"EARLY DIAGNOSIS OF GESTATIONAL DIABETES MELLITUS BY HbA1C AS A PREDICTOR - PROSPECTIVE OBSERVATIONAL STUDY"

BY

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Dissertation submitted to

B.L.D.E (DEEMED TO BE UNIVERSITY) VIJAYAPURA

In partial fulfilment of requirements for the award of the degree of

MASTER OF SURGERY

OBSTETRICS AND GYNAECOLOGY

Under the guidance of

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ABBREVIATIONS

Short form	Full form
ACOG	American College of Obstetricians and Gynecologists
ADA	American Diabetes Association
DIPSI	Diabetes in Pregnancy Study Group of India standards
FBG	Fasting blood glucose
FBS	Fasting blood sugar
GDM	Gestational diabetes mellitus
HbA1C	Glycosylated hemoglobin
IADPSG	International Association of Diabetes and Pregnancy Study Group
IFIGO	International Federation of Gynecology and Obstetrics
IGT	Impaired glucose tolerance
LGA	Large for Gestational Age
MNT	Medical Nutrition Therapy
NDDG	National Diabetes Data Group
NICE	National Institute for Health and Care Excellence
NPV	Negative Predictive Value
OGTT	Oral Glucose Tolerance Test
PPBS	Postprandial blood sugar
PPV	Positive Predictive Value
ROC curve	Receiver Operative Characteristic curve
QCG	Queensland Cardiovascular Group
SGA	Small for Gestational Age
SOGC	Society of Obstetricians and Gynaecologists of Canada

SPSS	Statistical Package for Social Sciences
T2DM	Type 2 diabetes mellitus
T2DM	Type 2 diabetes mellitus
WHO	World Health Organization

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ABSTRACT

Back ground

Gestational diabetes mellitus (GDM) is characterized by glucose intolerance of any severity identified for the first time during pregnancy. The OGTT is the definitive test for identifying GDM. Various criteria adopt different values for the OGTT in the diagnosis of GDM. The OGTT, while the gold standard, is a complex procedure for subjects and healthcare practitioners. The participant must be fasting, necessitating a minimum of 2 hours for sample collection, during which at least two blood samples are obtained. The duration and quantity of samples obtained may increase based on the criteria employed. The WHO recognized HbA1C as a diagnostic instrument for diabetes mellitus in 2011. Still, no guidelines support the utilization of HbA1C as a diagnosis for GDM. This study was conducted to find the efficacy of HbA1c for early diagnosis of GDM.

Methodology:

The present study was done among 123 antenatal women with confirmed intrauterine pregnancy of gestational age. All the antenatal were sent for blood investigations. If the FBS exceeded 92 mg/dl or the HbA1c was below 6.5% during the first trimester, these values were deemed abnormal, prompting further evaluation in the second trimester with a repeat HbA1c and a 75 gm 2-hour DIPSI as a single-step protocol. All patients received 75 grams of anhydrous glucose dissolved in 250-300 millilitres of water, and plasma glucose levels were measured after two hours. A number below 140 mg/dl was classified as normal, whereas a value beyond 200 mg/dl was diagnosed as overt diabetes. Any number ranging from 140 to 200 mg/dl was classified as impaired glucose tolerance. Patients with HbA1c levels below 6.5% and

a DIPSI value ranging from 140 to 200 mg/dl underwent a 75g oral glucose tolerance test to validate the diagnosis of GDM.

Results:

Most cases (57.7%) were 21-25 years, followed by 26-30 years (26.8%). Multi-para was seen in 62.6% of cases. Overweight was seen in 15.4% of cases, and obesity was seen in 43.1% of cases. Vaginal delivery was seen in 65% of cases, and 2.4% of cases had pre-term deliveries. Low birth weight was seen in 3.3% of cases, and NICU admission was 28.5%. Of the total cases, 13.8% of cases were diagnosed as diabetic. Regarding diagnosing GDM, HBA1C had a sensitivity of 94.1%, specificity of 86.5%, PPV of 98.1% and NPV of 85.2%, with 5.65 as a cut-off level of HBA1C to diagnose as diabetic.

Conclusion:

HbA1C is considered as a reliable test for diagnosing GDM by WHO, which can help identify at-risk mothers so that care can be provided for a better outcome. GDM cases were counselled and advised regarding dietary as well as life style modification, which helped to reduce the complications in the latter half of pregnancy.

