA,** post graduate student, Department of Obstetrics & Gynaecology, ESIC Medical College & PGIMSR, Chennai-78, under my guidance and supervision in fulfillment of regulations of The Tamilnadu Dr. M.G.R Medical University for the award of M.S. Degree during the academic year 2013-2016

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DECLARATION

I solemnly declare that this dissertation entitled **"CORRELATION BETWEEN EXCESSIVE EARLY PREGNANCY WEIGHT GAIN & RISK OF GESTATIONAL DIABETES MELLITUS"** has been conducted by me at ESIC Medical College & PGIMSR, Chennai, under the guidance and supervision of Associate **Prof.Dr.MAYA MENON, DNB (O&G),** Head, Department of Obstetrics & Gynaecology, ESIC Medical College & PGIMSR, Chennai. This dissertation is submitted to **The Tamil Nadu Dr.M.G.R. Medical University, Chennai** in partial fulfillment of the University regulations for the award of the degree of **M.S. Branch - II (**Obstetrics & Gynaecology**)**.

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Finally, I wholeheartedly thank the mothers and children, who were the subjects of the study, without whom this would not have become a reality.

Dr.K.VASANTHA

CERTIFICATE OF APPROVAL

TO

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Dear Dr. K. Vasantha,

The Institutional Ethics Committee of ESI PGIMSR reviewed and discussed you application for approval of the proposal entitled "Correlation Between Excessive Early Pregnancy Weight Gain and Risk of Gestational Diabetes Mellitus" , No. 09/20/11/2013.

The following members of the Ethics Committee were present in the meeting held on 20.11.2013 conducted at ESI PGIMSR, KK Nagar, Chennai-78.

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3.	Prof. A.V. Srinivasan, EMERITUS Professor, TN MGR Medical University, EC Member
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12.	Dr. P. Venkatesan, Scientist, EC Member
13.	Shri. K M Venugopal, Advocate, EC Member

The proposal is approved to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and significant adverse effects occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

Saradha Suresh

[DR. SARADHA SURESH]
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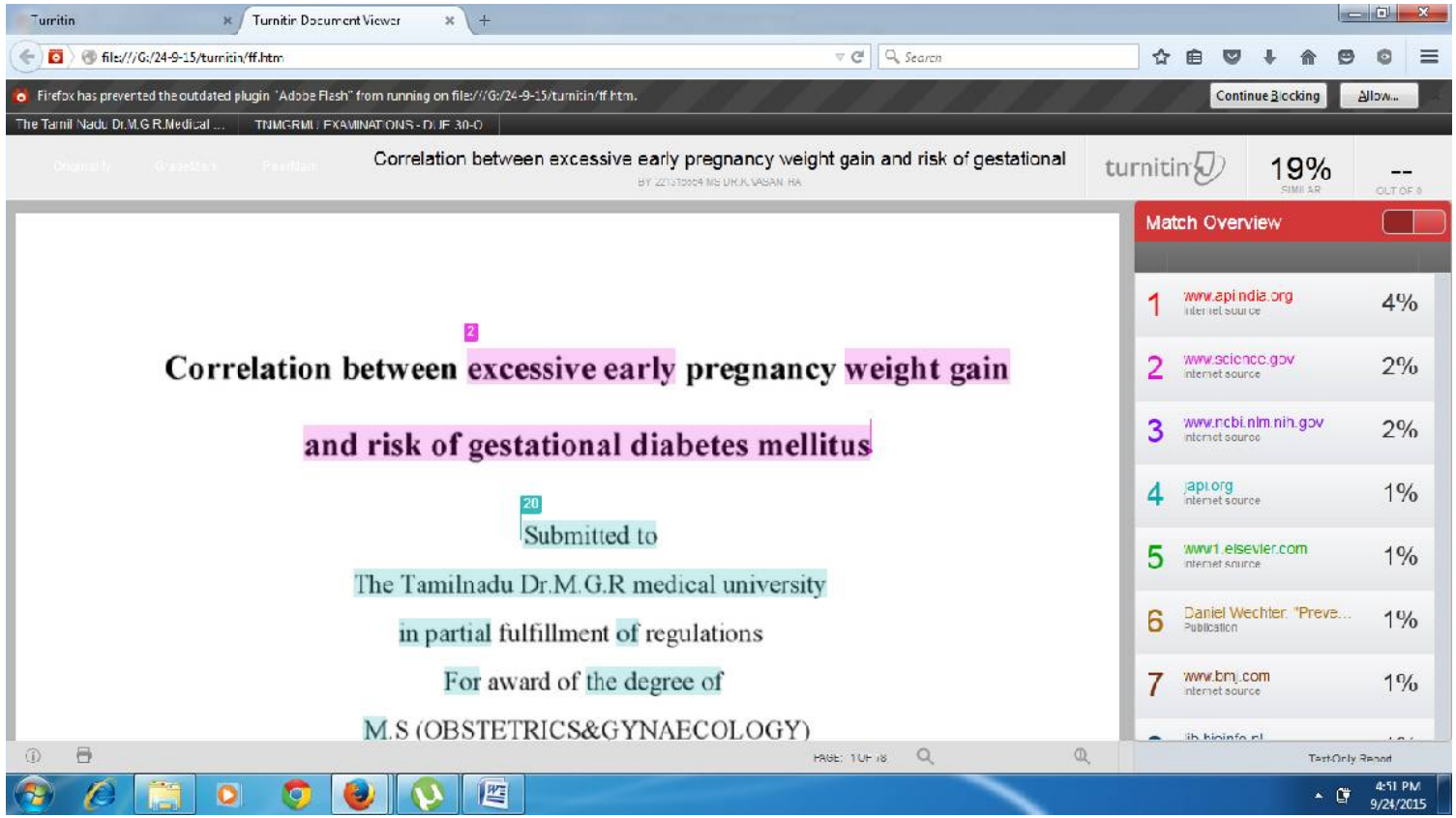
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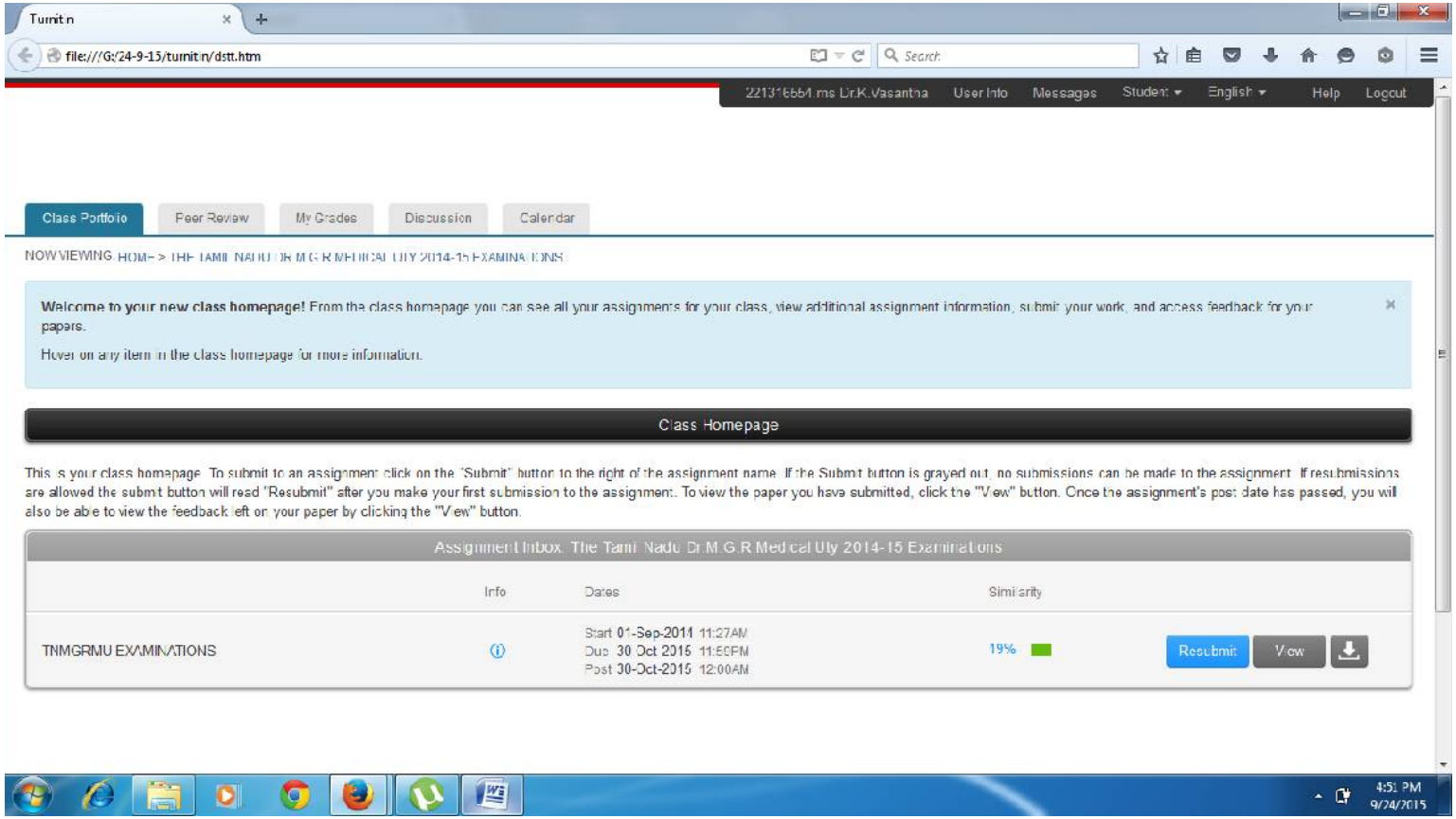
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CONTENTS

ABSTRACT

STUDY: Correlation between excessive early pregnancy weight gain and risk of gestational diabetes mellitus

GUIDE: Dr.Maya Menon., DNB (O&G)
CO GUIDE:Dr.K.Mythili.,M.D .,DNB (O&G)

Objective

To study the correlation between excessive early pregnancy weight gain and the risk of developing gestational diabetes mellitus and its complications.

Methods

This is a prospective observational study conducted in pregnant women attending antenatal outpatient Department in ESIC Medical College – PGIMSR ,K.K. Nagar,Chennai-78. Maternal weight gain from prepregnancy (self-reported) to 14 weeks of gestation was measured, and expected gestational weight gain was determined using the Institute of Medicine (IOM) 2009 guidelines. Excessive early pregnancy weight gain was defined as gestational weight gain greater than the upper range of the IOM guidelines for first trimester(>2kg). Risk of developing GDM, and its maternal and neonatal complications were estimated and compared between women with excessive early pregnancy weight gain and non excessive early pregnancy weight gain (within or below IOM guidelines).

Results

A total of 250 women were studied. 104 women developed GDM. Excessive early pregnancy weight gain occurred in 88 (35.2%) women.Out of 104 women with GDM ,62 (59.6%) women with excessive early pregnancy weight gain and 42 (40%) with normal weight gain developed GDM with significant 'p' $\leq .0001$. Out of 146 women 26 (17.8%) with excessive early pregnancy weight gain ,did not develop GDM. Risk of GDM, maternal and neonatal complications were higher in women with excessive early pregnancy weight gain especially in the first trimester.

Conclusions

Our study population showed, excessive early pregnancy weight gain especially in the first trimester is associated with risk of developing GDM, and its related maternal and neonatal complications. Excessive GWG can be represented as a modifiable risk factor.Lifestyle and dietary modifications can prevent GDM and its related complications.This costless early intervention can make healthy future generation.

Key Words: GDM-Gestational Diabetes Mellitus,IOM-Institute Of Medicine,GWG-Gestational Weight Gain .

INTRODUCTION

HISTORY

Until the mid 19th century diabetes was considered to be incompatible with successful pregnancy. Diabetes in pregnancy was considered as a major risk factor for serious maternal and fetal complications until 1922(1).During this period pregnancy was discouraged in young women with type 1 diabetes mellitus. Before 1922, only fewer than 100 pregnancies were reported in diabetic women. The reported " infant mortality rate were more than 90% and maternal mortality rate were of 30%".But few years after the discovery of insulin, the efforts of Priscilla White and other pioneers created the subspeciality of diabetes in pregnancy which changed the outlook of the deadly disease .since 1980 neonatal and maternal mortality started declining because of better treatment protocols for maternal plasma glucose control with self monitoring - blood glucose (SMBG) and HbA1C evaluation and ultra sonogram availability.

Concept of Gestational Diabetes was developed in the middle of 19th century by a German physician Bennewitz in 1824 ,who described a single case with diabetes following conception which disappeared after delivery(baby weighed 12 pounds and was told to be robust and healthy). The term Gestational Diabetes was first coined by

O'Sullivan in 1961, following the lead of the term metagestational diabetes by Hoet from Belgium.

DEFINITION

Gestational diabetes is defined as "any degree of glucose intolerance with onset or first recognition during pregnancy".

Pathophysiology

Researchers quoted that "during pregnancy the complicated changes in maternal intermediary metabolism for accommodating the needs of growing fetus have major influence on maternal health and physiology"[(2)].During first half of pregnancy the changes occur which promote storage of energy and nutrients. It is a state of "facilitated insulin action" which stores energy in the form of fat. The accumulated energy stores can be used in second half of pregnancy for the demands of rapidly growing fetus. The second half of pregnancy is a state of "diabetogenic stress" which is developed because of insulin resistance in the mother. These are due to high hormone level [elevation of oestradiol, oestriol, oestrone, progesterone, cortisol, prolactin and HPL (main driver of insulin resistance)], delayed disposal of glucose, elevated serum insulin (fasting) levels, and reduction in insulin release after food[(3,4)]. Insulin resistance in the mother decreases the carbohydrate uptake by the maternal peripheral tissues like adipose tissue which diverts glucose to

the fetus. The free fatty acids and ketone bodies are used as fuels for mother's energy requirements. Increased glucose uptake by the fetus results in low maternal fasting plasma glucose levels. In late pregnancy there is exaggeration of normal swing between fed-state anabolism and fasting catabolism compared to non pregnant individuals. During feeding there is compensatory hyperinsulinaemia as a result of insulin resistance[(1)].In the fasting state blood glucose level falls more rapidly than non pregnant individuals resulting in accelerated lipolysis and ketone body formation known as " accelerated starvation"[(1)].

PATHOGENESIS OF GDM:

Since the insulin resistance develops only during second half of pregnancy, GDM seldom develops before this period. The insulin resistance results in "Exhaustion of the β cell which reduces the capacity of the β cells to secrete required levels of insulin to compensate for the insulin resistance induced by the progression of pregnancy and therefore lead to the development of GDM"[(1,5)].

The risk factors include obesity[(6)],metabolic syndrome, diabetes in first degree relative, age more than 25 years, member of high risk ethnic group(Asian, Indian origin),polycystic ovary syndrome, previous unexplained perinatal loss, birth of malformed baby and polyhydromnios in previous pregnancy[(7)].

Gestational Diabetes Mellitus is associated with severe maternal and fetal morbidity and mortality[8,9,10]. The maternal complications[2,3,11,12] are pre eclampsia, polyhydramnios, increased caesarean delivery[13], perineal trauma, risk of type 2 diabetes in future. The fetal complications are macrosomia[14,15,16], birth trauma, prematurity, respiratory distress syndrome, neonatal metabolic complications like hypoglycaemia, hyperbilirubinaemia, polycythaemia, hypocalcaemia, risk of diabetes in future and metabolic syndrome[17,18,13,19,7,20].

Future risk of diabetes in mother:

GDM, though resolves in women after delivery, it persists in 5-10% of women and 35-60% will develop type 2 DM within the next decade. So GDM can be considered as unmasking of future type 2 DM[19,21].

Risk of future diabetes in offspring

Studies in pima Indians and other ethnic groups showed that infants of GDM develops type 2 DM during their 3rd decade and their risk of developing hypertension, central obesity and low HDL were also found to be high[19,22].

Scenario in Indian population:

Seshiah et al,[1] stated in 2008 that GDM was diagnosed in 17.8% women in urban population, 13.8% in semi urban population and 9.9% in rural population in a prospective study screened for GDM. Indian women reported highest frequency of GDM, compared to other ethnic groups in South Asian countries. The cause for raising prevalence rate in developing countries is related to urbanization, sedentary life style, dietary pattern change hypertension[23] and raising prevalence of obesity. This increased rate implies that Indian population with greater incidence of diabetes mellitus and IGT are more prone for developing GDM and is at a relatively increased risk of GDM. Recent multicentric study in India, showed highest prevalence of GDM in Tamilnadu and lowest in Kashmir. With the raising prevalence of type 2 DM and IGT, the incidence of GDM will be expected to go up further.

In our study the aim was to find out the "correlation between excessive early gestational weight gain and the risk of gestational diabetes mellitus" and its related maternal and fetal complications. The increased weight gain in early gestation accumulates fat in adipose tissue which increases insulin resistance and reduces the capacity of β cells, resulting in decreased insulin secretion which predisposes to GDM.

Researchers stated that "**Avoidance of excessive weight gain in early pregnancy"**[24,25,26,27,28] is an efficient method to prevent GDM &

its complications. But more studies are necessary to determine the possibility of early intervention and the best protocols for the pregnant women to help and meet the recommendations for gestational weight gain[25,29,30].

BMI limit :

	Indian	IOM 2009
Underweight	<18.4	<18.5
Normal	18.5 to 22.9	18.5to24.9
Overweight	23 to 24.9	25to29.9
Obese	> 25	>30

According to IOM 2009, guideline Calculations assume a 1.1–4.4 lb(2kg) recommended weight gain in the first trimester.

(Modified from Institute of Medicine (US). Weight gain during pregnancy: re-examining the guidelines. Washington, DC. National Academies Press; 2009. ©2009 National Academy of Sciences.)[30]

According to the Asian Indian guidelines, the waist circumference cut off of Indian men is 90 cm (102 cm globally) and for Indian women is 80 cm(88 cm at the international level).

Researches observed during the last several years and stated that "Indian bodies and genetics differ from their western counterparts as Indians have abdominal obesity compared to the western people whose bodies are uniformly obese"[31,32,33].This body constitution complicates and fix the Indian in the high risk region for hypertension and diabetes[29,34].

The importance of weight and fitness guidelines, in Asian countries, was first assessed in a randomized study by the World Health Organisation's (WHO) sub-committee group for obesity and metabolic syndromes (Syndrome X- heart disease, obesity and diabetes) in the Asia-Pacific region in 2000[34].

India compiled its weight and flab statistics for the first time officially, and the opinion is an important act to fight against obesity and its impact on Diabetes.

SCREENING FOR GDM AND DIAGNOSIS

Universal screening for GDM is necessary, as it is generally accepted that "women of Asian origin and especially Indians are at a higher risk of developing GDM and future type 2 diabetes".

World Health Organization Procedure

World Health Organization (WHO) recommends , 2-hour 75 g oral glucose tolerance test (OGTT) with a plasma glucose concentration of greater than 140 mg/dL at 2 hours, similar to that of IGT (more than 140 mg/dL and less than 199 mg/dL), outside pregnancy for diagnosis of GDM.

Time	Normal Tolerance	Impaired glucose tolerance	Diabetes
Fasting	<100	≥100 and<126	≥126
2 hour post glucose	<140	≥140 and<200	≥200

CARPENTER & COUSTON (1982) : DIAGNOSIS OF GDM BY 100G 3-HOUR OGTT

GDM is categorized, if any two values or met or exceeded. If only one value is abnormal, it is labeled as gestational impaired glucose tolerance.

Fasting	95 mg/dl
1 hour	180 mg/dl
2 hour	155 mg/dl
3hour	140 mg/dl

The International Association of the Diabetes and

Pregnancy Study Groups (IADPSG)

The IADPSG recommends that diagnosis of GDM is made "when any one of the following plasma glucose values meet or exceed" [19]:

OGTT is done in the morning after overnight fast of at least eight hours

Fasting	≥ 92 mg/dL (5.1 mmol/L)
1hour	≥ 180 mg/dL (10.0 mmol/L)
2hour	≥ 153 mg/dL (8.5 mmol/L)

• The IADPSG also suggests:

* Fasting plasma glucose: (FPG)>7.0mmol/L (126 mg/dL)

* HbA1C > 6.5% - in the early weeks of pregnancy is diagnostic of overt diabetes.

* Fasting plasma glucose : > 5.1 mmol/L and < 7.0 mmol/L is diagnosed as GDM.

Disadvantages of the IADPSG suggestions are:

- Fasting state at first antenatal visit is impractical .

- In all GDM, fasting plasma glucose values donot reproduce the 2-hour postglucose (with 75 g oral glucose) , which is the hallmark of GDM in Asian Indian women. In this population by following FPG > 92 mg/dl as cut-off value, 76% of pregnant women will be missed for the diagnosis of GDM made by WHO criteria.

Diabetes in Pregnancy Study Group India : A single step procedure

"A Single-step procedure was developed because of the practical difficulty in doing glucose tolerance test in the fasting state"[1,2].

Procedure

A pregnant woman after clinical examination, has to be given a 75g oral glucose load, irrespective of fasting or nonfasting state or the time of the last meal. A venous blood sample is collected at 2 hours for calculating plasma glucose by the GOD-POD method. GDM is diagnosed if 2-hour PG is ≥ 140 mg/dL .

Advantages of the DIPSI procedure are:

- No need for fasting state.
- pregnant woman's routine activities are not affected.
- Screening and diagnostic procedure.
- This procedure has been approved by Ministry of

Health, Government of India and recommended by WHO.

- **DIPSI** guideline for diagnosis of GDM, according to 75g oral glucose tolerance test (WHO criteria)

Criteria	In Pregnancy	Outside Pregnancy
2hr ≥200mg/dl	Diabetes	Diabetes
2hr ≥140mg/dl	GDM	IGT
2hr ≥120mg/dl	DGGT	-

Abbreviation:

GDM: Gestational diabetes mellitus; DGGT: Decreased gestational glucose tolerance; IGT: Impaired glucose tolerance

In this study GDM was diagnosed according to DIPSI (Diabetes in pregnancy study group in India) guidelines where the 2 hr post prandial glucose level more than 140 mgs is diagnosed as GDM.

Cunningham FG et al in Williams Text Book of Obstetrics (24th edition,2014) [35],quoted weight gain recommendations according to the Institute of Medicine (2009) guideline[30],which was endorsed by the American Academy of Pediatrics and the American College of Obstetrics and Gynaecologists (2012) .**The guideline recommends narrow range of weight gain for obese women. Also the same recommendations**

apply to adolescents, short women and women of all racial and ethnic groups.

According to Williams Text Book of Obstetrics (24th edition,2014) early gestation (first trimester) corresponds to 14 weeks gestation.[30]

So in our study, Gestational **weight gain more than 2 kg(upper limit of IOM 2009 recommended weight gain for I st trimester) till 14 weeks** , was taken as excessive early gestational weight gain.

Waist hip ratio:

World Health Organisation protocol: It should be measured at the midpoint between lower margin of the last palpable rib and the top of the iliac crest with a tape. Hip circumference should be measured around the widest portion of the buttocks, with the tape parallel to the floor. The WHO(World health organization) states that " abdominal obesity is defined as a waist–hip ratio above 0.90 for males and above 0.85 for females, or a body mass index(BMI) above 30.0"[46,33].

Since gestational diabetes mellitus is related with adverse maternal and fetal outcomes, protocol for management of GDM is essential.

MANAGEMENT OF GDM:

Target

Sustaining a mean plasma glucose (MPG) level ~105–110 mg/dL is appropriate for a good neonatal outcome[48,49]. This can be achieved if fasting blood sugar and 2-hour postprandial peaks are ~90 mg/dL and ~120 mg/dL, maintained respectively[39].

Medical Nutrition Therapy

All pregnant women with GDM should be counselled about proper nutrition. The recommended calorie intake depends on the pre pregnancy body weight, 30 kcal/kg for the normal weight pregnant women, 35 kcal/kg for underweight and 25 kcal/kg for obese women. The total calories (3 meals and 3 snacks) should be contributed by about 50-60% from carbohydrates, 20% from protein and 25-30% from fats with less than 10 % from saturated fats [51, 52].

INITIATING INSULIN THERAPY

After the diagnosis of GDM, medical nutritional therapy (MNT) is recommended initially for 2 weeks[44]. If there is no adequate response to MNT to maintain control, i.e. FPG ~90 mg/dL and/or post-meal glucose ~120 mg/dL, insulin may be started[39].

Preferable to start with Premix insulin 30/70

Initial dose: 4 units before breakfast

↓

Every 4th day increase 2 units till 10 units

↓

If FPG remains > 90 mg/dL advise→6 units before

breakfast and 4 units before dinner

↓

Review with blood sugar test →Adjust dose further

Total insulin dose per day can be divided as two-thirds in the morning and one-third in the evening. The morning dose is constituted by two thirds of intermediate acting insulin and one third regular or rapid acting insulin whereas the evening dose contains half intermediate and half regular insulin.

Initially if post-breakfast plasma glucose is high → Start Premix 50/50

If GDM is diagnosed in the third trimester; MNT is advised for a week. Insulin is started if MNT fails.

If 2-hour Postprandial glucose > 200 mg/dL , 8 units of Premixed insulin can be started initially before breakfast and the glucose level is

monitored and the dose is titrated on follow-up. With insulin therapy, Medical nutrition therapy should also be advised.

INSULIN ANALOGS

If postprandial glucose, control is not achieved— rapid acting insulin analogues should be considered.

MONITORING GLYCEMIC CONTROL

2-hour post prandial glucose monitoring is preferred as the diagnosis of GDM .The blood tests must be done at the same time at each visit for monitoring the target glucose level and adjusting insulin dose .Self-monitoring of blood glucose (SMBG) should be advised on day to day basis for the women who are not well controlled on multiple insulin injection regimen. If it fails at least weekly monitoring should be advised.

ORAL ANTIDIABETIC DRUGS

Glibenclamide is used in a few centers of India and abroad, but drug controller of India not yet approved the drug.

Metformin

Metformin crosses the placenta, but many observational studies on metformin used in early pregnancy have found no teratogenic effects

and can, therefore be used as an adjunct or alternative to insulin after counseling the patient.

MEASURING OTHER PARAMETERS

Maternal

The blood pressure should be checked during every antenatal visit. Fundus examination and urine examination for microalbuminuria, every trimester is suggested particularly in women with pregestational diabetes.

Fetal surveillance:

Ultrasonogram: Ultrasound monitoring is recommended once in a trimester atleast. Fetal echo should be done in second trimester particularly in women with pre GDM.

Timing of delivery:

Patients with gestational diabetes well controlled on diet can be followed till 40 weeks, at that time induction of labour can be considered. Delivery before full term is indicated for obstetric indications. Since GDM on insulin represents a greater degree of glucose intolerance pregnancy is usually terminated at 38-39 weeks[(1)].

Delivery:

During labor, maintaining good glycemic control is essential, and at the same time hypoglycaemia should be avoided. Insulin requirements

will become lesser in the process of labour (insulin may not be necessary).Monitoring of maternal blood glucose level should be done postnataly and 24 hours postpartum. If it is high, blood sugar level should be checked again on follow-up. The availability of neonataologist should be essential at the time of delivery if neonatal morbidity is suspected[1].

FOLLOW-UP

Gestational diabetic women requires follow-up. An OGTT should be performed at 6–8 weeks postpartum. If it is normal, glucose tolerance test is repeated after 6 months and annually. They should be counseled regarding diet, exercise and weight reduction which can reduce their chances or delay in developing type 2 diabetes later.

AIM

- To study the correlation between the rate of excessive early gestational weight gain & risk for gestational diabetes mellitus.

OBJECTIVE

- To study effects of abnormal weight gain in early pregnancy
- To study the risk of developing GDM
- To study maternal complications during delivery due to GDM
- To study neonatal complications due to GDM

OUTCOME MEASURE

Primary outcome:

To correlate the association between excessive early gestational weight gain and risk of developing GDM.

Secondary outcome:

Maternal & Neonatal complications are compared between women developing GDM with excessive early gestational weight gain and normal gestational weight gain.

Maternal complication:

- Pre eclampsia
- Induction of labour
- Operational delivery
- Polyhydominios

Fetal complications:

- Large for gestational age baby
- Small for gestational baby
- Birth asphyxia
- Hyperbilirubinaemia
- Hypoglycaemia

REVIEW OF LITERATURE

EARLY PREGNANCY WEIGHT GAIN AND RISK OF GDM

1. **Herring** et al (2009)[26] states that " higher the GWG, higher the risk of developing GDM in the third trimester of pregnancy". They observed that "maternal obesity and PostPartum Weight Retention (PPWR) have also been associated with excessive GWG". In addition to maternal outcomes, they also recognized that "excessive GWG has been associated with large for gestational age (LGA) babies and excessive neonatal weight"

2. **Hedderson MM et al(2010),[6]** studied a multiethnic cohort of 345 women with GDM and 800 women as control, delivered between 1996 and 1998, screened for GDM at 24–28 weeks of gestation. They found that the "association between gestational weight gain and risk of GDM was mainly attributable to excessive weight gain in the first trimester"

3. **Jeannine stein 2010[25]** studied Early-pregnancy Weight Gain association with Gestational Diabetes and declared "excessive weight gain during the first trimester can increase the risk of GDM, maternal and neonatal morbididy". They concluded that "gaining

weight before conception or being overweight at the start of pregnancy results in higher risk for gestational diabetes". But a new study observed that "first trimester is the most crucial time and excessive weight gain is the independent risk factor that can increase the danger of GDM"

4. **Zilko M** et al (2010)[20] collected data from 4,496 births during 1979 in the National Longitudinal Survey of Youth and described the "correlation between GWG and increased rates of GDM, cesarean delivery , PPWR, LGA as well as childhood overweight". They concluded that, "40% of women with excessive GWG retained greater than 2.5 kg from 12 to 24 months after delivery and 29% of the children had a BMI greater than the 85th percentile"

5. **Metzger JJ** et al(2011)[18] conducted a study to determine if women with normal glucose tolerance who gain weight beyond the 2009 Institute of Medicine guidelines (based on pre-pregnancy BMI) have newborns with increased fat mass. Obese women gained weight beyond recommended guidelines (70% vs. 31%) than healthy weight women. Maternal fasting glucose, C-peptide (marker of insulin secretion) and leptin were significantly higher in

the excessive GWG group. Newborns of mothers with excessive GWG had significantly higher fat mass (490 vs. 390 g, p=0.04) and they tended to weigh more (3.61 vs. 3.38 kg). Neonatal length, abdominal circumference, cord blood glucose and leptin were similar between the groups, but cord blood C-peptide was definitely higher in the excessive GWG group.

6. **Morisset AS** et al (2011)[46] conducted a study to "associate gestational weight gain in women and GDM" The collected datas were retrospective reviews of medical records in 294 women. According to the 2009 recommendations by the Institute of Medicine (IOM), gestational weight was assessed. Women with GDM were treated according to the Canadian Diabetes Association guidelines. Compared to controls weight gain in the first trimester was significantly higher in GDM patient. They concluded that "First trimester gestational weight gain may need clinical intervention as it was identified as an independent and significant risk factor for GDM"

7. **Carreno CA et al(2012),**[29] executed a randomized study on 7,985 women who were studied to estimate the " correlation between excessive early gestational weight gain and the subsequent risk of

gestational diabetes mellitus (GDM) and fetal macrosomia". The studied population showed, excessive early pregnancy weight gain in 93% of women, where the total pregnancy weight gain more than the IOM guidelines. They also observed that in nulliparous, low risk women, excessive early pregnancy weight gain is correlated with the risk of GDM and fetal macrosomia. They Concluded that "excessive early GWG (Gestational Weight Gain) is associated with adverse outcomes including gestational diabetes mellitus, cesarean delivery and Large for gestational age baby in particular, the largest effect of excessive early pregnancy weight gain was found in women with a pre pregnancy normal BMI."

8. **Devi** NS et al (2014) [27] in May 2014, determined the " prevalence of overweight and obese pregnant women, and maternal and fetal associations with overweight and obese pregnant women classified using the revised consensus guidelines for BMI in Asian Indians". They analyzed retrospectively case records between January 2010 and December 2012 at a tertiary care institute in India. BMI was classified using the "revised consensus guidelines for Asian Indians and the World Health Organization (WHO) criteria". The prevalence of obesity increased from 11.81% with the WHO criteria to 43.11% with the Asian Indian guidelines and led to the

re-classification of 1,345 (18.47%) pregnant women from a low risk category to a high risk category. Maternal and fetal complications were associated with overweight or obesity (both Indian and WHO guidelines). Obesity (both Indian and WHO guidelines) was also significantly associated with caesarean sections. They concluded that the "revised guidelines led to a larger classification of high risk Asian Indian pregnant women and retention of adverse associations of overweight and obesity support adoption of the revised guidelines in obstetric management of Asian Indians".

9. **Egan** AM et al(2014)[45] conducted a study along the Irish Atlantic seaboard at five antenatal centers. 802 women with diabetes in pregnancy participated in this study. Maternal outcomes examined and included were gestational hypertension, pre eclampsia and caesaerean delivery. Fetal complications included were large for gestational age (LGA), fetal macrosomia, and small for gestational age(SGA).Excessive GWG was observed in 59% of women. In all women, excessive pregnancy weight gain resulted in increased odds for LGA and macrosomia . Excessive pregnancy weight gain was also associated with an higher odds for gestational hypertension in women with GDM. They concluded that "in the

"already high-risk odds for both GDM and PGDM (Pre Gestational Diabetes mellitus), excessive pregnancy weight gain confers an increased risk for LGA birth weight, macrosomia, and GHT "

BODY MASS INDEX

10. **Hedderson MM et al (2008)**,[22] studied 123,040 women without history of pre GDM between 1995 and 2006. The observation revealed "the risk of GDM, increased with increasing BMI". They concluded that "the prevalence of GDM could be prevented if all pregnant women belongs to normal weight category according to IOM guideline ranging from 65% for Africo American women and for Asian women it is only 23% and the risk is more at relatively low BMI cutoffs in Filipina and Asian women".

11. **Whiteman VE** et al (2011)[31] observed "whether changes in interpregnancy body mass index has any influence on the development of the gestational and type 2 diabetes in a cohort of women with two consecutive live, singleton births of 20–44 weeks gestation". Mothers who progressed from a low BMI category into a high category had risk for the developing diabetes and also mothers who progressed from prepregnancy normal weight in the first pregnancy to obese prepregnancy weight in the next

pregnancy showed elevation in risk. Mothers who maintained their interpregnancy BMI weight or who progressed to a lower BMI category had decreased risk for GDM and type 2 diabetes. They concluded that " interpregnancy weight gain is associated with a increase in risk of diabetes. Maintaining a normal interpregnancy BMI may decrease the risk of diabetes"

12. **Alberico S** et al (2014),[42] conducted a prospective study of 14109 women and associated with " mode of delivery and maternal and neonatal outcomes to evaluate the independent role of pre-pregnancy body mass index , gestational weight gain and gestational diabetes on the risk of fetal macrosomia". They concluded that " maternal obesity, excess gestational weight gain and diabetes should be recognised as independent risk factors for fetal macrosomia"

OBESITY

13. **Tovar A** et al (2009)[6] conducted a prospective cohort study in 813 Hispanic prenatal patients in Massachusetts. In this prospective cohort study, they tried to associate "the role of gestational weight gain and development of AGT and found that exceeding target weight gain during pregnancy increased AGT risk only in women

with obese category". They concluded that "weight gain was associated with AGT among class II and class III obese women".

14. **The Institute of Medicine** (2009)[30] gives recommendation for total pregnancy weight gain based on prepregnancy BMI. These ranges are: 12.7 to18.1 kg for underweight, 11.3 to 15.9 kg for normal weight, 6.8 to11.3 kg for overweight and at least 6.8 kg for obese women (BMI > 29). The IOM also recommends "trimester-specific weekly GWG again upon prepregnancy BMI, Specifically for the second and third-trimesters, and recommends 0.490 kg/week for underweight women, 0.440 kg/week for normal weight women and 0.30 kg/week for overweight women".

15. **Black** MH et al (2013)[3] studied 10,459 women who met the IADPSG criteria. They suggested that " prepregnancy overweight and obesity contributes to the prevalence of LGA, and other adverse outcomes and the effects of GDM and maternal BMI appear to be additive, but only a small proportion of LGA cases were associated with GDM in the absence of overweight or obesity".

WAIST HIP RATIO AND GDM

16. Branchtein L et al (2001)[43] in 1997 evaluated the " relationship of central fat distribution with gestational glucose tolerance during the usual time for screening gestational diabetes". Waist-hip ratio (WHR) and waist circumference were independently related with higher 2-h post prandial glycemia. They concluded that "Central fat distribution is an independent risk factor of gestational diabetes mellitus"

17. Madhavan A et al (2009) [36] in 2008 observed that the prevalence of GDM was higher in pregnant women with higher waist–hip ratio compared with those having a lower WHR(Waist Hip Ratio). They observed that "waist circumference of 85.5 cm (with sensitivity of 75%, specificity 81.4%) and a BMI of 24.3 kg/m2 (sensitivity 75%, specificity 86.5%) had the best predictive value". In conclusion, they found that "WHR is more important risk factor for GDM in overweight and obese women than women with normal weight and lean women"

18. **Basraon** S et al (2015)[49] in 2013 determined the " relation of early pregnancy waist to hip ratio (WHR), a measure of central adiposity, versus body mass index (BMI), a measure of total body

fat, with abnormal glucose tolerance and insulin resistance (IR)". They concluded that " increased WHR and BMI in early pregnancy are related with development of insulin resistance and GDM" BMI is a better predictor of IR compared with WHR.

FAMILY HISTORY OF DIABETES MELLITUS (FHD)

19. **Rethnakaran R** et al (2007)[3] investigated whether " the family history of type 2 DM represents as a unique risk factor for GDM**"** GDM risk factors were evaluated in 90 women with FHD and in 83 women without FHD, at the time of OGTT (Oral glucose tolerance testing)in late pregnancy. They concluded that the "established risk factors for GDM are significant in women with FHD but may not be the principal risk factor of gestational hyperglycaemia in women without Family history of diabetes" Moreover, FHD may be more significant to risk of GDM in nulliparous women than in parous women. They concluded that these findings highlight relationship between FHD and gestational diabetes mellitus, and may suggest implications for selective screening for GDM.

OPERATIONAL DELIVERY

20. **Cheng YW** et al (2008)[47] conducted a retrospective study of GDM women with singleton pregnancies enlisted between 2001 and 2004 in the Sweet Success California Diabetes and pregnancy Program. This study investigated the "correlation between gestational weight gain and perinatal outcomes in women with GDM". They concluded that "women with GWG above the IOM guidelines were more likely to have a primary cesarean delivery and large birth weight, whereas women with less-than-recommended weight gain were likely to maintain glycemic control with dietary modifications but had increased risk of SGA and suggests that excessive weight gain above the IOM guidelines is associated with abnormal perinatal outcomes"

NEONATAL COMPLICATIONS.