Key words: Pregnancy, HbA1C, FBS, GDM, PPV, NPV

INTRODUCTION

Gestational diabetes is defined as carbohydrate intolerance of variable severity with its onset of first recognition during pregnancy.[1] Insulin resistance becomes pronounced in the latter half of pregnancy due to the placental hormones.[2] Pregnant women with GDM face a heightened risk of adverse maternal and neonatal outcomes. [2] GDM pregnant mothers and their offspring are at risk of developing diabetes in the near future, therefore implicating two generations at risk.[3] Adverse maternal consequences include preeclampsia, hypertension, urinary tract infections, polyhydramnios, elevated rates of surgical interventions, and eventual diabetes mellitus.[4-8] In fetuses and neonates, it is linked to macrosomia, congenital malformations, metabolic disorders, respiratory distress syndrome, and obesity. Consequently, early diagnosis and timely intervention are essential.[9,10]

Gestational diabetes mellitus impacts 7% of pregnancies globally,[4] with prevalence in India 6 - 9% in rural [5] and 12 - 21% in urban regions.[6] Diagnosis occurs in 16.3% of cases at or before the gestational age of 16 weeks,[7] 22.4% between 17 - 23 weeks of gestation,[8] and 61.3% after 23 weeks.[5] The elevated incidence of diabetes mellitus and susceptibility among Asian Indian women predisposes them to gestational diabetes mellitus. Early detection and diagnosis of GDM are crucial, as timely intervention mitigates potential consequences. There is a necessity for economical universal screening and diagnosis techniques. Regrettably, there exists no global agreement on the screening and diagnostic standards for GDM.

Need for the study

The international agreement on scheduling, timing of the last meal fasting/non-fasting), method, glucose test dosage, sample type (venous/capillary), and thresholds for screening is inconsistent. Moreover, there are disputes on the affordability, accuracy, and utility of a screening method. Indian government suggests the screening of all pregnant women for GDM by the National Guidelines for Diagnosis and Management of GDM, revised in February 2018, utilizing diagnostic criteria established by the WHO.[6] The other two primary criteria employed in India are the DIPSI (2004)[9] and the IADPSG (2010).[10] This study aimed to monitor the efficiency of HbA1c as a predictor for the early diagnosis of gestational diabetes mellitus.

AIM AND OBJECTIVES

AIM

- To determine the efficacy of HbA1c as a predictor for early diagnosis of gestational diabetes mellitus.

OBJECTIVES

Primary Objective:

- The use of FBS and HbA1c as a new screening approach in the first trimester.

Secondary Objective:

- New screening prediction model created by using multivariable risk estimation based on HbA1c and FBS.

REVIEW OF LITERATURE

Pregnancy causes gradual alterations in the glucose metabolism of the antenatal mother. As pregnancy progresses, resistance to insulin and diabetogenic stress induced by hormones from the placenta requires an appropriate rise in insulin secretion. Insufficient compensation leads to the development of GDM.[11] The severity of problems in women with GDM is comparable to that of women without GDM. Universal screening is highly advised among those with a demonstrated high prevalence of T2DM.[12]

In many regions globally, GDM is diagnosed when blood glucose levels are over 140 mg/dl after 2 hours.[13-16] Women with gestational diabetes mellitus exhibit a heightened incidence of cesarean sections, preeclampsia, and macrosomia.[17]

Intensive care during pregnancy not only ensures safety in antenatal but also prevents obesity, impaired glucose tolerance, and diabetes in newborn babies when they grow. Metabolic adjustments during pregnancy occur to support the quickly growing foetus. This foetus induces changes in maternal hormonal levels.

Metabolic changes during normal pregnancy

In normal pregnancy:

Pregnancy increases insulin resistance, predominantly influenced by placental hormones.[18] This physiological insulin resistance, appearing paradoxical, fulfils an essential role: to guarantee a continuous and sufficient supply of glucose for the swiftly expanding foetus. As a result, maternal glucose utilization in peripheral tissues, such as skeletal muscle, is diminished, although hepatic glucose synthesis is sustained or may be slightly increased.[19] This alteration in glucose metabolism prioritizes foetal requirements, guaranteeing sufficient nutrition transfer across the

placenta. Moreover, the intricate equilibrium of glucose homeostasis during gestation is sustained by a compensatory elevation in maternal insulin secretion.[20]

Pancreatic β-cells experience hyperplasia and hypertrophy to accommodate increased insulin requirements. This compensation mechanism is precisely calibrated and can be exceeded in circumstances like gestational diabetes mellitus.[21] Enhanced lipolysis and raised free fatty acid concentrations, coupled with modifications in adipokine production, further influence altered glucose metabolism.[22] The exact regulation of these intricate connections, encompassing the function of gut microbiota and inflammatory indicators, continues to be a subject of ongoing investigation. Comprehending these physiological changes is crucial for differentiating normal pregnancy from pathological conditions and for formulating measures to promote healthy maternal and foetal outcomes. [11]

- Enhanced insulin efficacy during the first half
- Diabetogenic stress during the latter half.

During the initial weeks of gestation:

- Elevated fasting insulin levels
- Enhanced glucose-induced insulin secretion (high at 18-20 weeks)[23]
- Augmented estrogen and progesterone levels, resulting in hyperplasia of beta cells.

Ultimately, this results in hyperinsulinemia, with insulin serving as an anabolic and anti-catabolic hormone that promotes:[23-25]

- Storage of Glycogen in tissues
- Uptake of glucose in peripheries from the liver
- Requirement to decrease fasting blood sugar by 10%.

An elevation in human placental lactogen, prolactin, and cortisol induces stress. Consequently, the sensitivity of insulin in the mother diminishes by half and metabolic changes induced by insulin and hormones released by the placenta promote anabolism during feeding and increase catabolism at fasting. Fasting glucose levels remain consistently reduced because of the continuous foetal absorption of glucose. Low FBG results from a lack of glucogenic amino acids, referred to as "substrate deficiency syndrome." [21]During the latter half of pregnancy, resistance to insulin and stress induced by hormones released by the placenta require a corresponding rise in the secretion of insulin; GDM develops when this compensation is insufficient.[11]

Metabolic alterations in GDM

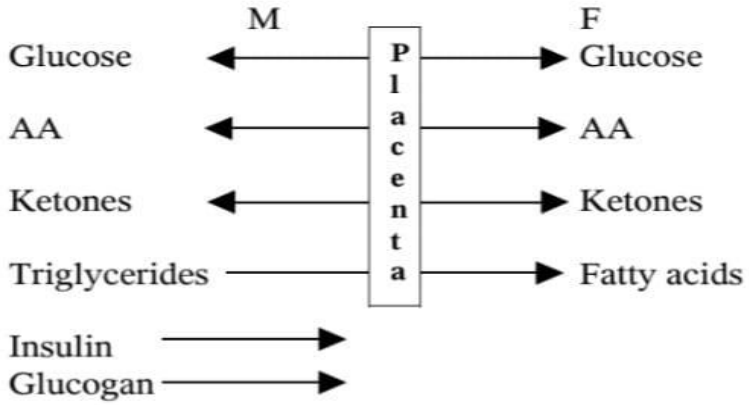
1. Decreased FBS concentration. [24]
2. Elevated fasting and postprandial plasma insulin concentrations.
3. Elevated postprandial blood sugar levels.
4. Hypertrophy as well as hyperplasia of β-cell.[25]
5. Diminished sensitivity of insulin and enhanced lipolysis.

Pathogenesis of GDM

Advanced age of antenatal and obesity are substantial hereditary contributors. In approximately 20% of GDM cases,[26] diminished early insulin secretion may not be evident, attributed to augmented secretion to the prenatal counter-regulatory hormones.[11] A post-receptor malfunction in the insulin signalling cascade seems to contribute to diminished sensitivity of insulin among pregnant with normal glucose tolerance and GDM.[24] GDM will occur when insulinogenic compensation of pregnant is insufficient to counteract these risks. Impaired β cell function observed in women with GDM may signify future vulnerability to diabetes.[21]

Effects on foetus

1. Hyperglycemia among pregnant and raised insulin in the foetus are the main effects of metabolic alterations. Glucose crosses the placenta via facilitated diffusion, while amino acids utilize active transport to reach the circulation of the foetus. [27]



2. As gestational age progresses, there is a two to three-times rise in the production of syncytiotrophoblast glucose transporters,[28] with amino acids being transported actively. Insulin controls the above mechanism; any disruption in its secretion and function affects the fetus's overall nutritional makeup and results in foetal hyperinsulinemia.[29] Maternal nutritional abnormalities infiltrate the growing fetus and alter the phenotypic expression of genes in newly forming cells, resulting in enduring short- and long-term impacts on the progeny.