21. **De Veciana M** et al(1995)[21] investigated "66 women with gestational diabetes mellitus who are treated with insulin therapy at 30 weeks of gestation or earlier and the women were randomly selected to have their diabetes managed according to the results of preprandial or postprandial glucose monitoring (one hour after meals) of blood glucose concentrations". They concluded that

"adjustment of insulin therapy in women with gestational diabetes according to the results of postprandial, rather than preprandial, blood glucose values improves glycemic control and decreases the risk of neonatal hypoglycemia, macrosomia, and cesarean delivery"

22. **Jovanovic-Peterson L** et al (1997)[54]designed a study to " observe the impact on birth weight and on cost of a treatment program for GDM women in The Santa Barbara County Health Care Services (SBCHCS)". Based on the result with increasing glucose levels resulting in increasing prevalence of fetal overgrowth. They observed that "After introduction of the screening and treatment program, the prevalence of macrosomia in 1992 was 7% and the cesarean section rate had dropped from 30 to 20%". The cost was $233,650 to educate and treat GDM women. They also assessed $833,870 per year can be saved if an additional 398 macrosomic infants were prevented from being born by good glycaemic control. They concluded that, "treatment of GDM women was associated with a decrease in risk of macrosomia and may be cost-effective".

23. **Negrato CA** et al(2008)[34] conducted this study to examine "the prevalence of metabolic syndrome (MS) in a cohort of 136

pregnant women with glucose tolerance, pre-pregnancy risk factors for MS during pregnancy and the adverse perinatal outcomes". They concluded that the "prevalence of MS increases with the worsening of glucose intolerance and is an independent risk factor of adverse perinatal outcomes and impaired glycaemic profile identifies pregnancies with important metabolic abnormalities that are associated with adverse perinatal outcomes even in the presence of a normal OGTT, in patients that are not currently diagnosed as having GDM".

24. **Landon MB** et al(2009)[(44)]studied 958 Women in the 24th to 31st week of gestation and who diagnosed as mild gestational diabetes mellitus selected for usual prenatal care, dietary intervention, self-monitoring of blood glucose, and insulin therapy. "The primary outcome was composed of stillbirth or perinatal death and neonatal complications, including hyperbilirubinemia, hyperinsulinemia, hypoglycaemia and birth trauma". They concluded that "although treatment of mild gestational diabetes mellitus did not significantly reduce the frequency of a complex outcome, it reduced the risks of fetal macrosomia, shoulder dystocia, cesarean delivery, and hypertensive disorders".

25. **Park JE** et al (2011)[17] conducted a hospital-based study of GDM women with BMI of ≥25 kg/m^2. Weight, glucose levels, lipid profiles, insulin treatment, and maternal outcomes were studied and associated with neonatal birth weight. Excessive pregnancy weight gain resulted in fetal macrosomia, HbA_{1c} at delivery, and PPBS levels, but pre prandial blood glucose levels were not affected . They concluded that "minimal weight gain, well below IOM recommendations, and strict control of blood glucose levels during pregnancy with proper medical management and dietary and lifestyle modification may alleviate most of the adverse outcomes in pregnancy experienced by obese GDM Asian women".

RELATED STUDIES

26. **Goldberg JD** et al (1986) [15] studied two groups of 58 gestational diabetic women matched for age, parity, prepregnancy weight and height. The home glucose monitoring study group performed . The control group followed up by conventional treatment. The incidence of macrosomia was significantly reduced in the home glucose monitoring group. They concluded that "intensive home glucose monitoring will allow for the early identification of those gestational diabetic patients needing insulin and thus reduce the incidence of macrosomia and large for gestational age infants".

27. **Langer O** et al (1989)[14] assessed the relationship of optimal levels of glycemic control and perinatal outcome in a study group matched for control of obesity, race, and parity. They concluded that "relationship exists between level of glycemic control and neonatal weight".

28. **Wechter DJ** et al(1991)[48] conducted a study to "determine if intensive dietary therapy, home blood glucose monitoring, and the insulin can be effective in combating fetal overgrowth". All pregnant women were screened at 24 to 28 weeks gestation using a modified O'Sullivan's criteria. The 153 GDM patients advised on

an 1800 to 2000 Kcal, American Diabetes Association diet and taught home glucose monitoring. Insulin therapy started only if plasma glucose control was insufficient. There were no significant differences ($p > 0.05$) between the study and control group with regard to mean birth weight or macrosomia. They conclude that the "incidence of fetal macrosomia in gestational diabetes can be kept equal to that of the general population by a program of intensive dietary therapy and home glucose monitoring, with insulin being used only therapeutically, not prophylactically"

29. **Pettitt DJ** et al(1991)[39] studied the "long-term effects on the offspring of GDM women detected during pregnancy were examined in 552 Pima Indian offspring 5-24 yr of age". Fasting hyperinsulinemia, arise at the earlier age in the offspring of GDM women, and they were more obese and increasing rates of abnormal glucose level. They concluded that, "the metabolic abnormalities associated with the diabetic pregnancy results in long-term effects on the offspring, including insulin resistance, obesity, diabetes, which in turn may contribute to transmission of risk for developing the same problems in the next generation".

30. **Metzger BE** et al (1991)[24] conducted a multicenter, multinational study, to examine the association of neonatal adiposity with plasma glucose levels of pregnant women and cord serum C-peptide thereby linking the Pederson hypothesis of maternal glycemia and fetal hyperinsulinemia to neonatal adiposity. Among 23,316 HAPO Study participants , findings confirm the association between maternal glucose and neonatal adiposity and concluded that the "relationship is mediated by fetal insulin production and that the Pedersen hypothesis describes a basic biological relationship influencing fetal growth".

31. **Catalano PM** et al (1993)[4] was conducted a study to "characterize carbohydrate metabolism associated with the development of gestational diabetes". Insulin sensitivity decreased during gestation and was primarily decreased in GDM compared with control. They concluded that the "findings closely resemble those of non-insulin-dependent, predominantly insulin-resistant diabetes, which is often a sequel of GDM".

32. **Mello G** et al (1997)[50] studied the "relationship between perinatal outcome and daily glucose profile throughout pregnancy ". The study population divided into two groups Group I patients

without neonatal complications and Group II patients with at least one form of neonatal complications. They concluded that “an optimum perinatal outcome can be achieved only if the pre-pregnancy diabetic women can achieve a metabolic equilibrium during the second trimester which matches the daily excursions of glycemia present in a non-diabetic pregnant women avoiding individual episodes of night-time hypoglycaemia” .

33. **Bevier WC** et al(1999)[16] studied pregnant women with positive GCT, but negative 3-hr,100g OGTT. The pregnant women were grouped into either experimental or control groups with experimental women receiving counseling regarding diet and home blood glucose monitoring instruction (HBGM). The aim of this study was to “examine the effectiveness of the treatment program in decreasing neonatal macrosomia, maternal and neonatal morbidity, maternal complications, and operative delivery”. In Santa Barbara County, pregnant women were screened at 24-28 weeks with a 50-g, 1-hr glucose challenge test for GDM. GDM women were given standard euglycemic diet and perform HBGM of fasting and postprandial glucose levels. Women with abnormal GCT, but normal OGTT and thus not diagnosed as GDM are still at risk for delivering a macrosomic baby and operative delivery.

They concluded that “treatment for all pregnant women with abnormal GCT improves outcome by reducing infant birth weight and the number of cesarean sections”

34. **Kang CH** et al(2001)[37] conducted a retrospective study of “ 402 GDM women with singleton pregnancies with cephalic presentation delivered at Ilsin Christian Hospital during the period January 1, 1997, to December 31, 1999”. These women were compared with a nondiabetic control group randomly selected and the effects of confounding variables were analyzed and compared maternal behaviour and pregnancy outcomes. Pregnancy outcomes of gestational diabetic women were not improved by the conventional management, and more strict but acceptable and compliable treatment should be tried.

35. **Ben-Haroush A** et al (2004)[49] conducted a study to “determine postprandial glucose profile in the diabetic pregnancy”. Pregnant women were connected to 72 consecutive hours continuous glucose monitoring system. They concluded that “the time interval for postprandial glucose peak in diabetic pregnancies is approximately 90 minutes after meals throughout the day and is not affected by

the level of glycemic control and this information should be considered in the treatment of diabetes mellitus in pregnancy".

36. **Brawarsky P** et al (2005) [18] conducted a study in a group of pregnant women who completely answered the questions regarding diet and pregnancy weight gain and singleton full-term infant. Conclusion: " Interventions to prevent excessive gestational weight gain should be started before pregnancy and women at risk for inadequate gain would also benefitted from interventions directed toward modifiable factors during gestation."

37. **Seshiah V** et al (2008) [1] reported practice guidelines for GDM in the Indian women. Due to increasing prevalence, screening by DIPSI is recommended. Screening is usually recommended between 24 and 28 weeks of gestation The maternal and fetal outcome depends on the care given by the team of diabetologists, obstetricians and neonatologists. They concluded that "a short term intensive care gives a long term pay off in the primary prevention of obesity, IGT and diabetes in the offspring, as the preventive medicine starts before birth".

38. **Hedderson MM** et al(2008)[13] conducted a study in women with hypertension either 5 years before pregnancy or the first trimester

of pregnancy, had a twofold increased risk of developing GDM during pregnancy. While attenuated, these associations were persisted after adjusting for BMI. They implies that the "association is independent of BMI and the association between Blood pressure and GDM was stronger among women who were overweight (BMI _25.0 kg/m2)".

39. **Gayle C** et al(2010)[11] concluded that " diagnosis of GDM with OGTT 2-h PG ≥ 7.8 mmol/L and treatment in a combined diabetes antenatal clinic is worthwhile with a decreased macrosomia rate and fewer emergency cesarean sections".

40. **Balaji V** et al(2011)[2]conducted a study, in which women were given 75 g oral glucose load irrespective of their last meal and 2-h PG ≥ 7.8 mmol/L were diagnosed as GDM. The explanation is that, "after a meal, woman with normal glucose tolerance would be able to maintain euglycemia despite glucose challenge because of brisk and adequate insulin secretion, but in a woman with GDM who has impaired insulin response, her glycemic level increases with a meal and with glucose challenge". This cascading effect is advantageous as this would not result in false positive diagnosis of GDM. In India more than 70% of population live in rural settings

and facilities for diagnosing DM is limited. In this scenario, performing OGTT recommended by other associations [e.g., American Diabetes Association, National Diabetes Data Group, International Association of Diabetes and Pregnancy Study Groups] to diagnose GDM is not feasible as the cost involved is more to perform three blood tests and thus not accepted by both health care providers and seekers. DIPSI criterion suggests estimation of plasma glucose level in one blood sample to diagnose GDM. This cost-effective and evidence-based procedure meets and offers "a single-step definitive glucose test" to all pregnant woman belonging to any socio-economic status. This study has validated the credibility of DIPSI criterion.

41. **Ehrlich SF** et al (2011)[38] conducted a retrospective cohort analysis **t**o correlate the " association between inter-pregnancy change in body mass index (BMI) and the risk of gestational diabetes (GDM) in a second pregnancy". 22,351 women were studied and women with inter-pregnancy BMI gains showed an elevated risk of the disease in the second pregnancy. They realised that "the loss of BMI units was associated with a decreased risk of GDM only in women who were overweight/obese in the first pregnancy". They concluded that "Inter-pregnancy increases in

BMI may elevate a woman's risk of GDM pregnancy, but reductions in BMI will be protective, particularly in overweight and obese women".

42. **Katon J** et al (2013)[32] conducted a retrospective cohort study in "Overweight and obese women with live singleton pregnancies treated for GDM at a large diabetes and pregnancy program located in Charlotte, NC between November 2000 and April 2010". The association of weight loss in GDM women and birth weight were examined by maternal pre-pregnancy overweight or obesity class (I, II/III). Out of 322 women in the study 19 % lost weight between diagnosis of GDM and delivery. They concluded that "weight loss, after diagnosis of GDM, is associated with lower infant birth weight in overweight women, but not in obese class II/III women".

43. **Sivaraman SC** et al (2013)[5] conducted this observational cohort study to "determine the long term risk of diabetes in a cohort of women with previous GDM, and investigate which ante-partum and post-partum factors are related with the size of the risk". There was no correlation with age, gestational age at diagnosis of GDM, and parity. They Concluded that "Women with fasting antenatal glucose ≥ 7.0 mmol/L and/or an antenatal two-hour glucose ≥ 11.1

mmol/L are at higher risk of developing GDM and need close monitoring".

44. **Wendland EM** et al (2013)[12] selected two criteria of World Health Organization (WHO), and International Association for Diabetes in Pregnancy Study Group (IADPSG), generated in the HAPO (hyperglycaemia and adverse pregnancy outcome) study. The aim is to " review the evidence for the associations between GDM (according to these criteria) and adverse outcomes". They concluded that "The WHO and the IADPSG criteria for GDM identified women at a small elevated risk for adverse pregnancy outcomes and associations were of similar magnitude for both criteria".

45. **Cunningham FG** et al (2014)[35]recommends risk factors to decide which pregnant women to test for gestational diabetes . In these women, World Health Organization's diagnostic criteria used for screening gestational diabetes. In women with history of gestational diabetes in a previous pregnancy, offer early self monitoring or an OGTT at 16-18 weeks and at 28 weeks if the results are normal. They showed the association between risk factors and development of GDM.

46. **Kuhl C** (2014) [53]reviewed ,collected data and observed additional informations from articles that the " diabetogenicity of pregnancy is related to a increased peripheral resistance to insulin". They concluded that the " resistance which is of similar magnitude in pregnant women with NGT(normal glucose tolerant women) and women with GDM, is caused by post insulin receptor events and it probably because of the effect brought about by cellular effects of the high plasma levels of pregnancy associated hormones and free cortisol".

MATERIALS & METHODS

❖ STUDY DESIGN

Prospective observational study

❖ SELECTION

Pregnant women attending antenatal OPD in study centre (ESIC MC PGIMSR,K.K Nagar)

❖ SAMPLE SIZE

According to qualitative analysis **$(n=4pq/l^2)=250$ patients.**

❖ STUDY PERIOD

18 Months (NOV 2013 to MAY 2015)

INCLUSION CRITERIA:

- Singleton pregnancy
- Regular antenatal visits
- No associated co morbidity

EXCLUSION CRITERIA:

- Multiple pregnancy
- Anaemia complicating pregnancy

- chronic hypertension
- Pregestational diabetes mellitus
- Thyroid disorders complicating pregnancy
- Molar pregnancy

METHODS

- Maternal weight gain from self reported prepregnancy weight (upto 6 weeks) to 14 weeks of gestation is measured. Maternal weight gain more than 2kg is considered as excessive early gestational weight gain.

PROCEDURE

- During first antenatal visit height, weight and waist & hip ratio is measured
- Body mass index is calculated according to quetlet index(weight in kg/height in meter squared)
- Categorisation of the pregnant women according to ASIAN INDIAN BMI guidelines. Detailed history, general examination and obstetric examination done.

1. Blood samples (6-8ml) will be taken for

- Hb
- Renal function test
- Liver function test
- HbA1C
- Oral glucose challenge test (since fetal beta cell starts secreting insulin by 9-11 weeks gestation)

FOLLOW UP:

- ❖ monthly till 28 weeks/ fortnightly till 36 weeks/weekly till term(each time weight is measured)
- ❖ If the patient is not diagnosed as GDM in the first trimester, OGCT will be repeated at 24-28 weeks and 32-34 weeks gestation.
- ❖ If diagnosed as GDM patient, she will be treated as per protocol.
- ❖ Patient will be followed up till delivery

RESULTS

STATISTICAL TOOLS USED:

The information collected were recorded in a Master Chart in Excel sheet. Data analysis done with the help of computer using **SPSS statistical package- Version 17.**

Using this software range, ‘t’ value and 'p' values were calculated with frequencies, percentages, means, standard deviations. Student’s ‘t’ test was used to test the significance of association between early pregnancy weight gain and quantitative variables. For qualitative variables chi square test was used. A 'p' value less than 0.05 will denote significant relationship.

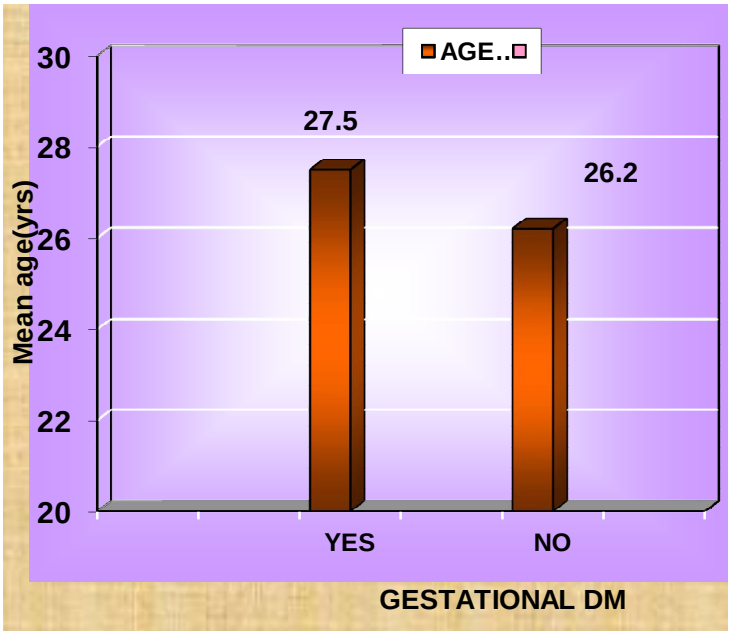
285 women who attended antenatal out patient department in ESIC MC PGIMSR , K.K Nagar were recruited in the original trial. Out of 285 women 12 antenatal women lost follow up, 8 women's prepregnant weight report were not available and 15 women developed obstetrical complications like anaemia, hypothyroidism and molar pregnancy.

In our study height measurements and self reported prepregnancy weight (upto 6 weeks gestation) to assess the BMI and upto 14 weeks of gestation[35] were taken. The early gestational weight gain more than 2 kg (according to upper limit of IOM guideline for first trimester)[30] was considered as excessive weight gain and was correlated with developing risk of GDM .Of the 250 women studied 104 women developed GDM who were compared with control of 146 women belonged to non GDM group .

The age distribution datas of the 250 women are summarized in Table 1.The age distribution had a high proportion between 25 -29 yrs and above 30 years are 58.

Table 1 : Gestational DM and Age

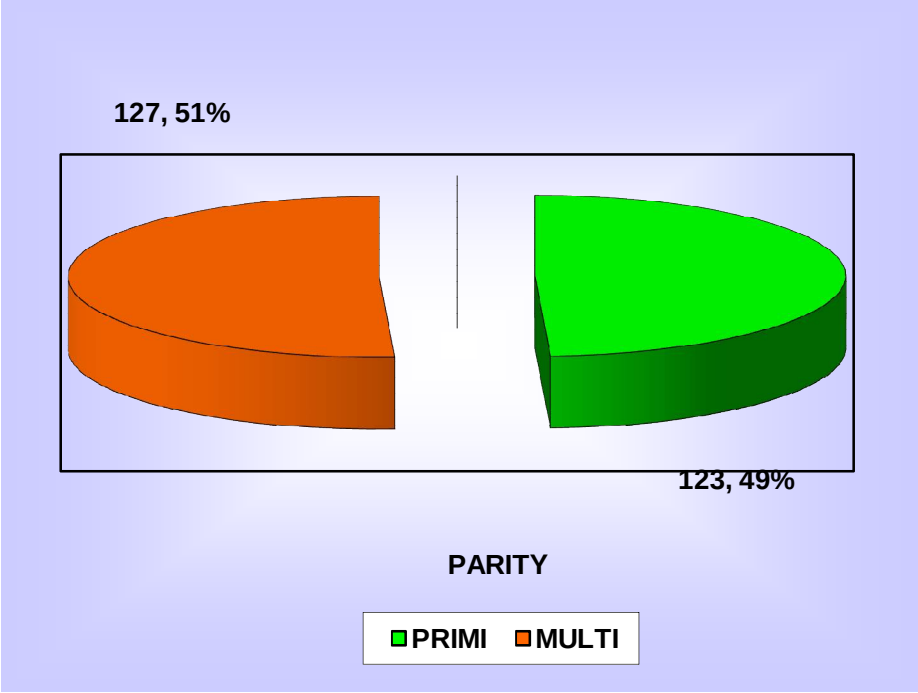
Gestational DM	Age of Mother		Age distribution	Case	
	Mean	S.D.		N	%
Yes	27.5	4.0	Below 20 yrs	5	2.0
No	26.2	3.9	20 – 24 yrs	82	32.8
'p'	**0.0101 Significant**		25 – 29 yrs	105	42.0
			>30yrs	58	39
			Total	250	100
			Range	**19 – 42 yrs**	
			Mean	**26.8 yrs**	



The mean age for GDM observed was 27.5 according to our study.

Table 2 : Gestational DM & Parity

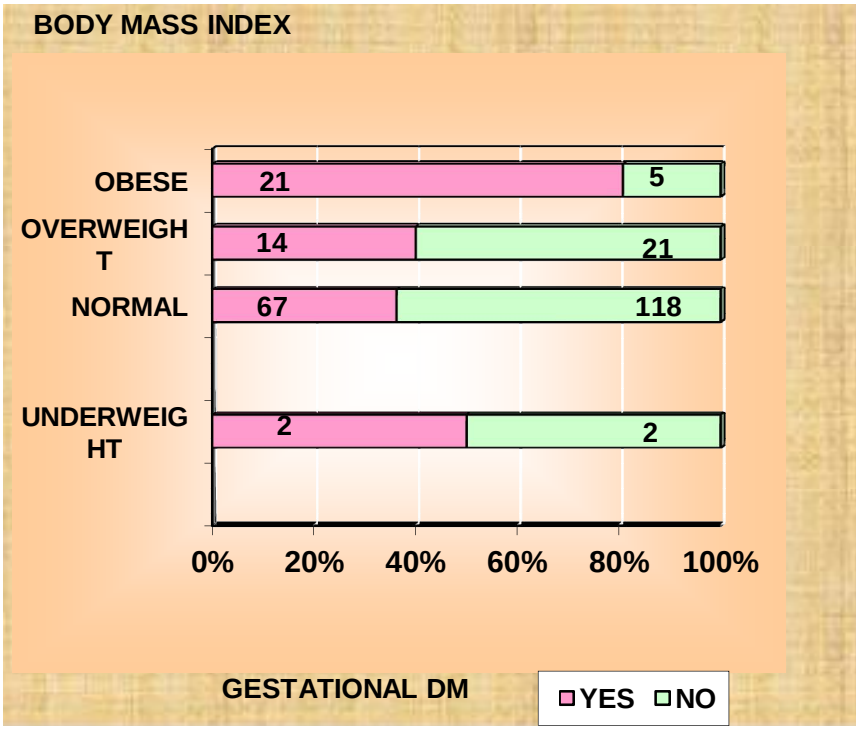
Gestational DM	Parity			
	Primi		Multi	
	N	%	N	%
Yes (104)	56	53.8	48	46.2
No (146)	67	45.9	79	54.1
'p'	0.2662 Not Significant			



In this study there was no significant correlation demonstrated between parity and GDM

Table 3 : Body Mass Index & Gestational DM

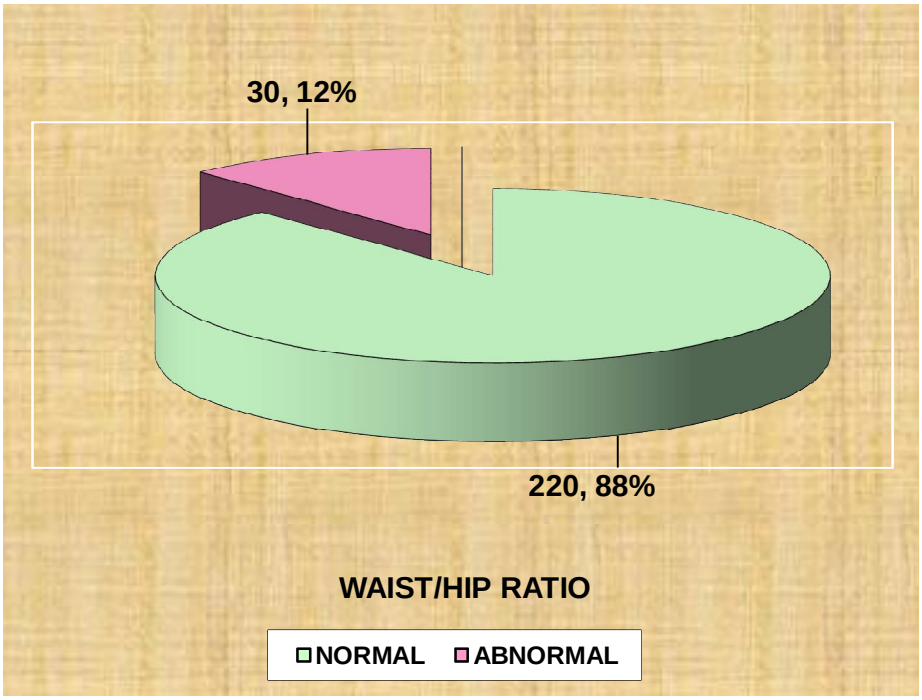
Body Mass Index	Gestational DM			
	Yes		No	
	N	%	N	%
Underweight (≤ 18.4)(4)	0	0	4	100.0
Normal (18.5 – 22.9) (185)	67	36.2	118	63.8
Overweight (23 – 24.9) (35)	21	60.0	14	40.0
Obese (≥ 25) (26)	21	80.8	5	19.2
'p'	**0.0064 Significant**			



80% Obese women and 60% overweight women developed GDM and showed a significant correlation of 'p' =.0064

Table 4 : Waist / Hip Ratio % and Gestational DM

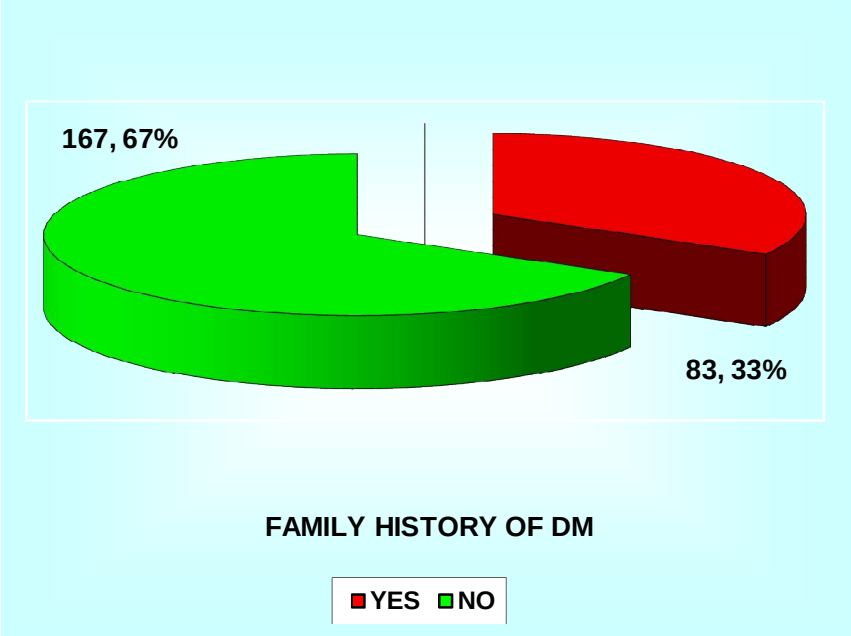
Waist / Hip Ratio %	**Gestational DM**			
	Yes		**No**	
	N	**%**	**N**	**%**
Normal (220)	82	37.2	137	62.3
Abnormal (30)	22	73.0	8	27.0
'p'	**0.0015 Significant**			



73% of the women with abnormal waist hip ratio developed GDM with significant correlation of 'p' =.0015

Table 5 : Family History of DM & Gestational DM

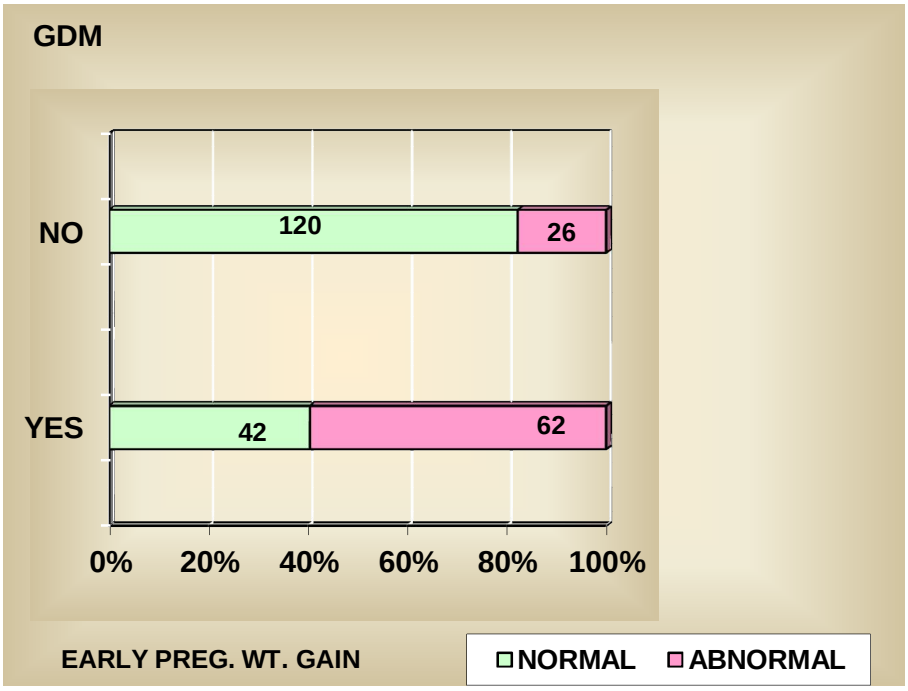
Family History of DM	Gestational DM			
	Yes		No	
	N	%	N	%
Yes (83)	68	56.6	15	43.4
No (167)	36	40.7	131	59.3
'p'	<0.0001 Significant			



56.6% of women with family h/o DM developed GDM with significant correlation of 'p' = <0.0001.

Table 6 : Gestational DM & Early Pregnancy Weight Gain

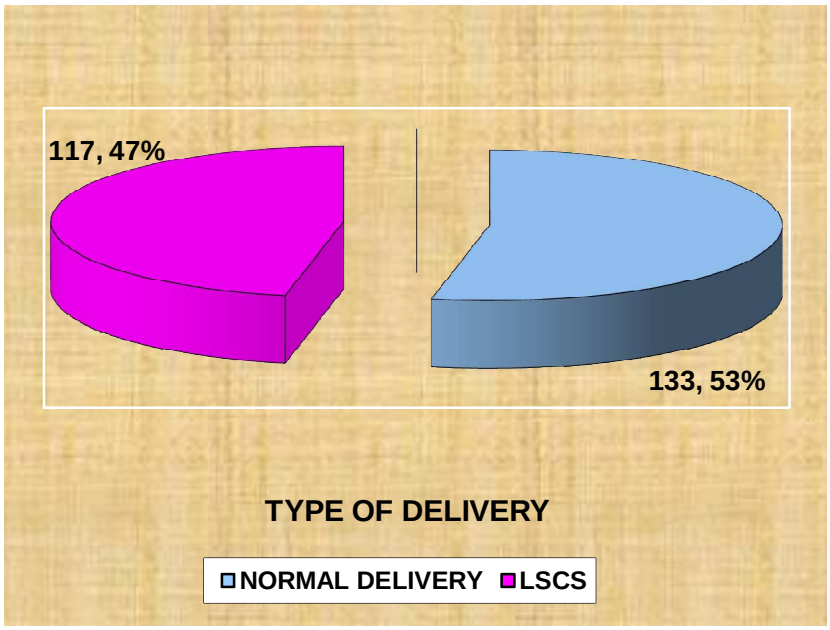
Gestational DM	Early Pregnancy Weight Gain			
	Normal		Abnormal	
	N	%	N	%
Yes (104)	42	40.4	62	59.6
No (146)	120	82.2	26	17.8
'p'	**< 0.0001 Significant**			



62 women with excessive early pregnancy weight gain developed GDM with Significant correlation of 'p' < .0001.

Table 7 : Gestational DM & Type of Delivery

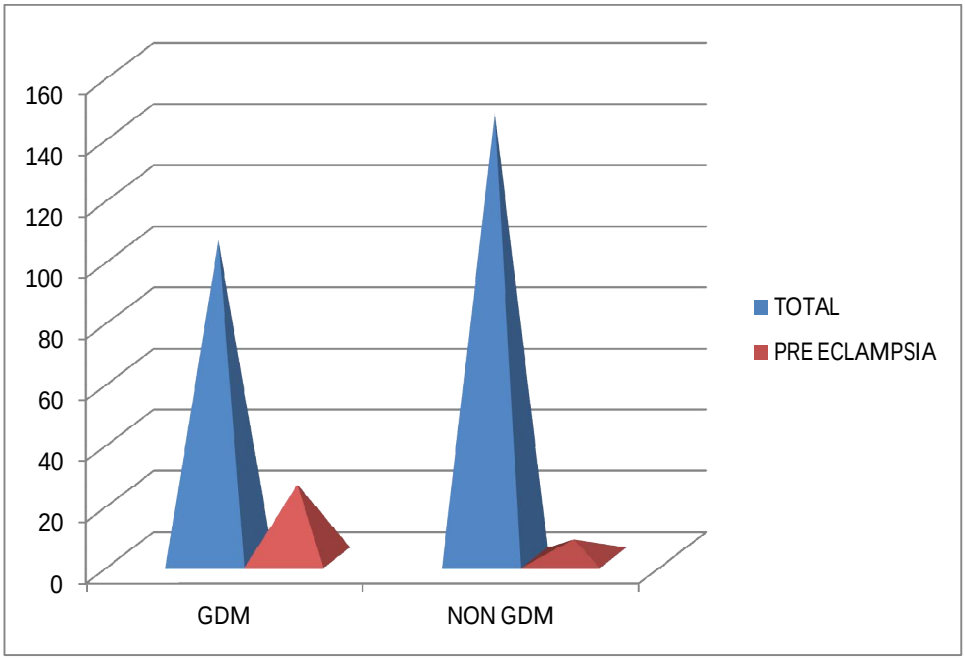
Gestational DM	Type of Delivery			
	Normal		LSCS	
	N	%	N	%
Yes (104)	39	37.5	65	62.5
No (146)	94	64.4	52	35.6
'p'	<0.0001 Significant			



Out of 104 women who developed GDM, 62.5% had operational delivery which showed significant correlation 'p' < .0001.

TABLE 8 :Pre eclampsia & GDM

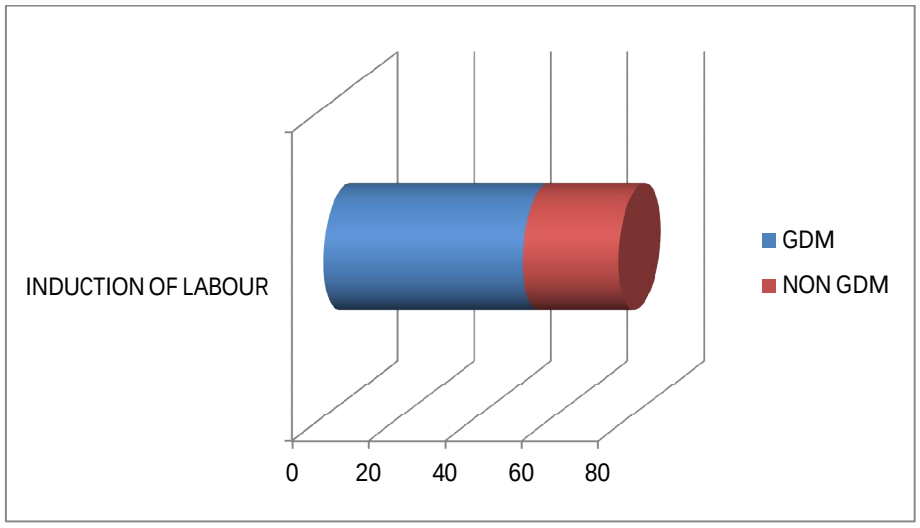
	TOTAL	PRE ECLAMPSIA
GDM	104	24 (80%)
NON GDM	146	6 (20%)



Our study observed that out of 30 women who developed preeclampsia , 24 were belonged to GDM group and 6 were non GDM.

TABLE 9 : INDUCTION OF LABOUR & GDM

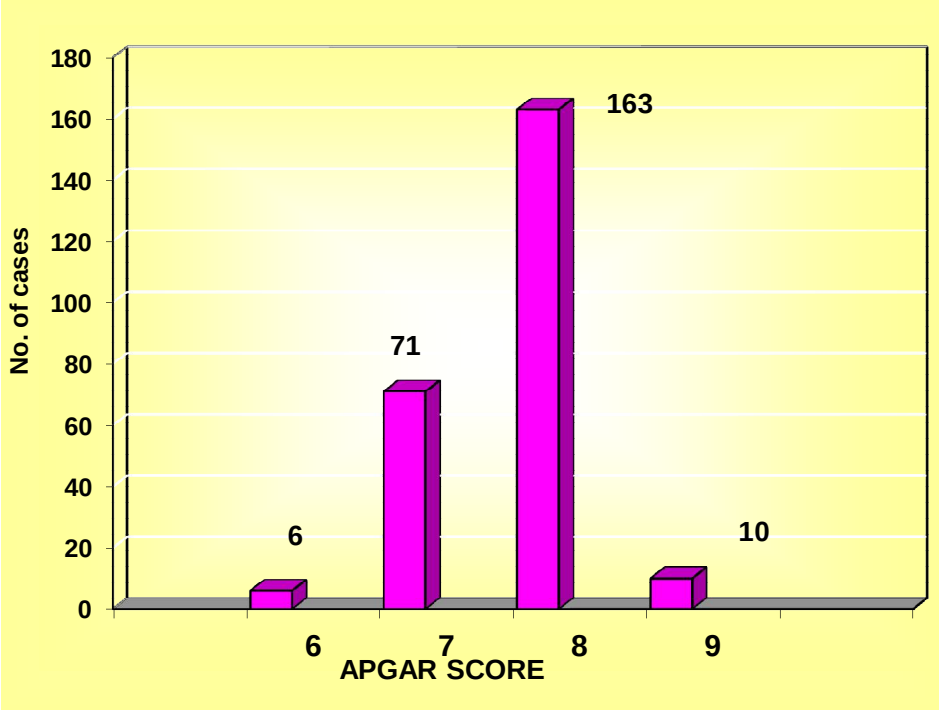
INDUCTION OF LABOUR	GDM	NON GDM
77	52(67%)	25(33%)



Out of 77 women who were induced for labour,52 belonged to GDM group and 25 belonged to non GDM group.