Table 1: Complications of foetus caused by maternal hyperglycemia: [21]

First Trimester	Second Trimester	Neonatal
Malformations	Hypertrophic	Hypoglycemia
Growth	Cardiomyopathy	Hypocalcemia
retardation	Polyhydramnios	Hyperbilirubinemia
Fetal Wastage	Placental	Respiratory Distress
	insufficiency	Macrosomia >4 kg
	Preeclampsia	Hypomagnesemia
	Fetal loss	Low IQ

The yolk sac controls the distribution of nutrients from the plasma of the mother to the embryo in the development of the neural tube. It is impacted in the embryo's early post-implantation stage. Oxidative metabolism [30] and the production of free oxygen radicals [31] that could be detrimental to the embryo. Glucose promotes mutations in the DNA of the embryo. Therefore, optimal regulation of maternal metabolism should begin prior to conception and be sustained during the initial eight weeks. Folic acid supplementation and antioxidants contribute to a reduction in abnormalities up to 0.8%. [32,33]

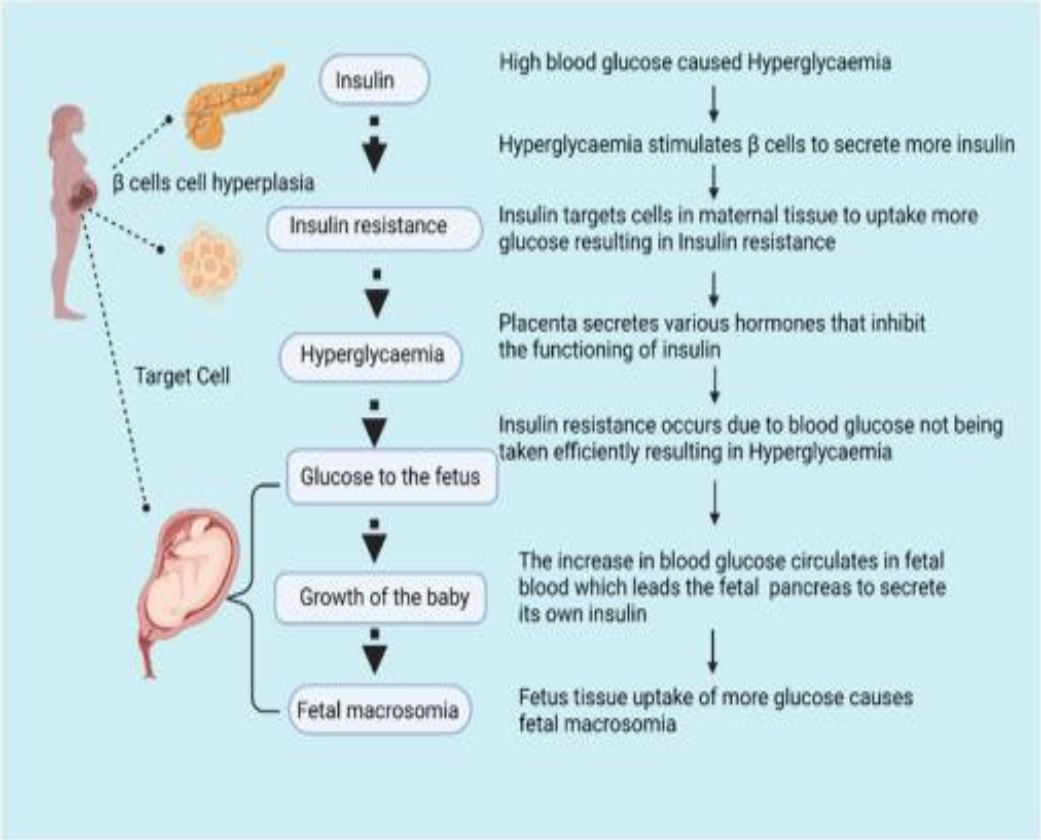


Figure 1: Effects of GDM on foetus [29]

Gestational diabetes mellitus is characterized by intolerance to carbohydrates, which is identified or first recognized in the current pregnancy. GDM results from impaired first-phase insulin production and the influence of anti-insulin hormones on glucose consumption and insulin secretion during pregnancy.

Table 2: Insulin levels [23]

	GDM	Non GDM
Insulin secretion	↑	↑
Glucose stimulated Insulin Secretion	↑	↑
Peak Plasma insulin	Late	N
1st phase insulin	-	More
2nd phase insulin	Same	Same

In a woman with normal tolerance towards glucose, secretion of insulin is elevated, and does not develop GDM. Thirty percent of GDM cases will advance to T2DM within the near future.[29]

Causes and pathology: [30-32]

1. β cells autoimmune destruction

2. β-cell impaired function

3. Enhanced insulin breakdown

4. Diminished tissue response to insulin

a. Dysfunctional binding of insulin receptor

b. Deficient signalling of insulin in the cell

Indian women have an elevenfold greater likelihood of developing gestational diabetes mellitus compared to White.

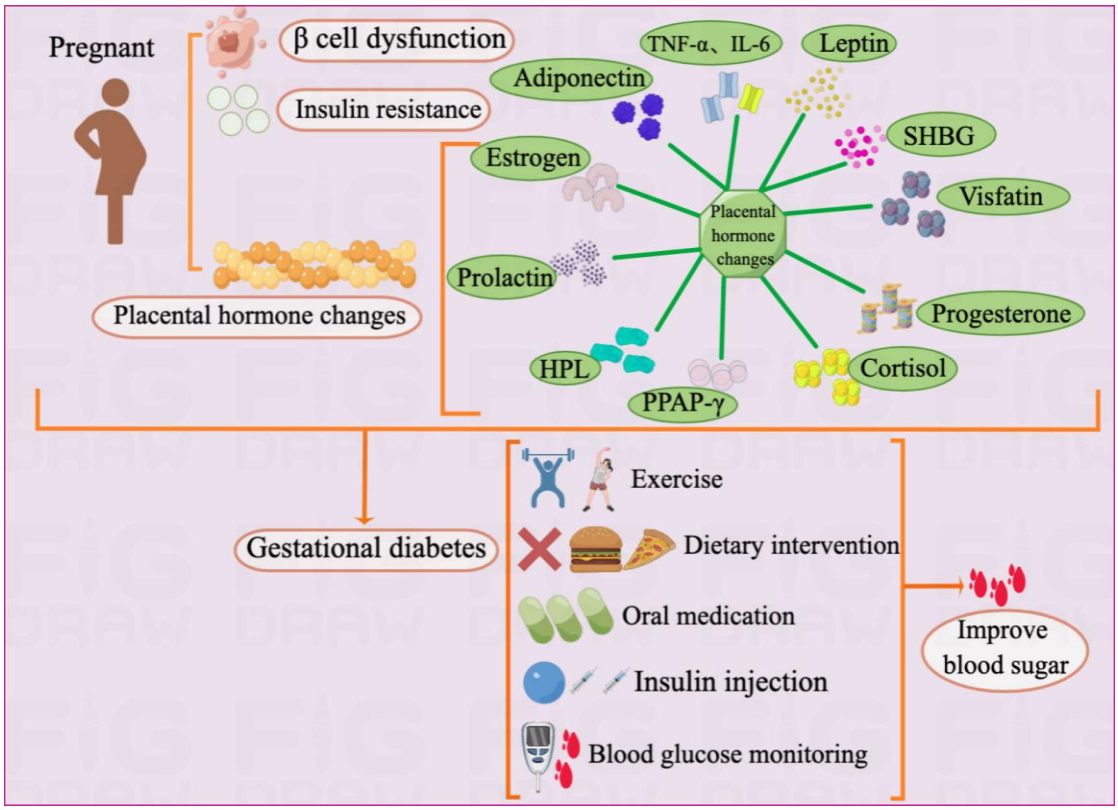


Figure 2: Metabolic changes in GDM [33]