Table 10 : Gestational DM & APGAR Score

Gestational DM	APGAR Score	
	Mean	S.D.
Yes (104)	7.73	0.54
No (146)	7.69	0.61
'p'	0.6012 Not Significant	



Our study showed no significant association in relation to APGAR score.

TABLE 11:NEONATAL COMPLICATIONS:

Neonatal Complications	Cases	
	N	%
Large for Gestational age	20	8.0
Small for Gestational age	12	4.8
Hypoglycemia	51	20.4
Birth Asphyxia	19	7.6
Hyper Bilirubin	69	27.6

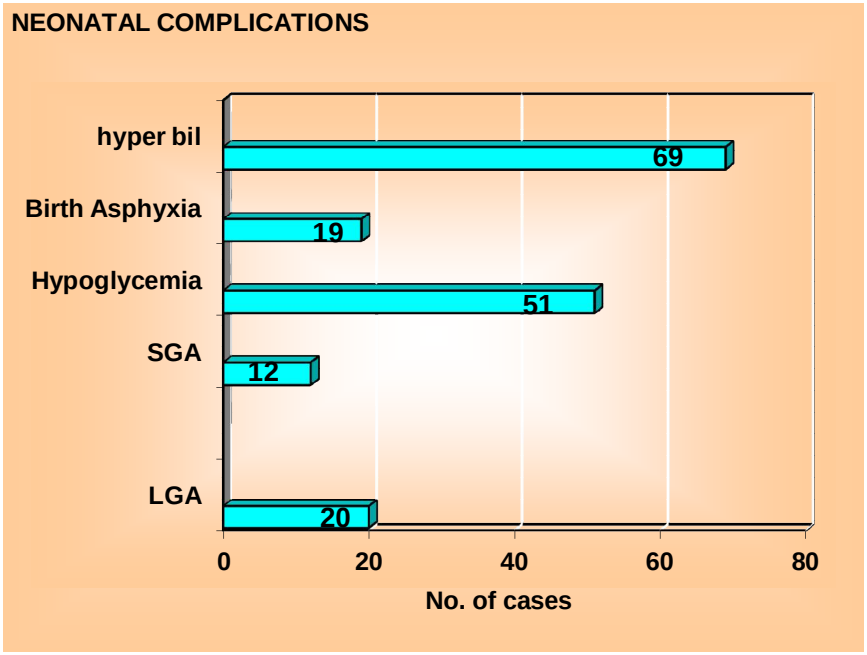
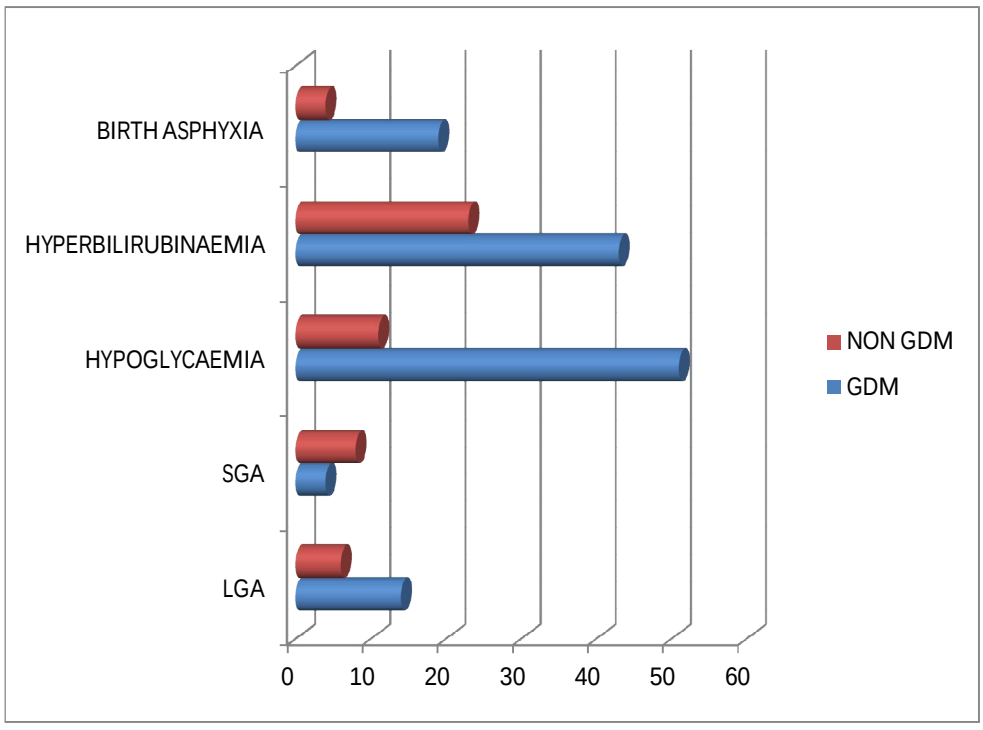


Table 12: NEONATAL COMPLICATIONS

NEONATAL COMPLICATIONS	TOTAL	GDM	NON GDM
LGA	20	14 (70%)	6 (30%)
SGA	12	4 (34%)	8(66%)
HYPOGLYCAEMIA	62	51(82%)	11(18%)
HYPERBILIRUBINAEMIA	69	43(62%)	23(38%)
BIRTH ASPHYXIA	23	19(82%)	4(18%)



DISCUSSION

In our study pregnant women were given 75 g oral glucose load irrespective of their fed state and 2hour post prandial glucose more than 140 mgs were interpreted as GDM. The explanation is that, “after a meal, women with normal glucose tolerance would be able to maintain normal glucose level despite glucose challenge because of brisk and adequate insulin secretion, Whereas, a woman with gestational diabetes mellitus who has reduced insulin response, her glucose level increases with a meal and with glucose challenge and the glycemic excursion exaggerates further[23,30]”. This cascading effect is convenient as this would not result in false positive diagnosis of GDM. Gestational weight gain more than 2 kg till 14 weeks[35] ,according to IOM[30,10] guidelines was taken as excessive early gestational weight gain.

In our study, we found that excessive early gestational weight gain in early pregnancy, particularly in the first trimester was associated with increased risk of GDM.

AGE:

In our study 58 women were in the age group >30 yrs, 31(53 %) of them developed GDM. And also in our study the mean age group is 27.5 yrs who developed GDM, which is in favour as the age advances the risk of developing GDM also increases (P=.0101)

Parity:

In our study 123 of the enrolled population were primis of which 56 women(53.8%) developed GDM and 127 of the population were multi paras of which 48 women (46.3%) developed GDM which has no significant correlation.(P=.2662)

BODY MASS INDEX:

In our study underweight women with BMI ≤ 18.4 were 4 and none of them developed GDM .Women with normal BMI between (18.5-22.9) were 185 and in this population 36.2% developed GDM ,63.8% did not develop GDM. Overweight women with BMI between (23-24.9) were 35,out of which 21(60%) developed GDM and 14 (40%) did not develop GDM. Obese women with BMI(≥25) were 26 out of which 21(80.8%) developed GDM and 5 (19.2%) did not develop GDM[40]. Carreno CA et al in 2012[29] and Hedderson MM et al in 2010[28,41] Valerie C Whiteman

et al [31], Alberico[42] reported similar findings that the correlation was greater among overweight and obese women.

STUDY (Et al)	P Value
Monique Hedderson[28]	<0.001
Valerie C Whiteman[31]	<0.002
Alberico[42]	<0.001
This study	0.0064

WAIST HIP RATIO:

Out of 250 enrolled women 30 women were with abnormal waist hip ratio and 22(73%) women developed GDM which has a significant correlation with P value of .0015. Sanmorn Basraon et al,[33] Leandro Branchtein et al,[43] Anju madhavan et al[36] al also showed similar correlation.

STUDY (Et al)	P Value
Sanmorn Basraon [33]	<0.02
Leandro Branchtein[43]	<0.01
Anju madhavan[36]	<0.003
This study	0.0015

FAMILY HISTORY OF DM:

In our study out of 250 women enrolled 68 (56.6 %)of the women with family history of DM developed GDM and 15 (43.4%) did not develop GDM .In our study the representative population showed significant correlation between GDM and family history of DM. Ravi Rethanakaran et al[3] study shows similar correlation.

STUDY (Et al)	P Value
Ravi Rethanakaran[3]	<0.02
This study	<0.0001

EARLY PREGNANCY WEIGHT GAIN:

In our study we realised that the correlation of GWG and risk of GDM was strongly attributable to increased weight gain in the first trimester[29]. "Excessive gestational weight gain in early pregnancy causes increased insulin resistance and leads to exhaustion of the B cell which reduces the capacity of B cells to secrete required insulin response to compensate for the insulin resistance induced by progression of pregnancy and development of GDM".

In our study 88 women with excessive early pregnancy weight gain , of which 62(59.6%) developed GDM and 26 (17.8%) did not develop GDM with P value of $\leq .0001$,which shows a strong association between excessive early pregnancy weight gain and risk of GDM. The following researchers showed similar association.

STUDY (Et al)	**P Value**
Hedderson[28]	<0.001
Carlos A Carreno[29]	<0.001
Aoife M Eagen[45]	<0.001
Morrisset As[46]	<0.002
Herring[36]	<0.001
Margerison	<0.002
Jeanine stein[25]	<0.001
Josefson J Metzger[18]	<0.03
This study	<0.0001

MATERNAL COMPLICATIONS

MODE OF DELIVERY:

In our study out of 104 women who developed GDM ,39 (37.5%) women delivered normally and 65 (62.5%) women delivered through LSCS.As GDM increases the risk of caesarean delivery, our study showed significant correlation with 'p' value of <0.0001

STUDY (Et al)	**'p' Value**
Cheng Yvonne [47]	<0.01
This study	<0.0001

INDUCTION OF LABOUR:

Out of 77 women in whom induction of labour was done with prostaglandin E2 gel, 52(67%)were women with GDM.

Pre eclampsia:

Our study observed that out of 30 women who developed preeclampsia, 24 (80%) were belonged to GDM group and 6 (20%) were non GDM.

POLYHYDROMINIOS:

In our study out of 104 women who developed GDM ,6 women developed polyhydramnios and 2 of them developed preterm labour due to pre mature rupture of membrane.

NEONATAL COMPLICATIONS

In our study,20 babies belonged to large for gestational age group with birthweight more than 3.5 kg in which 14(70%) babies were born to GDM mothers and 6(30%) were born to non GDM mothers. out of 12 small for gestational age babies 4(34%),belonged to GDM mothers and 8 (66%)belonged to non GDM mothers. 51(82%) babies born to GDM mothers developed hypoglycaemia and required NICU admissions. Out of 69 babies who developed hyperbilirubinaemia 43(62%) were born to GDM mothers and 23(38%) were born to non GDM mothers.19 babies developed birth asphyxia and got admitted in NICU.

SUMMARY

Gestational DM and Age:

This study shows the age distributions with high proportion between 25 -29 yrs and above 30 years are 58. 39% of women are above 30 yrs of age and in this age group 31(53%) women developed GDM. The mean age for GDM observed was 27.5 according to our study.

Gestational DM & Parity:

In this study 56(53.8%) were primi and 48(46.2%) were multi, who developed GDM, and there is no significant correlation between these two variables (P value .2662)

Body Mass Index & Gestational DM:

In this study, out of 26 obese women, 21(80%) pregnant women developed GDM and Out of 35 overweight women, 21(60%) developed GDM. In this study overweight and obese women demonstrated significant correlation for developing GDM with P value of .0064.

Waist / Hip Ratio % and Gestational DM:

In our study 37.2% of women developed GDM with normal waist hip ratio and 22 (73%) developed GDM with abnormal waist hip ratio with significant correlation of P =.0015

Family History of DM & Gestational DM:

In our study 56.6% of women with family history of DM demonstrated a significant association with regard to rate of GDM with significant correlation of P = <0.0001

Gestational DM & Early Pregnancy Weight Gain:

In this study, out of 88 women enlisted had excessive early gestational weight gain. 26(17.8%) normoglycaemic women, gained excessive weight during first trimester and 62(59.6%) of women with excessive weight gain developed GDM with significant correlation of P value < .0001.

Gestational DM & Type of Delivery:

In our study,Out of 104 women who developed GDM,65 (62.5%) women had operational delivery,39 (37.5%) had normal delivery with significant correlation of P value <0.0001

Gestational DM and Pre eclampsia:

Our study observed that out of 30 women who developed preeclampsia, 24 (80%) were belonged to GDM group and 6 (20%) were non GDM.

Gestational DM and Induction of labour:

In this study,Out of 77 women who were induced for labour,52(67%) belonged to GDM group and 25(33%) belonged to non GDM group.

Gestational DM & APGAR Score:

Our study showed no significant correlation with apgar score with insignificant P value of .6012

Gestational DM and Neonatal complications:

In our study, 20 babies belonged to **large for gestational age** group with birth weight more than 3.5 kg in which 14(70%) babies were born to GDM mothers and 6(30%) were born to non GDM mothers. Out of 12 **small for gestational age** babies 4(34%) belonged to GDM mothers and 8(66%) belonged to non GDM mothers.51 (82%) babies born to GDM mothers developed **hypoglycemia** and required NICU admissions. Out of 69 babies who developed **hyperbilirubinaemia** 43

(62%)were born to GDM mothers and 23(38%) were born to non GDM mothers.19(82%) babies developed **birth asphyxia** and got admitted in NICU and 4(18%) babies were belonged to non GDM group.

CONCLUSION

Excessive early pregnancy weight gain, primarily in the first trimester may increase a women's risk of GDM and it's related maternal and fetal complications. Excessive gestational weight gain can be represented as a modifiable risk factor occurring during early pregnancy[6]. Lifestyle modifications like simple excercises (walking) and dietary modifications can prevent GDM and it's related complications. This costless early intervention can make healthy future generations[54].

Monique M. Hedderson et al[28], Herring et al in 2009[26], Margerison et al in 2010, Jeannine stein in feb 2010[25], Sallyn Boyles investigated the correlation between excessive early gestational weight gain and the risk of gestational Diabetes mellitus and they demonstrated the stronger correlation between weight gain during the first trimester and GDM.They also demonstrated there was no association between weight gain during the second trimester and getstational diabetes mellitus.

Hedderson said "As all pregnancies progress an increase in insulin resistance occurs" due to decreased β cell capacity caused by excessive weight gain". And also prepregnant obesity could be a high risk factor for developing GDM that any increased weight gain during first trimester

will thrust them into GDM. So early interventions of life style modifications can have a big impact on reducing GDM and it's related complications.

Cuilin Zhang and Yining et al[51] in may 2011 studied the effect of dietary modification and excercises on the risk of developing GDM. The collected datas were investigated on the basis of Nurses' Health Study II. They suggested that "high fibre intake and polyunsaturated fat intake a may guard against glucose intolerance during pregnancy, and increased intake of saturated fat may be harmful". In an another prospective observational study of pregnant women, decreased vitamin C and vitamin D concentrations in the plasma were significantly associated with increased risk of GDM. They also observed 30 minutes of brisk walking reduces the risk of developing GDM.

Thobias DK et al, [52] in 2014 quantified the correlation between combination of healthy lifestyle modifications before and during pregnancy (recommended weight, dietary habits, daily exercise, and no smoking) and the risk of gestational diabetes. They concluded that, "adopting a healthy lifestyle in the period before and during pregnancy is associated with a substantially lower risk of gestational diabetes".

From our study, we conclude that excessive early pregnancy weight gain especially in the first trimester correlates with developing the complication of GDM. Thus preventing excessive early pregnancy weight gain in the first trimester which can be modified by lifestyle changes and simple exercises which is a **costless intervention** supported by the studies of researchers may decrease maternal and neonatal morbidity effectively. So here are some advises for the pregnant women to reduce excessive weight gain during pregnancy.

ADVISE FOR CONTROLLING EXCESSIVE WEIGHT GAIN IN PREGNANCY

- Consuming regular meals and small healthy snacks between meals (3 meals and 3 snacks), will also help controlling pregnancy related nausea.
- Avoiding sweets and sweetened drinks
- Consuming only about 100 to 300 calories a day
- Reducing fat intake
- Drinking less fruit juice and eat more whole fruit
- Consuming food high in fiber
- Decrease white flour products like white bread and cereals
- Consuming whole grain foods
- Avoiding fried foods
- Choosing grilled or baked foods
- Meals should not be skipped
- Being active
- 30 min of exercise on most days of the week(swimming, brisk walking, indoor stationary cycling, and low-impact aerobics)

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LIST OF ABBREVIATION

1.SMBG	-	SELF MONITORING - BLOOD GLUCOSE
2.GDM	-	GESTATIONAL DIABETES MELLITUS
3.HPL	-	HUMAN PLACENTAL LACTOGEN
4.DM	-	DIABETES MELLITUS
5.IGT	-	IMPAIRED GLUCOSE TOLERANCE
6.IOM	-	INSTITUTE OF MEDICINE
7.WHO	-	WORLD HEALTH ORGANISATION
8.OGTT	-	ORAL GLUCOSE TOLERANCE TEST
9.OGCT	-	ORAL GLUCOSE CHALLENGE TEST
10.IADPSG	-	THE INTERNATIONAL ASSOCIATION OF THE DIABETES AND PREGNANCY STUDY GROUPS
11.DIPSI	-	DIABETES IN PREGNANCY STUDY GROUP INDIA
12.GWG	-	GESTATIONAL WEIGHT GAIN
13.PPWR	-	POSTPARTUM WEIGHT RETENTION
14.LGA	-	LARGE FOR GESTATIONAL AGE
15.SGA	-	SMALL FOR GESTATIONAL AGE
16.BMI	-	BODY MASS INDEX
17.WHR	-	WAIST HIP RATIO
18.MNT	-	MEDICAL NUTRITION THERAPY
19.FPG	-	FASTING PLASMA GLUCOSE
20.GOD-POD	-	GLUCOSE OXIDASE- PEROXIDASE
21.MPG	-	MEAN PLASMA GLUCOSE
22.IR	-	INSULIN RESISTANCE
23.AGT	-	ABNORMAL GLUCOSE TOLERANCE

PROFORMA

- NAME: LMP:
- AGE : EDD:
- SEX :
- SOCIO ECONOMIC STATUS HEIGHT:
- PARITY: PREPREGNANCY WEIGHT:

BODY MASS INDEX :

- GESTATIONAL AGE :
- MENSTRUAL HISTORY
- MARITAL HISTORY
- OBSTETRIC HISTORY
- PAST HISTORY
- FAMILY HISTORY
- GENERAL EXAMINATION
- PER ABDOMEN EXAMINATION:
- **WEIGHT GAIN** **1st TM** **2nd TM** **3rd TM**
- **OGCT VALUE**

PREGNANT WOMEN

GROUP A GROUP B

NORMAL WEIGHT GAIN

GDM NO GDM

EXCESSIVE WEIGHT GAIN

GDM NO GDM

- MATERNAL OUTCOME

GDM

NORMAL DELIVERY

INDUCTION OF LABOUR

CAESAREAN SECTION

PRE ECLAMPSIA

- FETAL OUTCOME

LGA

SGA

HYPOGLYCAEMIA

BIRTH ASPHYXIA

HYPERBILIRUBINAEMIA

RESULTS

Statistical Analysis Plan

Results will be assessed and tabulated using SPSS EXCEL SOFTWARE System.