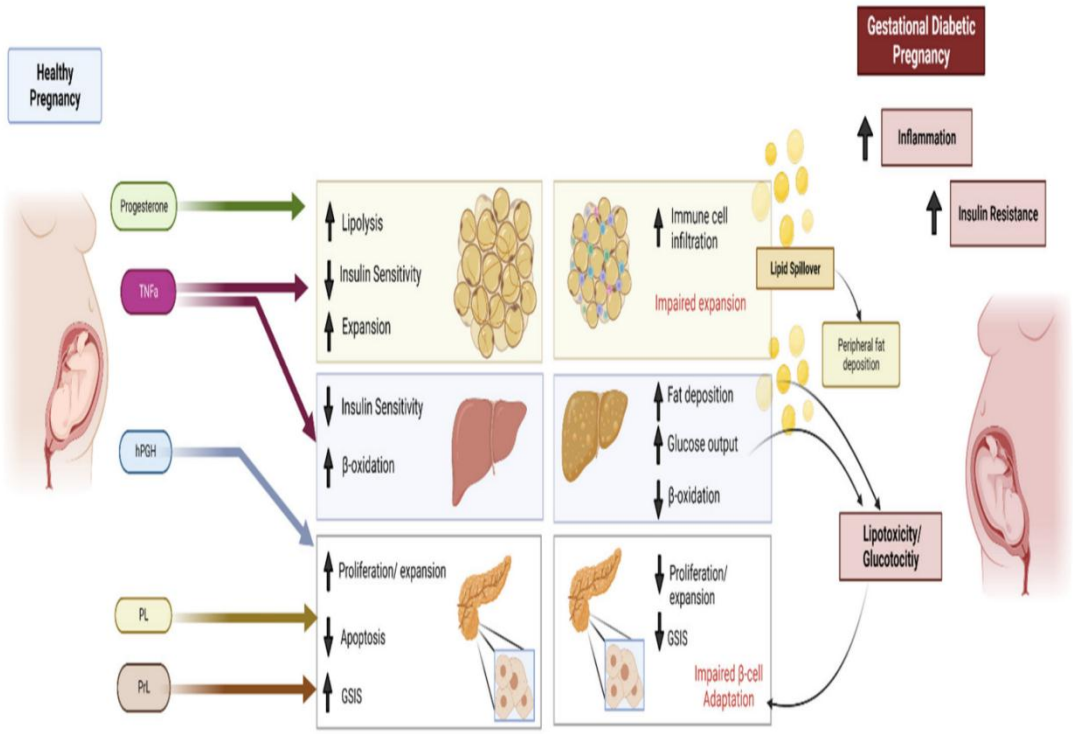


Figure 3: Comparison of Healthy and GDM pregnancy [34]

Evaluation of GDM:

Urine glucose: The threshold of the kidney for glucose is reduced during pregnancy. It makes glycosuria less selective for identifying gestational diabetes mellitus, so it is not suggested as a screening test.[11]

Blood glucose:

The standard prescription for screening occurs from 24 to 28 weeks of gestational age.

The prevailing notion is that antenatal should have a GTT during the first trimester. If negative, retest at approximately 24 to 28 weeks and again at 32 to 34 weeks.[35]

Table 3: Different criteria for diagnosis of GDM

Diagnostic criteria	Glucose intake	OGTT positivity threshold [mg/dl (mmol/L)]			
		Fasting	1 hour	2 hour	3 hour
O'Sullivan and Mahan[28]	50 gm (3 hours)	≥140 (7.8).	≥165 (9.2)	≥145 (8.1)	≥125 (6.9)
NDDG [29]	100 gm (3 hours)	≥105 (5.8)	≥190 (10.6)	≥165 (9.2)	≥145 (8.1)
Carpenter and Coustan[30]	100 gm (3 hours)	≥95 (5.3)	≥180 (10.0)	≥155 (8.6)	≥140 (7.8)
IADPSG (2010)[31] WHO (2013)[32]	75 gm (2 hours)	92–125 (>5.1)	180 (>10.0)	153–199 (> 8.5)	-
IFIGO (2015)[33]	75 gm (2 hours)	≥92(≥5.1)	≥180(≥10.0)	≥153 (≥8.5)	
DIPSI [34]	75 gm (2 hours)	-	-	≥140 (7.8)	-
NICE (2015)[35]	75 gm (2 hours)	≥100 (5.6)	-	≥140 (7.8)	-
SOGC (2019)[36]	75 gm (2 hours)	≥95 (5.3)	≥191 (≥10.6)	≥162 (≥9.0)	
ACOG (2018)[37]	Non-fasting (First time) Second time, ≥95 mg/dL	≥180 (10.0)	≥153 (8.5)	≥140(7.8)	
National guidelines by Government of India (2018)[38]	-	-	≥140	-	
QCG (2021)[39]	≥92 (5.1)	≥180 (10.0)	≥153 (8.5)	-	
ADA (2024)[40]	75 gm (2 hours)	<95(<5.3)	<140(<7.8)	<120(<6.7)	

Medical Nutrition Therapy (MNT) as per ADA

Medical nutrition treatment for GDM is a personalized dietary plan created by the pregnant individual and a registered dietitian nutritionist (RDN) experienced in GDM care. [41] The dietary plan must ensure sufficient caloric intake to support fetal, newborn, and mother health, attain glycemic targets, and facilitate healthy weight growth. No conclusive study exists that determines a specific ideal calorie intake for individuals with GDM or indicates that their caloric requirements differ from those of pregnant individuals without GDM. The advised dietary reference is at least 175 g of carbohydrates, 71 g of protein, and 28 g of fibre. [42,43]

The diet should have monounsaturated and polyunsaturated fats. The quantity and kind of carbohydrates will influence glucose levels in individuals with diabetes, as with all nutritional therapy. Prioritizing for superior, nutrient-rich carbs leads to regulated fasting and postprandial glucose levels, decreased free fatty acids, enhanced insulin sensitivity, and vascular advantages and may mitigate excessive obesity among the neonate.[44] Individuals who replace carbs with fat may inadvertently increase lipolysis and increase resistance to insulin. Urine ketone monitoring at a fast pace may be beneficial for identifying individuals who are significantly limiting carbohydrate intake and regulating blood glucose levels. Simple carbs will lead to elevated postprandial levels.