Signature of the investigator:

Signature of the Patient:

Witness:

PATIENT CONSENT FORM

STUDY TITLE: CORRELATION OF EXCESSIVE EARLY PREGNANCY WEIGHT GAIN AND RISK OF GESTATIONAL DIABETES MELLITUS

Study center: ESI PGIMSR

Participant name : age: sex: IP No:

I confirm that I have understood the purpose of this study. I have the opportunity to clarify my queries and doubts and they have been answered to my satisfaction.

I understand that my parcipation is purely voluntary and I am free to withdraw any time without giving reasons

I have understood that the investigator ,regulatory authorities and the ethics committee will have access to my health records both in respect to current study and any further research that may be conducted in relation to it ,even if I decide to withdraw from the study.i have understood that my identity will not be revealed in anyway and information related 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.

Without any compulsion I am willing to give consent for the participation of myself in this study.

DATE:

PLACE:

Signature of Patient

Signature of the investigator:

Name of the investigator

ஆய்வுக்கான ஒப்புதல் படிவம்

மருத்துவர் தனது முதுநிலை பட்ட படிப்பு முழுமை பெறுவதற்காக மேற்கொள்ளும் பிரசவ கால எடை அதிகரிப்பு பற்றி எனக்கு என் தாய் மொழியில் விளக்கப்பட்டது.இந்த ஆய்வில் பிரசவ கால அதிக எடை அதிகரிப்பால் நீரிழிவு நோய் ஏற்பட வாய்ப்பு உள்ளதை பற்றி அறியப்பட உள்ளது .

இந்த ஆய்வின் அனைத்து அம்சங்களும் விளக்கப்பட்டது .இதில் என் முழு விருப்பத்துடன் கலந்து கொள்கிறேன் .எனினும் எந்த நேரத்திலும் எந்த காரணமுமின்றி இந்த ஆய்விலிருந்து விலகி கொள்ள எனக்கு முழு உரிமை உள்ளது .இதனால் எனது நலனில் எந்த பாதிப்பும் ஏற்படாது என்று உறுதி அளிக்கப்படுகிறது .மேற்கூறிய உறுதி மொழிகள் யாவும் பின்பற்றப்படும் பட்சத்தில் நான் இந்த ஆய்வு கட்டுரைக்கு என்னுடைய விவரங்களை அளிக்க முன் வருகிறேன்.இதற்க்கு முழு மனதுடன் என்னுடைய ஒப்புதலை தந்து இந்த ஆய்வு அறிக்கையில் பங்கேற்கிறேன் .

இப்படிக்கு ,

(கையொப்பம்)

no	name	age	parity	GA	HT	WT	BMI	W/H %	F/H/ DM	HB	RFT	LFT	HBA1C	1TM	2TM	3TM	R.FAC	p.e	IOL	ND	LSCS	LGA	SGA	HYPO. GLY	BA	HYP .BIL	B.WT	APG	pre. preg	till 14 wks	EP Wt. GAIN	t.wg
1	bharathi	19	primi	8w3d	153	43	18.4	74	1	11.3	n	n	5.05	122	131	155.9	gdm/mp	1	1	0	2/fd	0	0	0	0	0	2.6	8	43	43.5	0.5	50
2	madhudevi	26	g2p1l1	7w4d	153	45	19.2	72	1	9.8	n	n	4.8	98	184	122	gdm/mp	0	0	1	0	0	0	1	0	0	3.6	8	45	47.5	2.5	54
3	nancy	27	primi	9w	153	60	25.6	84	1	11.3	n	n	5.1	108	152	120	gdm/ins	1	1	0	2/fd	1	0	1	1	0	3.74	7	60	64	4	72
4	muniammal	28	primi	7w6d	157	55	22.3	72	0	11.7	n	n	5.3	98	148	140	gdm/mp	0	0	0	2/b.in l	0	0	0	0	0	2.74	8	55	56	1	63
5	parameshwari	27	g3p2l1	8w1d	152	61.5	26.6	70	1	11.4	n	n	5.2	102	154	123	gdm/mp	1	0	0	2/cpd	0	0	0	0	1	3.3	8	61.5	63.6	2.1	70
6	vasanthakumari	23	g3p2l1	7w4d	145	45	21.4	72	1	10.7	n	n	5.4	84	162	112	gdm/poly	0	0	0	2/prom	0	0	1	0	0	3.24	7	45	46	1	54
7	lavanya	24	primi	8w2d	148	53	24.2	82	1	11	n	n	4.8	122	164	104	gdm/mp	0	1	1	0	0	0	0	0	1	2.9	8	53	55.5	2.5	63
8	sumathi	29	g2p1l1	9w3d	160	50	19.5	68	1	9.8	n	n	5.1	123	158	112	gdm/mp	0	0	1	0	0	0	0	0	0	2.96	8	50	51	1	59
9	dhanalaxmi	32	g2p1l1	8w6d	154	56	23.6	71	0	8.8	n	n	6.0	87	174	82	gdm/mp	1	0	0	2/pls	0	0	0	0	0	3.36	7	56	58.2	2.2	74
10	tamilselvi	32	g2p1l1	9w2d	158	52	20.8	76	1	12	n	n	5.2	130	130	152	gdm/mp	1	1	1	0	0	1	0	0	0	2.3	8	52	54.5	2.5	60
11	veronica	28	g2a1	10w	156	47	19.3	69	1	9.5	n	n	5.3	73	148	82	gdm/mp	0	0	0	0	0	0	0	0	1	2.92	8	47	48	1	63
12	kalaiarasi	24	primi	11w	158	52	20.8	78	1	11.6	n	n	5.6	113	120	167	gdm/ins	1	1	0	2/fi	0	0	1	0	1	2.96	8	52	55	3	61
13	gunasundari	27	g2p1l1	6w2d	156	48	19.7	74	0	11.9	n	n	5.8	84	135	106	0	0	0	1	0	0	0	0	0	1	2.76	8	48	48.5	0.5	58
14	kannagi	33	primi	7w4d	160	52	20.3	76	1	10.9	n	n	4.7	86	148	114	gdm/mp	0	1	1	0	0	0	0	1	0	2.85	6	52	53	1	61
15	vidhya	25	g3p1l1a1	8w3d	162	58	22.1	84	1	10	n	n	5.7	105	162	142	gdm/ins	0	1	1	0	0	0	1	0	0	3.1	8	58	60.2	2.2	69
16	divya	24	primi	9w2d	158	52	20.8	74	1	14.9	n	n	5.8	106	144	156	gdm/ins	1	0	0	2/pcs	1	0	1	0	0	3.56	8	52	52.5	0.5	62
17	vedavalli	28	g2p1l1	8w4d	162	51	19.6	76	0	12.3	n	n	5.6	124	138	134	0	0	0	1	0	0	0	1	0	0	2.9	8	51	51.5	0.5	62
18	santhana roselin	31	g3p2l2	9w4d	160	58	22.7	78	0	10	n	n	5.7	122	176	138	gdm/ins	0	1	0	2/fd	0	0	0	0	1	3.3	8	58	58	0	69
19	arokiamary	33	g2p1l1	7w3d	148	54	24.7	82	1	11.9	n	n	5.5	106	141	164	gdm/ins	0	0	0	2/cpd	1	0	0	0	1	3.1	8	54	57	3	64
20	saraswathi	24	primi	8w3d	157	67	27.2	84	1	11	n	n	5.2	108	112	150	gdm/mp	1	1	0	2/fd	0	0	0	0	0	2.8	8	67	68.8	1.8	78
21	shanthi	30	primi	9w4d	154	62	26.1	73	1	11.9	n	n	5.5	106	178	142	gdm/ins	1	0	0	2/b.baby	1	0	1	0	1	3.8	8	62	64.3	2.3	72
22	manjula	28	g2p1l1	14w3d	151	70	30.7	86	1	12	n	n	5.8	134	189	144	gdm/poly	0	0	0	2/fd	1	0	1	0	1	3.6	8	70	74	4	82
23	anandhi	23	g2a1	16w	168	70	24.8	79	1	11.1	n	n	5.6	120	147	120	gdm/mp	1	1	1	0	0	1	0	0	1	1.9	7	70	74	4	82
24	muthulaxmi	29	primi	12w	160	52	20.3	68	0	10.6	n	n	5.4	86	124	90	0	0	0	1	0	0	0	0	0	0	3	8	52	52.5	0.5	62
25	bhuvaneshwari	29	primi	13w	162	57	21.7	69	0	11	n	n	5.3	97	162	98	gdm/poly	0	0	0	2/fd	1	0	0	0	1	3.4	8	57	59.4	2.4	65
26	janet	30	g2p1l1	16w	155	50	20.8	70	1	10	n	n	4.8	96	161	145	gdm/ins	1	1	1	0	0	0	0	0	0	3	8	50	52.3	2.3	61
27	kavitha	21	primi	12w	151	60	26.3	69	1	11	n	n	5.2	123	142	121	gdm /mp	0	0	0	2/fd	0	0	0	1	0	2.8	8	60	60.5	0.5	70
28	sangeetha	30	primi	14w3d	156	58	23.8	72	0	10.8	n	n	5.4	132	122	124	0	0	0	1	0	0	0	0	0	1	2.8	8	58	58.5	0.5	68
29	alamelu	24	primi	12w4d	154	46	19.4	68	1	12	n	n	6	124	164	126	gdm/mp	0	1	1	0	0	0	1	0	0	2.6	7	46	47.5	1.5	55
30	shamugapriya	20	primi	13w3d	156	58	23.8	76	1	11	n	n	5.4	101	132	128	0	0	0	0	2/fd	0	0	0	1	0	3.37	8	58	58.5	0.5	65
31	padmavathi	21	primi	12w	153	41	17.6	64	0	12	n	n	4.8	114	156	124	gdm/mp	0	0	0	2/cpd	0	0	0	0	0	2.56	8	41	43.2	2.2	48
32	sivagami	34	g4p1l1a2	14w3d	150	51	22.7	74	1	11	n	n	5.6	103	112	138	0	0	0	0	2/fd	0	0	1	0	0	2.68	9	51	52	1	60
33	kala	25	primi	16w	154	54	22.8	74	1	10.5	n	n	5.2	112	156	114	gdm/mp	1	0	1	0	0	0	1	0	1	2.8	7	54	56.3	2.3	60
34	sri kanya sarasu	32	G2P1L1	8w	159	50	19.8	68	0	10	n	n	5.8	89	145	132	gdm/mp	0	1	0	2/cpd	0	0	0	0	1	2.9	8	50	53	3	61
35	venda	20	G2P1L1	8w3d	159	59	23.3	72	0	10.4	n	n	5.4	108	124	123	0	0	0	1	0	0	0	0	0	1	2.45	8	59	59.5	0.5	67
36	veena	30	G3P1L1A1	7w4d	146	45	21.1	65	1	11	n	n	5.7	92	126	152	gdm/mp	1	1	0	2/cpd	0	0	1	0	0	3.1	8	45	47.5	2.5	55
37	udayadeepika	24	primi	11w	172	56	18.9	78	0	9.8	n	n	5.6	98	148	126	gdm/mp	0	1	0	2/fd	0	0	0	0	1	3.6	8	56	59	3	65
38	gangadevi	30	primi	12w	140	51	26	68	1	10.2	n	n	5.4	102	145	168	gdm/ins	1	1	0	2/fd/bb	1	0	1	0	1	3.67	7	51	54	3	61
39	nirmala	21	G4P3L1A0	10w1d	158	52	20.8	70	1	12	n	n	4.8	112	123	156	gdm/mp	0	0	1	0	0	0	0	0	0	2.86	8	52	53	1	61
40	revathy	30	G3P1L1A1	12	160	58	22.7	81	1	11	n	n	5.2	102	143	148	gdm/mp	0	1	0	2/cpd	0	0	1	0	1	2.88	8	58	60.3	2.3	67
41	muthumari	27	primi	8w3d	156	52	21.4	73	1	10.3	n	n	4.9	86	167	132	gdm/mp	0	0	0	2/fd	0	0	1	1	0	2.5	6	52	52.5	0.5	62
42	sangeetha	24	G3P1L1A1	9w4d	152	86	37.2	90	1	11	n	n	5.6	104	192	147	gdm/ins	1	1	0	2/fi	0	0	0	0	1	3.6	8	58	60.5	2.5	80
43	manimegalai	36	G2P1L1	10w1d	158	74	29.6	78	0	12	n	n	5.2	88	122	134	0	0	0	0	2/fd	0	0	1	0	0	2.8	8	74	74.5	0.5	85
44	ambika	25	primi	10w3d	160	65	25.4	74	0	12	n	n	5.4	124	166	126	gdm/mp	0	0	1	0	0	0	0	0	1	2.7	8	65	67.2	2.2	75
45	suganya	32	G2P1L1	11w2d	158	60	24	72	1	10.6	n	n	5.6	112	158	132	gdm/poly	1	0	0	2/cpd	0	0	0	1	0	2.8	8	60	62.5	2.5	70
46	muthulaxmi	32	G3P1L1A1	11w4d	162	58	22.1	78	1	9.8	n	n	5.4	130	178	124	gdm/ins	0	1	0	2/fd	0	0	1	0	1	3.4	8	58	59.5	1.5	68

47	selvi	25	primi	8w3d	151	54	23.8	70	1	10.3	n	n	5.2	124	148	156	gdm/mp	0	1	0	2/fd	0	0	0	0	1	3.2	8	54	56.2	2.2	65
48	deepa	30	primi	9w3d	152	50	21.6	76	1	11	n	n	5.3	78	156	123	gdm/mp	0	0	1	0	0	0	1	1	0	3.5	7	50	51	1	61
49	sangeetha	35	primi	7w4d	156	52	21.4	77	1	10.2	n	n	4.9	98	168	128	gdm/mp	1	1	0	2/fi	0	0	0	0	0	3.4	8	52	54.8	2.8	62
50	sudha	19	primi	8w4d	153	45	19.2	76	1	10.6	n	n	5	132	158	146	gdm/ins	0	0	1	0	0	0	1	0	1	3	8	45	47.5	2.5	55
51	krishnaveni	29	G3P1L1A1	8w4d	154	60	25.3	72	0	12	n	n	5.3	124	120	124	0	0	1	0	2/fd	0	0	1	0	0	2.9	8	60	61.5	1.5	70
52	bhuvaneshwari	29	G3P1L1A1	9w2d	148	44	20.1	69	1	10.2	n	n	5.3	132	146	145	gdm/mp	0	1	0	2/fd	0	0	0	0	0	2.8	8	44	45.7	1.7	55
53	lavanya	27	G3P1L1A1	8w4d	153	48.5	20.7	70	1	9.6	n	n	6	88	156	123	gdm/mp	0	1	1	0	0	0	1	0	1	2.6	8	48.5	49	0.5	56
54	vijayalaxmi	28	primi	9w3d	148	60	27.6	72	0	11	n	n	5.2	106	152	124	gdm/mp	1	0	0	2/fd	0	0	1	0	0	2.6	8	60	61.7	1.7	70
55	sakila	28	primi	10w4d	166	83	30.1	82	1	12	n	n	5.8	98	197	152	gdm/ins	0	0	1	0	1	0	1	0	0	3.62	7	83	86	3	92
56	laxmi	29	primi	11w2d	158	50	20	75	1	11.2	n	n	5.4	86	156	146	gdm/ins	1	1	0	2/fi	0	0	1	0	1	3.2	8	50	52.5	2.5	62
57	stellamary	21	primi	10w3d	161	50	19.3	76	1	12	n	n	5.3	124	134	167	gdm/ins	0	0	1	0	0	0	0	0	0	3.1	8	50	51	1	60
58	bhuvaneshwari	29	G2P1L1	12w	155	72	30	75	0	11	n	n	5.4	132	112	126	0	0	1	1	0	0	0	0	0	1	2.9	8	72	71.5	-0.5	80
59	amudhavalli	24	primi	12w4d	156	54	22.2	72	0	11	n	n	5.5	128	199	178	gdm/ins	0	1	0	2/fd	0	0	1	0	0	3.5	8	54	57.5	3.5	61
60	anandhi	23	G2A1	13w6d	153	53	22.6	78	1	10.6	n	n	5.3	124	163	145	gdm/mp	0	0	1	0	0	0	1	0	0	1.9	8	56	58.4	2.4	65
61	sulochanapriya	27	g2p1l1	14w3d	156	54	22.2	75	1	11.2	n	n	2	124	192	138	gdm/ins	0	1	0	2/fd	0	0	0	0	0	3.2	9	56	57.5	1.5	66
62	selvi	28	g3p1l1a1	15w	162	68	25.9	76	0	10.3	n	n	4.8	122	156	142	gdm/mp	0	0	0	2/fd	0	0	0	0	1	2.88	7	68	69.7	1.7	75
63	padmavathi	24	primi	14w	154	63	26.6	74	1	9.8	n	n	5	123	192	124	gdm/ins	0	1	0	2/c.pp	0	0	1	0	0	2.43	7	58	57	-1	67
64	subha	25	primi	12w3d	152	53	22.9	72	1	10.1	n	n	5	118	154	126	gdm/ins	0	1	1	0	0	0	1	0	0	3.29	9	52	54.5	2.5	63
65	jency	25	primi	13w3d	168	73	25.9	81	0	10.3	n	n	5.7	123	134	158	gdm/poly	0	0	0	2/cpd	0	0	0	0	1	3.33	8	54	56.5	2.5	65
66	parimala	27	g2p1l1	14w2d	157	54	21.9	76	1	12	n	n	5.5	133	156	124	gdm/mp	1	0	1	0	0	0	0	0	0	2.75	7	57	59.7	2.7	66
67	abithabee	37	g3p1l1a1	12w4d	156	56	23	80	1	11.3	n	n	5.3	123	120	155	gdm/mp	0	1	0	2/fi	0	1	0	0	0	2.3	8	58	58	0	67
68	mahalaxmi	27	g2p1l1	16w	153	57	24.3	83	0	10.8	n	n	5.2	124	126	172	gdm/mp	0	1	1	0	0	0	0	0	0	2.9	7	56	58.6	2.6	66
69	manjula	33	g2p1l1	14w	154	52	21.9	76	1	10	n	n	5.1	126	135	167	gdm/mp	0	0	0	2/fd	0	0	0	1	0	2.96	8	54	53	-1	62
70	jayalaxmi	22	primi	12w	153	51	21.8	78	1	10.2	n	n	5.4	121	153	138	gdm/mp	0	1	1	0	0	0	0	0	1	2.7	8	52	54.5	2.5	62
71	jagadeeswari	19	primi	11w2d	156	52	21.4	62	0	11	n	n	5.4	127	156	123	gdm/mp	0	0	0	2/fd	0	0	0	0	1	2.97	8	53	53.5	0.5	61
72	ilavarasi	35	e.primi	9w3d	153	49	20.9	65	1	10.8	n	n	5.6	112	156	138	gdm/mp	0	1	0	2/fi	0	0	1	0	0	2.4	8	49	51.3	2.3	59
73	vasanthi	24	primi	10w4d	158	53	21.2	70	0	9.8	n	n	5.2	145	158	140	gdm/ins	0	1	1	0	0	0	0	0	1	2.7	7	51	52.5	1.5	61
74	sangeetha	28	primi	10w1d	154	60	25.3	72	1	10.1	n	n	5.1	123	156	138	gdm/mp	0	0	1	0	0	0	0	0	0	3	8	60	61.8	1.8	71
75	jagadeeswari	33	g2p1l1	9w3d	150	52	23.1	71	0	11	n	n	5.6	148	132	138	gdm/mp	0	0	0	2/cpd	0	0	0	0	0	2.3	8	52	51	-1	61
76	sakila	33	primi	9w4d	152	54	23.4	73	1	11	n	n	5.4	124	156	132	gdm/mp	1	1	0	2/fd	0	0	1	0	1	2.5	8	52	54.4	2.4	64
77	vijayalaxmi	29	g3p1l0	9w6d	156	52	21.4	74	0	10	n	n	5.2	123	168	142	gdm/ins	0	0	0	2/cpd	0	0	1	0	1	2.5	7	52	52.5	0.5	62
78	sangeetha	23	g2p1l1	10	157	54	21.9	75	1	9.8	n	n	5.1	122	146	141	gdm/poly	0	0	1	0	0	0	0	0	0	2.8	7	54	56.2	2.2	63
79	vasanthalaxmi	29	primi	10w2d	154	48	20.2	69	0	10.1	n	n	4.9	124	152	132	gdm/mp	0	1	0	2/fi	0	0	1	0	0	3.23	8	52	54.5	2.5	58
80	vijayalaxmi	23	primi	8w4d	156	52	21.4	74	1	11.1	n	n	4.8	126	148	136	gdm/ins	0	1	0	2/fd	0	0	0	0	0	3.04	7	52	52.5	0.5	62
81	anandhi	23	g2a1	7w4d	152	48	20.8	73	0	10.2	n	n	4.3	124	112	136	0	0	0	1	0	0	1	1	0	1	1.9	8	48	49	1	52
82	shobana	25	primi	8w3d	148	46	21	71	1	11.2	n	n	4.5	122	164	144	gdm/mp	0	0	0	2/fi	1	0	1	0	0	3.5	8	46	48.5	2.5	57
83	shakila	37	primi	9w4d	153	45	19.2	68	0	10.8	n	n	5.3	132	167	145	gdm/ins	0	0	1	0	0	0	0	0	1	2.9	7	45	47.2	2.2	57
84	shanawaz	28	g3p2l2	8w2d	156	53	21.8	70	1	11.2	n	n	5.4	123	156	138	gdm/ins	0	1	0	2/pcs	0	0	0	0	0	3.1	8	53	54	1	62
85	nithya	27	primi	8w4d	158	54	21.6	71	0	11	n	n	5.2	122	158	123	gdm/mp	0	0	0	2/oligo	0	0	0	1	0	2.2	8	52	54.5	2.5	64
86	saraswathy	42	g4p2l0a1	8w2d	154	52	21.9	74	0	12	n	n	5.1	123	124	134	0	0	0	0	2/pcs	0	1	1	1	1	1.7	7	52	53	1	61
87	ilavarasi	35	primi	9w3d	148	52	23.7	80.5	0	11	n	n	5.5	112	154	123	gdm/mp	0	1	0	2/fi	0	0	0	0	0	2.4	8	52	52	0	63
88	vasanthi	24	primi	7w4d	150	48	21.3	74	1	10.2	n	n	5.3	108	167	142	gdm/ins	0	0	1	0	0	0	0	0	0	2.7	7	48	50.5	2.5	58
89	bhavani	28	primi	8w4d	148	54	24.7	81	0	10	n	n	5.2	110	156	138	0	0	1	0	2/fi	0	1	0	0	0	2.2	8	54	54.5	0.5	64
90	deepa	26	primi	9w	156	54	22.2	74	1	11	n	n	4.9	110	114	156	gdm/ins	0	1	0	2/fd	0	0	0	0	0	3.29	8	54	54.5	0.5	63
91	pavithra	27	primi	8w4d	149	45	20.3	69	1	10.1	n	n	4.8	108	152	124	gdm/mp	0	0	0	2/fd	0	0	0	0	0	2.66	8	45	47.4	2.4	56
92	saraswathi	32	g2p1l1	9w1d	152	49	21.2	70	0	9.8	n	n	5.1	110	132	122	0	0	1	1	0	0	0	0	0	1	2.8	8	49	49.5	0.5	58
93	kavitha	31	g2p1l0	10w	154	51	21.5	72	1	10.2	n	n	5.4	108	152	124	gdm/ins	0	0	0	2/fi	0	0	0	0	0	3	7	51	52	1	54