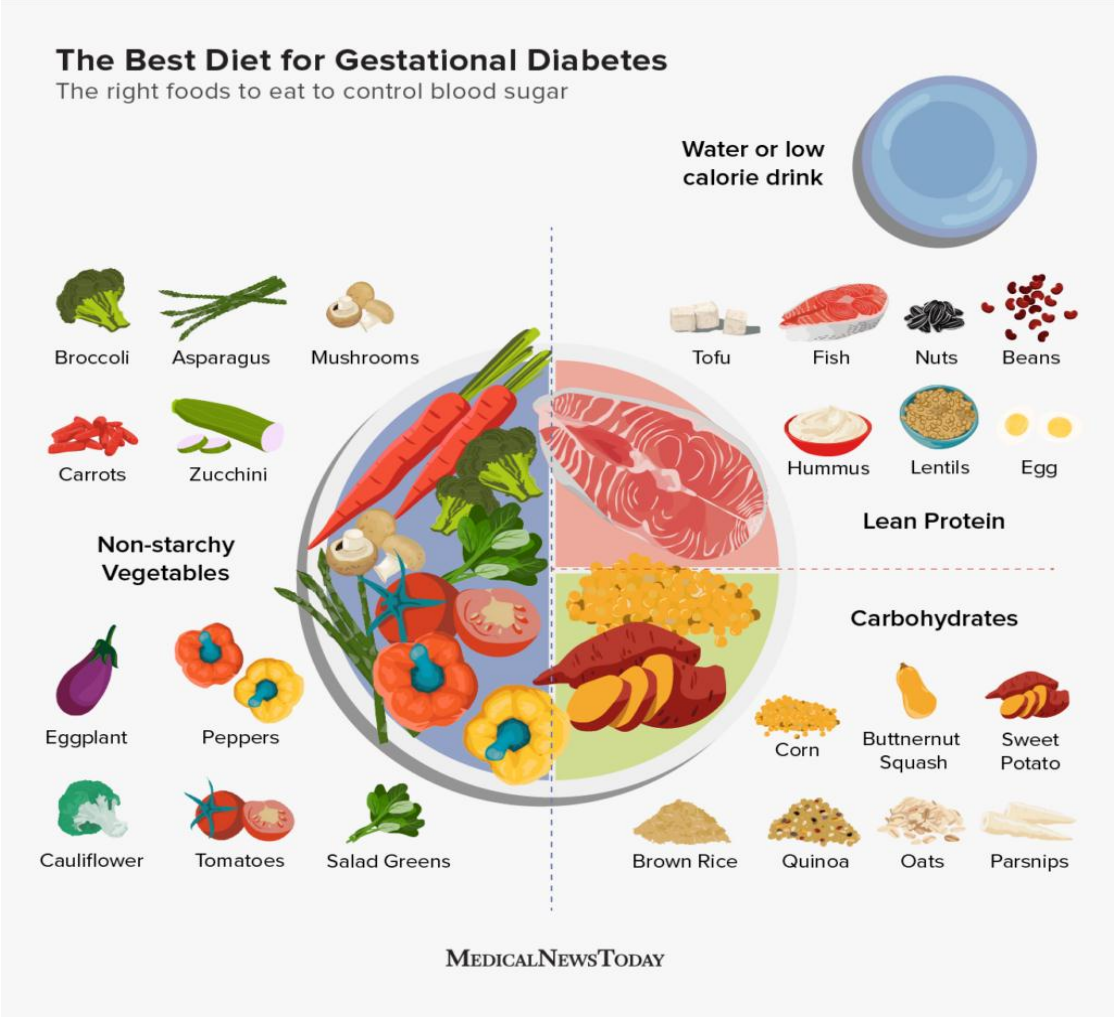


Figure 4: Medical Nutrition Therapy [44]

Physical activity as per ADA

Aerobic, resistance, or a combination and the amount of time of exercise is 20–50 minutes per day, 2–7 days per week at moderate level. [45]