94	divya	24	primi	6w1d	152	50	21.6	73	0	11	n	n	5.3	124	112	134	0	0	0	0	2/fd	0	0	0	1	1	3.3	8	50	52.2	2.2	62
95	lavanya	24	primi	6w5d	148	42	19.2	67	0	10	n	n	5.6	112	154	132	gdm/ins	0	1	1	0	0	0	0	0	1	2.6	7	42	43	1	53
96	sumathi	28	g2p1l1	7w2d	154	49	20.7	64	1	10.2	n	n	5.2	104	134	146	gdm/mp	0	0	0	2/cpd	0	0	1	0	0	2.9	8	49	49	0	56
97	kanniga	28	g2p1l1	7w4d	147	49	22.7	76	1	10.4	n	n	5.4	109	132	148	gdm/mp	0	1	1	0	0	0	0	0	1	3.29	8	49	51.3	2.3	60
98	tamilselvi	25	g3p2l2	7w2d	156	52	21.4	75	0	11	n	n	5.3	110	123	110	0	0	0	0	2/fd	0	0	1	0	0	2.3	7	52	52.5	0.5	62
99	indumathi	25	g2p1l1	7w3d	153	51	21.8	72	0	10.2	n	n	5.2	106	152	116	gdm/ins	0	0	0	2/fd	0	0	0	0	0	3.1	8	52	52.5	0.5	65
100	nithya	32	g2p1l1	8w3d	152	48	20.8	74	0	10	n	n	5.1	108	145	152	gdm/mp	0	0	0	2/pcs	0	0	0	1	0	2.68	7	54	54.5	0.5	58
101	sakthi sangeetha	27	primi	6w4d	148	45	20.5	68	1	9.8	n	n	5.5	118	154	108	gdm/mp	0	1	1	0	0	0	0	0	1	3	7	45	47.5	2.5	58
102	amudhalavanya	30	primi	7w2d	152	58	25.1	82	0	11.6	n	n	5.2	102	124	156	gdm/mp	0	0	0	2/fi	1	0	1	1	0	3.7	8	58	60.5	2.5	67
103	lalitha	29	g3p2l2	8w	149	52	23.4	81	1	10.6	n	n	5.3	106	156	123	gdm/mp	0	1	1	0	0	0	1	0	1	2.3	8	52	54.5	2.5	62
104	vijayalaxmi	26	primi	7w2d	150	53	23.6	80.5	0	10.2	n	n	4.9	107	148	124	gdm/mp	0	0	0	2/fd	0	0	0	0	0	3	8	54	55	1	58
105	rajeswari	30	g2p1l1	8w2d	154	51	21.5	81	0	11.2	n	n	5.4	116	116	148	gdm/mp	1	1	0	2/fd	0	0	1	0	1	3.3	8	51	52.8	1.8	61
106	geethaumachanda	29	g2p1l1	7w2d	153	49	20.9	74	0	12	n	n	4	120	152	124	gdm/mp	0	0	0	2/fd	0	0	0	0	0	3.25	7	49	51.5	2.5	59
107	logeshwari	25	g2p1l1	8w	147	52	24.1	84	1	13	n	n	5.6	103	156	120	gdm/mp	1	1	1	0	0	0	0	0	0	2.6	8	52	54.3	2.3	62
108	dillidevi	25	primi	7w3d	152	51	22.1	80	0	11	n	n	5.7	112	124	123	0	0	1	0	2/fd	0	0	0	0	1	2.9	8	51	51.5	0.5	61
109	sulochana	31	primi	7w3d	151	49	21.5	79	0	12	n	n	5.8	124	156	134	gdm/mp	0	0	0	2/fd	0	0	1	0	1	2.5	8	49	51.6	2.6	58
110	shabeen	24	g3p2l2	7w4d	149	54	24.3	82	0	10.6	n	n	6	108	124	138	0	0	1	0	2/fi	0	0	0	0	0	2.6	8	54	54.5	0.5	64
111	mary	29	g2a1	6w4d	153	64	27.3	85	0	10.1	n	n	6	110	174	145	gdm/ins	0	1	1	0	1	0	1	0	0	3.6	8	64	67	3	78
112	radha	30	g2p1l1	7w	156	53	21.8	73	0	10.2	n	n	5	102	134	172	gdm/mp	0	0	0	2/fd	0	0	0	0	0	2.6	8	53	54	1	64
113	meena	28	g2p1l1	8w2d	153	50	21.4	76	1	10.5	n	n	5.6	112	162	124	gdm/mp	0	1	1	0	0	0	0	0	1	2.75	8	50	52.6	2.6	58
114	santhanalaxmi	37	primi	7w4d	162	58	22.1	74	0	11	n	n	5.4	103	176	146	gdm/ins	0	0	1	2/fd	0	0	1	0	0	2.75	8	58	58.5	0.5	67
115	yasodha	27	primi	7w6d	149	50	22.5	71	0	12	n	n	5.5	111	154	146	gdm/ins	1	1	1	0	0	0	0	1	0	3.09	8	50	53	3	59
116	bhavani	26	g2p1l0	8w	150	48	21.3	70	0	11.4	n	n	5	106	124	123	0	0	0	1	0	0	0	0	0	0	3	8	48	48.5	0.5	58
117	laxmi	32	g2p1l1	8w1d	152	47	20.3	69	1	10.2	n	n	5.4	109	142	157	gdm/ins	0	0	0	2/fd	1	0	1	0	1	4.1	6	47	49.5	2.5	60
118	tenmozhi	24	g2p1l0	10w3d	156	65	26.7	83	1	9.9	n	n	5.3	138	154	146	gdm/mp	0	1	1	0	1	0	1	0	0	3.63	8	65	67.3	2.3	75
119	deepa	26	primi	10w2d	153	64	27.3	86	1	10.4	n	n	5.2	118	156	128	gdm/ins	0	1	0	2/fi	0	0	0	0	0	3.2	8	62	64.5	2.5	66
120	hefsiba	35	g3p2l2	11w	152	56	24.2	81	1	10.2	n	n	5.6	118	123	134	0	0	0	0	2/pcs	0	0	0	0	0	3.2	8	54	56.5	2.5	58
121	sumithradevi	25	primi	10w	158	72	28.8	84	0	12	n	n	5.4	120	120	178	gdm/ins	0	0	0	2/fd	1	0	1	0	1	4	8	72	74.5	2.5	84
122	amirtham	35	primi	6w4d	149	43	19.4	68	0	11	n	n	5.2	98	110	146	gdnm/mp	0	1	1	0	0	0	1	0	0	3.2	8	43	45.3	2.3	54
123	athilaxmi	26	g3p1l1a1	7w1d	152	49	21.2	78	0	10.2	n	n	4.9	124	112	153	gdm/mp	0	0	1	0	0	0	0	0	0	3	8	49	51.5	2.5	59
124	maryrubina	24	primi	12w4d	156	63	25.9	80.5	1	10.2	n	n	4.3	118	146	126	gdm/mp	0	1	0	2/fd	0	0	1	0	1	3.1	8	44	46.2	2.2	53
125	sangeetha	24	g2p1l1	13w	157	52	21.1	69	0	9.8	n	n	4.8	120	132	124	0	0	0	0	2/cd	0	0	0	0	0	3.17	8	52	53	1	63
126	kavitha	23	primi	9w	158	56	22.4	71	0	10.6	n	n	4.9	117	124	123	0	1	0	0	2/cpd	0	0	0	0	0	2.8	8	56	55	-1	66
127	parvathy	30	primi	10w	158	52	20.8	72	0	11.2	n	n	4.9	124	112	108	0	0	0	1	0	0	0	0	0	1	3.2	8	52	51	-1	63
128	punitha	28	g3p1l1a1	11w	156	42	17.3	64	0	10.8	n	n	5.3	122	110	124	0	0	0	1	0	0	0	0	0	0	2.8	7	45	45	0	55
129	priya	23	g2p1l1	9w	162	56	21.3	71	0	11	n	n	5.4	120	121	120	0	0	0	1	0	0	0	0	0	0	2.8	8	56	57	1	66
130	ananthi	23	primi	12w	157	51	20.7	76	0	11.6	n	n	5.8	116	108	112	0	0	0	1	0	0	1	0	0	0	1.9	7	51	50	-1	60
131	bhuvaneshwari	28	g2a1	8w2d	148	52	23.7	74	1	10.4	n	n	5.3	114	110	122	0	1	0	1	0	0	0	0	0	0	3.4	8	52	54.2	2.2	62
132	saritha	22	primi	9w4d	157	48	19.5	68	0	11	n	n	5	122	106	120	0	0	0	1	0	0	0	0	0	1	2.8	8	48	48	0	58
133	vidhubala	31	primi	8w2d	152	48	20.8	67	0	11.2	n	n	5.7	120	112	117	0	0	0	0	2/fd	0	0	0	0	0	2.8	7	48	47	-1	58
134	bhuvaneshwari	23	g2p1l1	10w2d	164	54	20.1	70	0	12	n	n	5	130	126	124	0	1	0	1	0	0	0	0	0	1	3.4	8	54	58.5	4.5	64
135	revathy	22	g2a1	11w	158	52	20.8	71	0	9.6	n	n	5.2	124	122	120	0	0	0	1	0	0	0	0	0	0	2.7	6	52	50.5	-1.5	63
136	umamaheswari	30	g2p1l1	9w	166	58	21	74	1	10	n	n	5.5	132	124	118	0	0	0	0	2/fi/bb	1	0	0	0	1	3.9	7	58	60.4	2.4	67
137	poongavanam	29	g2p1l1	10w	156	52	21.4	78	0	10.2	n	n	5.2	122	132	124	0	0	0	0	2/pcs	0	0	0	0	0	2.8	7	56	55	-1	66
138	gomathy	23	primi	9w3d	160	58	22.7	74	0	11	n	n	5.1	116	126	116	0	0	0	1	0	0	0	0	0	0	3.2	7	58	57	-1	68
139	vatchala	29	g2p1l1	10w	156	54	22.2	75	0	12	n	n	4.9	124	122	114	0	0	0	1	0	0	0	0	0	0	2.79	7	52	54.3	2.3	64
140	valli	30	g2p1l1	9w3d	158	54	21.6	68	0	11.6	n	n	5	130	124	118	0	0	1	1	0	0	0	0	0	0	2.75	8	52	51	-1	62

141	janet	30	primi	8w6d	157	50	20.3	68	0	13	n	n	5.1	114	122	116	0	0	0	1	0	0	0	0	0	0	3.1	8	50	49	-1	60
142	aruna	32	g3p1l1a1	9w	152	56	24.2	77	0	11	n	n	5.2	118	112	114	0	0	0	0	2/pcs	1	0	0	0	1	3.9	8	56	58.5	2.5	66
143	ananthi	26	primi	9w2d	156	52	21.4	75	0	10.8	n	n	5	112	129	122	0	0	0	0	2/fpd	0	0	0	1	1	3.4	8	52	51	-1	63
144	gunasundari	21	g2a1	9w5d	154	51	21.5	73	0	9.9	n	n	5.1	116	116	114	0	0	0	0	2/fd	0	0	0	0	0	3	6	51	53.3	2.3	59
145	kalaivani	21	primi	10w3d	149	50	22.5	74	0	10.1	n	n	4.9	114	98	107	0	0	0	1	0	0	0	0	0	0	2.8	7	50	49	-1	60
146	umamaheswari	28	g2p1l1	8w2d	154	48	20.2	69	0	10	n	n	5.3	123	124	128	0	0	0	1	0	0	0	0	0	0	2.4	8	48	48.5	0.5	57
147	kalpana	28	primi	9w	158	52	20.8	70	0	10.1	n	n	5.1	130	122	125	0	1	0	1	0	0	0	0	0	1	2.3	7	52	54.5	2.5	62
148	raziaamin	25	g2a1	9w3d	164	58	21.6	76	1	10.6	n	n	4.9	117	123	117	0	0	0	0	2/fi	0	0	0	0	0	2.8	7	58	57	-1	69
149	julieparveen	24	g3p2l1	10w1d	162	53	20.2	72	0	10.8	n	n	4.8	122	124	126	0	0	0	1	0	0	0	0	0	1	3.5	8	53	52.5	-0.5	62
150	suguna	29	primi	10w2d	160	54	21.1	73	0	11	n	n	4.9	110	116	108	0	0	0	0	2/fd	0	0	0	0	0	3.8	8	54	56.2	2.2	65
151	lilly	29	g2p1l1	11w	156	51	21	69	0	10.5	n	n	5.1	107	116	118	0	0	1	1	0	0	0	0	0	0	3.3	8	58	57	-1	68
152	vijayalaxmi	22	primi	11w3d	152	56	24.2	81	0	9.9	n	n	5.2	114	112	115	0	0	0	1	0	0	0	0	0	0	2.8	7	56	56.5	0.5	66
153	bhuvaneshwari	27	g2p1l1	8w5d	154	49	20.7	69	0	10.1	n	n	5.4	114	124	122	0	0	0	1	0	0	0	0	0	1	3.2	8	49	48	-1	58
154	ananthi	35	g3p1l1a1	9w	158	52	20.8	68	0	10.4	n	n	5.4	104	128	124	0	0	0	1	0	0	0	0	0	0	2.8	7	52	50.5	-1.5	62
155	vinodhini	28	primi	10w2d	149	48	21.6	70	0	10.2	n	n	5.3	102	106	114	0	0	0	0	2/fi	0	0	0	1	0	3.3	8	48	50.5	2.5	58
156	devi	25	primi	9w3d	150	50	22.2	71	0	11.4	n	n	5.1	108	112	124	0	0	0	1	0	0	0	0	0	0	2.3	7	50	49	-1	61
157	laxmi	23	g2p1l1	8w4d	152	51	22.1	76	0	9.9	n	n	5.2	112	123	130	0	0	0	1	2/fd	0	0	0	0	0	2.34	8	51	53.4	2.4	61
158	durgadevi	28	primi	9w4d	156	52	21.4	75	0	10.6	n	n	5.5	102	116	124	0	0	0	0	0	0	0	0	0	1	3.1	8	52	52.5	0.5	63
159	devika	24	primi	10w	154	51	21.5	73	0	10.4	n	n	5.2	108	114	126	0	1	0	0	2/fd	0	0	0	0	0	2.8	7	51	53.6	2.6	63
160	abitha	25	g2p1l1	8w3d	151	49	21.5	72	0	10.1	n	n	5.4	112	112	123	0	0	0	0	2/fd	0	0	0	0	0	2.8	8	49	50	1	59
161	santhiya	24	primi	9w	154	52	21.9	75	0	9.8	n	n	6	108	124	126	0	0	0	0	2/fd	0	0	0	0	1	3	8	52	51.5	-0.5	62
162	vanitha	31	g3p1l1a1	9w3d	156	54	22.2	73	0	10.2	n	n	6.1	112	123	122	0	0	0	1	0	0	0	0	0	0	2.7	7	55	54	-1	65
163	jayapradha	23	g2p1l1	10w3d	158	53	21.2	74	0	12	n	n	5.3	98	113	108	0	0	1	1	0	0	0	1	0	1	3.5	8	53	56.4	3.4	62
164	vijayalaxmi	27	g2p1l1	9w6d	153	51	21.8	76	0	11.1	n	n	5.2	100	114	109	0	0	1	1	0	0	0	0	0	0	3.1	8	51	51.5	0.5	61
165	bhuvaneshwari	30	g3p1l1a1	8w3d	152	50	21.6	75	0	10.3	n	n	5.1	101	123	105	0	0	0	1	0	0	0	0	0	0	3	7	52	51.5	-0.5	62
166	kavitha	25	g2p1l1	9w	152	49	21.2	76	1	11	n	n	5.8	108	126	108	0	0	0	1	0	0	0	0	0	1	2.9	8	49	51.5	2.5	59
167	thenmozhi	20	primi	9w1d	149	48	21.6	77	0	10.2	n	n	5.4	112	118	104	0	0	0	1	0	0	0	0	0	0	2.7	7	48	49	1	58
168	vinitha	37	g2p1l1	10w	156	52	21.4	78	0	9.9	n	n	5.3	96	114	118	0	0	0	1	0	0	0	0	0	0	3	8	49	50	1	59
169	malini	31	g3p1l1a1	10w1d	150	48	21.3	76	0	10	n	n	5.2	113	112	105	0	0	0	0	2/fd	0	0	0	0	0	3.25	8	48	51	3	57
170	arputhamani	21	primi	11w	154	54	22.8	72	0	11.2	n	n	5.1	108	111	116	0	0	0	1	0	0	0	0	0	0	3.1	6	54	54.5	0.5	64
171	megala	23	primi	9w	156	53	21.8	76	0	10.2	n	n	4.8	112	98	106	0	0	0	1	0	0	0	0	0	0	2.5	7	53	55.2	2.2	63
172	divya	23	g2p1l1	9w1d	150	51	22.7	73	0	9.8	n	n	4.9	108	106	105	0	0	1	1	2/fd	0	0	0	1	1	3.165	7	52	52.5	0.5	63
173	rekha	30	primi	8w2d	153	50	21.4	74	0	10	n	n	4.2	109	112	108	0	0	0	1	0	0	0	0	0	0	3.3	8	50	53	3	60
174	prema	27	primi	8w3d	163	62	23.3	72	0	10.1	n	n	4.5	112	108	112	0	0	1	1	0	0	0	0	0	0	2.9	9	62	60	-2	71
175	sasikala	24	g2p1l1	9w	158	54	21.6	69	0	10.6	n	n	4.7	108	114	112	0	1	0	1	0	0	0	0	0	0	2.8	9	54	52.5	-1.5	65
176	anitha	22	primi	7w6d	152	51	22.1	77	0	9.8	n	n	4.8	112	96	108	0	0	0	0	2/fd	0	0	0	0	0	3.2	8	51	53.7	2.7	62
177	sangeetha	28	primi	8w1d	154	52	21.9	69	1	10.2	n	n	4.7	108	101	116		0	0	1	0	0	0	0	0	0	2.87	8	51	50	-1	61
178	gowthami	26	g2p1l1	8w6d	154	54	22.8	70	0	10	n	n	5	112	112	107	0	0	0	1	0	0	0	0	0	0	2.7	9	54	53	-1	65
179	janagavalli	28	g2p1l1	7w5d	152	52	22.5	72	0	12	n	n	5.1	106	114	108	0	0	1	0	2/pcs	0	0	0	0	0	3.4	8	52	54.5	2.5	62
180	nandhini	23	primi	8w1d	151	48	21.1	69	0	11	n	n	5.2	113	109	104	0	0	0	1	0	0	0	0	0	0	2.4	7	48	48.5	0.5	58
181	kalpana	26	g2p1l1	8w4d	154	52	21.9	77	0	10.3	n	n	5.3	99	132	124	0	0	0	0	2/cpd	0	0	0	0	0	3.08	8	52	51	-1	61
182	mohanavalli	23	primi	9w6d	153	51	21.8	74	0	10	n	n	5.4	98	128	122	0	0	0	1	0	0	0	0	0	0	2.57	8	51	50	-1	59
183	chitra	26	g2p1l1	10w	154	52	21.9	72	0	10.1	n	n	5.5	101	124	126	0	0	0	1	0	0	0	0	0	1	3.03	8	52	54.2	2.2	62
184	sudha	27	g2p1l1	8w3d	151	49	21.5	74	1	11	n	n	5.3	106	122	108	0	0	0	0	2/fd	0	0	0	0	0	2.8	7	49	49.5	0.5	59
185	selvi	25	g2p1l1	9w	153	50	21.4	76	0	9.9	n	n	5.2	112	118	108	0	0	0	0	2/pcs	0	0	0	0	0	3.4	7	51	51.5	0.5	60
186	hemalatha	24	primi	9w1d	156	54	22.2	75	0	10.1	n	n	5.1	118	106	108	0	0	1	1	0	0	0	0	0	0	2.39	8	54	53.5	-0.5	64
187	indhuja	25	primi	9w1d	153	51	21.8	73	0	10.4	n	n	5.4	107	114	108	0	0	0	1	0	0	0	0	0	0	2.9	8	52	54.3	2.3	63

188	nalini	28	g2p1l1	10w	154	52	21.9	72	1	10	n	n	5.3	123	112	108	0	0	0	0	2/fd	0	0	0	0	0	2.4	8	52	52.5	0.5	63
189	revathy	24	primi	9w4d	150	48	21.3	74	0	11	n	n	5.2	98	118	123	0	0	0	1	0	0	0	0	0	0	3.35	7	48	47	-1	58
190	sarasu	25	primi	10w2d	154	52	21.9	71	0	12	n	n	5.1	84	116	106	0	0	0	0	0	0	0	0	1	1	3.24	9	52	51.5	-0.5	61
191	latha	25	g3p1l1a1	11w2d	156	54	22.2	76	0	11.5	n	n	5.3	98	112	109	0	0	0	1	0	1	0	0	0	0	3.57	8	54	54.5	0.5	63
192	geetha	27	primi	10w3d	158	56	22.4	74	0	10.8	n	n	5.2	112	108	110	0	0	0	0	2/pcs	1	0	0	0	0	3.58	7	54	56.6	2.6	65
193	anjalai	23	g2p1l1	10w	153	49	20.9	74	0	10.2	n	n	5.1	102	118	109	0	0	0	1	0	0	0	0	0	0	2.8	7	49	49.5	0.5	58
194	amdha	24	primi	10w1d	155	51	21.2	65	0	9.8	n	n	4.9	108	109	110	0	0	0	1	0	0	1	0	0	0	2.18	8	48	48.5	0.5	58
195	marysandhya	25	g2p1l1	9w6d	154	49	20.7	68	0	9.6	n	n	4.9	97	101	123	0	0	1	0	2/fi	0	0	0	0	0	2.5	8	49	51.2	2.2	59
196	banu	31	primi	10w2d	156	52	21.4	77	0	10.6	n	n	5.2	98	112	103	0	0	0	1	0	0	0	0	0	0	3.38	8	52	51	-1	62
197	muthumari	22	primi	10w3d	154	51	21.5	75	0	10.2	n	n	5.3	99	108	106	0	0	0	1	0	0	0	0	0	0	2.91	8	52	51.5	-0.5	63
198	nithya	23	primi	11w	156	53	21.8	75	0	10	n	n	5.5	101	112	124	0	0	0	1	0	0	0	0	0	0	2.54	7	54	53.5	-0.5	64
199	alamelu	24	g2p1l1	9w4d	148	43	19.6	74	0	10.1	n	n	6	102	114	127	0	0	0	1	0	0	0	0	0	0	2.9	8	53	54	1	63
200	selvameena	25	g2a1	8w35d	140	58	29.6	76	0	12	n	n	6.1	120	116	132	0	0	0	0	2/fd	0	1	0	1	1	1.7	7	58	57	-1	68
201	rosy	26	primi	9w3d	146	50	23.5	73	0	11.1	n	n	5.8	101	120	124	0	0	1	0	2/fi	0	0	0	0	0	2.3	7	50	52.1	2.1	60
202	laxmi	25	primi	8w2d	148	51	23.3	75	0	10.6	n	n	6.2	99	128	126	0	0	0	1	0	0	0	0	0	0	2.8	8	48	49	1	59
203	manjula	19	g2a1	10w	152	48	20.8	76	0	9.9	n	n	4.8	88	122	124	0	0	0	1	0	0	0	0	0	0	2.7	8	48	47	-1	58
204	thennarasi	24	primi	9w1d	154	52	21.9	77	0	10.1	n	n	4.9	96	123	116	0	0	0	1	0	0	0	0	0	0	2.3	7	52	51	-1	62
205	sangeetha	28	primi	9w4d	154	60	25.3	84	0	9.6	n	n	5.2	102	112	118	0	0	0	1	0	0	0	0	0	1	3	8	60	59	-1	70
206	pooja	19	primi	8w6d	150	52	23.1	81	0	10.2	n	n	5.3	120	113	115	0	0	1	0	2/fd	0	0	0	0	0	2.5	8	52	51.5	-0.5	61
207	shyamala	24	primi	9w1d	152	51	22.1	76	0	11	n	n	5.2	123	114	120	0	0	0	1	0	0	0	0	0	1	2.6	8	51	50	-1	61
208	indira	29	primi	9w3d	154	52	21.9	73	0	10.3	n	n	5.7	102	114	116	0	0	0	1	0	0	0	0	0	0	2.5	8	52	51.5	-0.5	56
209	radha	24	g3p1l1a1	10w	148	46	21	75	0	11	n	n	5.4	106	108	112	0	0	1	1	0	0	0	0	0	0	2.8	7	48	48.5	0.5	57
210	kaleeshwari	25	g2p1l1	7w3d	150	49	21.8	75	0	10.2	n	n	5	104	112	114	0	0	0	0	2/cpd	1	0	1	0	0	3.9	8	49	50	1	61
211	kanagasundari	24	g3p1l1a1	8w1d	163	49	18.4	73	0	11	n	n	6	112	108	112	0	0	0	1	0	0	0	0	0	0	2.5	8	49	48	-1	60
212	logeshwari	28	primi	9w1d	147	49	22.7	75	0	11.2	n	n	6.1	102	116	120	0	0	0	1	0	0	0	0	0	0	2.7	7	49	48.5	-0.5	59
213	nadhiya	24	primi	8w2d	148	48	21.9	76	0	11.6	n	n	6.2	101	126	118	0	0	0	0	2/fd	0	0	0	0	0	2.7	8	48	50.4	2.4	57
214	shanmugapriya	27	g3p1l1a1	9w	161	54	21	74	0	10.8	n	n	5.4	112	122	114	0	0	0	0	2/fd	0	0	0	0	0	2.3	7	54	53	-1	63
215	akiladevi	24	g3p1l1a1	9w2d	156	58	23.8	73	0	11	n	n	5.2	124	118	120	0	0	1	1	0	0	0	0	0	0	2.2	7	58	57	-1	68
216	sivaranjani	27	primi	8w6d	152	52	22.5	77	0	10.2	n	n	5.4	109	106	112	0	0	1	0	2/fi	1	0	0	0	0	3.6	8	52	52.5	0.5	64
217	nithiya	23	primi	9w	154	51	21.5	74	0	10	n	n	5.3	124	114	118	0	0	1	1	2/fd	0	0	0	0	0	2.4	7	52	51	-1	63
218	sathya	22	g3p1l1a1	8w3d	156	54	22.2	72	1	11.2	n	n	4.5	104	118	108	0	0	0	1	0	0	0	0	0	0	2.7	8	54	53	-1	64
219	shanthi	23	g2p1l1	9w1d	149	43	19.4	68	0	10.8	n	n	4.6	112	106	118	0	0	0	1	0	0	0	0	0	0	2.7	8	43	42	-1	52
220	prabha	28	primi	7w3d	152	49	21.2	70	0	10.2	n	n	5.1	101	123	106	0	0	0	1	0	0	0	0	0	0	3.1	8	49	49.5	0.5	58
221	mahalaxmi	33	g3p1l1a1	8w	148	50	22.8	72	0	12	n	n	5.4	111	121	117	0	0	0	0	2/nopl	0	0	0	0	0	3.4	7	50	51.5	1.5	59
222	celinemary	27	primi	8w2d	150	51.5	22.9	73	0	11.2	n	n	5.3	106	112	118	0	0	0	1	0	0	0	0	0	1	2.76	8	51.6	52	0.4	59
223	premalatha	30	g2p1l1	9w1d	152	50	21.6	70	0	10.4	n	n	5.2	104	108	110	0	0	0	1	0	0	0	0	0	0	2.6	7	50	51	1	60
224	jothi	32	primi	8w	155	53	22.1	74	0	10.2	n	n	4.7	114	110	108	0	0	0	0	2/fd	0	0	0	0	0	2.3	7	51	52	1	59
225	priya	26	g2a1	8w3d	149	49	22.1	74	0	10.1	n	n	4.6	112	102	108	0	0	0	1	0	0	1	0	0	0	2.1	7	49	48.5	-0.5	60
226	shanmugapriya	31	g2p1l1	8w4d	154	52	21.9	72	0	10	n	n	5.4	99	112	106	0	0	0	0	0	0	0	0	0	0	3.4	8	52	52.5	0.5	61
227	karthika	23	primi	7w6d	152	51	22.1	74	0	9.8	n	n	5.3	97	108	110	0	0	0	0	2/fpd	0	0	0	0	0	2.2	8	51	50	-1	60
228	vasanthakumari	23	g3p2l1	8w2d	148	50	22.8	75	0	10.6	n	n	5.2	102	106	117	0	0	0	1	0	0	0	0	0	0	3.2	9	50	49.5	-0.5	58
229	rubini	23	primi	9w	150	49	21.8	73	0	10.2	n	n	5.1	105	116	104	0	0	1	1	0	0	0	0	0	0	3.2	7	49	49	0	58
230	vaduvambal	24	g2p1l1	9w1d	153	51	21.8	77	1	10.6	n	n	4	102	107	121	0	0	0	1	0	0	0	0	0	0	2.8	8	51	51.5	0.5	62
231	banumathi	22	g2p1l1	8w3d	148	48	21.9	78	0	12	n	n	4.7	120	108	110	0	0	0	0	2/fpd	0	0	0	0	0	2.56	9	48	48	0	57
232	manjula	23	g2p1l1	8w5d	152	49	21.2	77	0	11.3	n	n	5.4	112	112	109	0	0	0	1	0	0	0	0	0	0	2.6	8	49	48.5	-0.5	59
233	logeshwari	24	primi	9w	156	52	21.4	76	0	10.3	n	n	5.3	112	109	110	0	0	0	1	0	0	0	0	0	0	2.5	8	52	52.5	0.5	62
234	bhuvaneshwari	24	g2a1	8w1d	157	56	22.7	75	0	10.4	n	n	5.2	109	110	109	0	0	0	1	0	0	0	0	0	0	2.4	9	56	55	-1	65

235	selvi	29	primi	8w5d	151	49	21.5	72	1	9.9	n	n	5.6	107	112	116	0	0	0	1	0	0	0	0	0	0	2.8	8	49	49.5	0.5	58
236	viji	27	g2a1	7w3d	149	51	23	71	0	10.2	n	n	5.2	125	106	112	0	0	1	1	0	0	1	0	0	0	2.3	8	51	51.5	0.5	62
237	kalpana	29	primi	7w5d	150	53	23.6	81	0	10.6	n	n	5	102	98	109	0	0	0	1	0	0	0	0	0	0	2.4	8	53	52	-1	62
238	kalaiselvi	22	primi	8w3d	147	45	20.8	74	0	10.2	n	n	5.1	106	96	107	0	0	0	0	2/fd	0	0	0	0	0	2.5	8	45	46	1	55
239	rekha	23	g2p1l1	8w	151	50	21.9	72	0	9.8	n	n	4.7	104	108	115	0	0	1	0	2/fd	0	0	0	0	0	3	8	50	50.5	0.5	61
240	jothi	30	primi	9w1d	149	54	24.3	71	0	10.3	n	n	4.5	98	116	107	0	0	0	0	2/fd	0	0	0	0	0	2.3	7	54	53	-1	64
241	ranjani	24	primi	8w6d	151	52	22.8	73	0	10.2	n	n	5.1	102	120	109	0	0	0	1	2/fd	0	0	0	0	0	2.9	8	52	52.5	0.5	62
242	madhumathi	21	g2p1l1	9w1d	154	51	21.5	75	0	10	n	n	4.8	119	113	124	0	0	0	1	0	0	0	0	0	0	2.6	7	51	50.5	-0.5	60
243	thenmoghi	22	primi	8w2d	148	50	22.8	71	0	10.1	n	n	4.7	111	107	117	0	0	0	1	0	0	1	0	0	0	2.2	7	54	52	-2	61
244	priya	25	primi	8w4d	156	52	21.4	74	1	10	n	n	5.6	101	108	118	0	0	0	1	0	0	0	0	0	0	2.6	7	52	52.5	0.5	59
245	manimegalai	30	g3p1l1	9w2d	152	54	23.4	73	1	12	n	n	5.4	112	110	124	0	0	0	1	0	0	0	0	0	0	3.1	7	54	52.5	-1.5	61
246	vanitha	27	primi	10w1d	156	52	21.4	74	0	13	n	n	5	103	112	120	0	0	0	1	0	0	0	0	0	0	3.3	7	52	51.5	-0.5	65
247	thilagavathy	25	g2a1	11w	154	52	21.9	76	0	11	n	n	5.2	104	114	123	0	0	0	1	0	0	0	0	0	0	2.9	8	54	52	-2	63
248	yuvasree	36	g4p1l1a2	10w3d	152	54	23.4	75	0	12	n	n	6	112	112	112	0	0	0	0	0	0	0	0	0	0	2.7	8	51	51.5	0.5	62
249	anjalai	32	g3p2l2	10w	148	52	23.7	73	0	10.4	n	n	6.1	102	110	124	0	0	1	1	0	0	0	0	0	0	3.1	8	54	54.5	0.5	64
250	Rajammal	29	primi	7w5d	150	53	23.6	81	0	10.6	n	n	5	102	98	109	0	0	0	1	0	0	0	0	0	0	2.4	8	53	52	-1	62

0 - No 1 - Yes 2 - LSCS

KEY TO MASTER CHART

1 .GA	-	GESTATIONAL AGE
2. HT	-	HEIGHT
3. WEIGHT	-	WEIGHT
4. BMI	-	BODY MASS INDEX
5. W/H	-	WAIST HIP RATIO
6. F/H/DM	-	FAMILY HISTORY OF DIABETES MELLITUS
7. HB	-	HAEMOGLOBIN
8. RFT	-	RENAL FUNCTION TEST
9. LFT	-	LIVER FUNCTION TEST
10.TM	-	TRIMESTER
11.R.FAC	-	RISK FACTOR
12. GDM/MP	-	GESTATIONAL DIABETES MELLITUS/MEAL PLAN

13.GDM/INS	-	GESTATIONAL DIABETES MELLITUS/INSULIN
14.P.E	-	PRE ECLAMPSIA
15.IOL	-	INDUCTION OF LABOUR
16.ND	-	NORMAL DELIVERY
17.LSCS	-	LOWER SEGMENT CAESAREAN SECTION
18.LGA	-	LARGE FOR GESTATIONAL AGE
19.SGA	-	SMALL FOR GESTATIONAL AGE
20. HYPO GLY	-	HYPOGLYCAEMIA
21.BA	-	BIRTH ASPHYXIA
22. HYP.BIL	-	HYPERBILIRUBINAEMIA
23. PRE PREG WT	-	PRE PREGNANCY WEIGHT
24.EP WT GAIN	-	EARLY PREGNANCY WEIGHT GAIN
25.T.WG	-	TOTAL WEIGHT GAIN