WHO protocol

The WHO primarily recommends a one-step procedure using a 75-gram OGTT. As per WHO 1999, an FBS level of ≥ 126 mg/dl is universally regarded as excessive, prompting some groups to utilize only the 2-hour plasma glucose measurement without fasting plasma glucose, while others employ both measurements.[46] The criteria for the one-step screening and diagnostic test was established using a single cutoff point of 140 mg/dl, measured two hours post-

administration of a 75gm glucose load in a fasting condition. This became extensively adopted in numerous regions globally due to its remarkable convenience. The criteria need updating due to their arbitrary nature and probable reliance on the cutoff threshold for reduced glucose tolerance in a nonpregnant condition. [47]

Table 4: OGTT criteria for diagnosis of Diabetes Mellitus (WHO, FIGO, IADPSG) [44]

75 G - OGTT	Normal	GDM
FBS	<92 mg/dl (5.1 mmol/l)	92 - 125 mg/dl (>5.1 - 6.9 mmol/l)
1hr post prandial	<180 mg/dl (10 mmol/l)	≥180 mg/dl (≥ 10 mmol/l)
2hr post prandial	<153mg/dl (8.5 mmol/l)	153 to 199 mg/dl (8.5 - 11.0 mmol/l)

As per WHO (2018) Women should aim to keep:

- Fasting blood sugar between 65- 95 mg/dl (3.6 mmol/l-5.3 mmol/l)
- 1-hour postprandial below 140 mg/dl (7.8 mmol/l)
- 2-hour postprandial below 120 mg/dl (6.7 mmol/l)

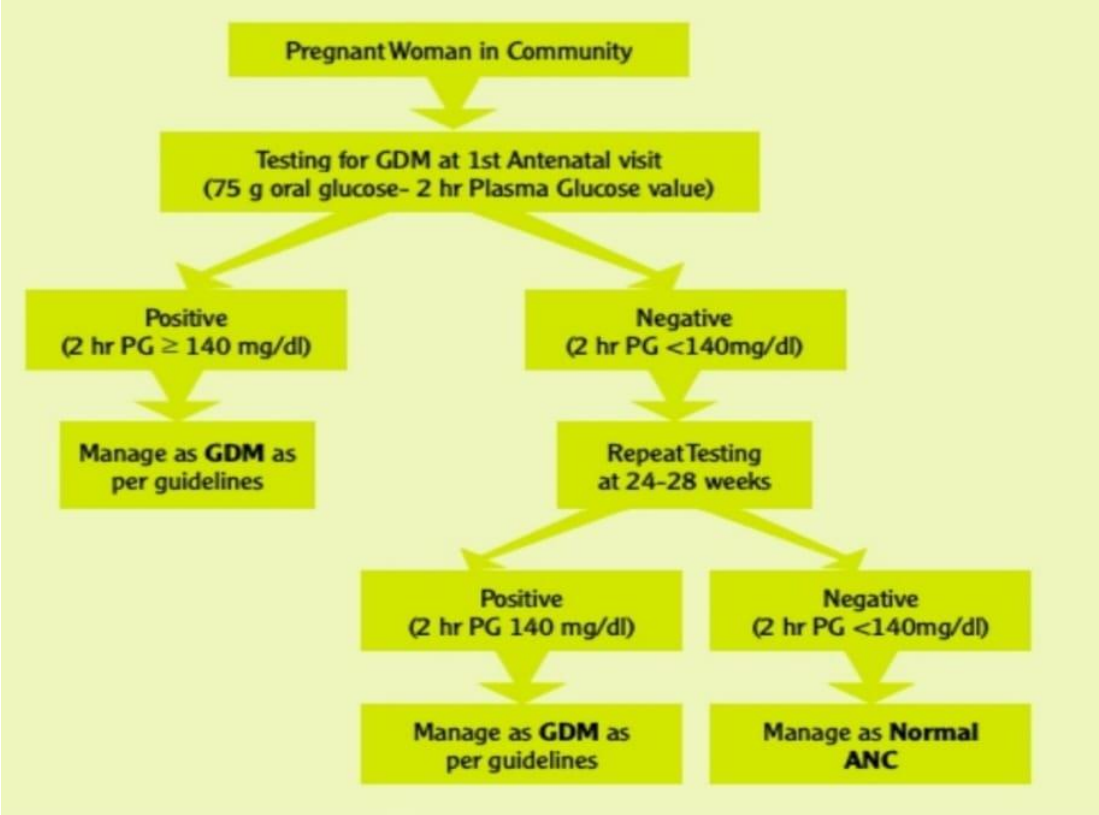


Figure 5: DIPSI flow chart [44]

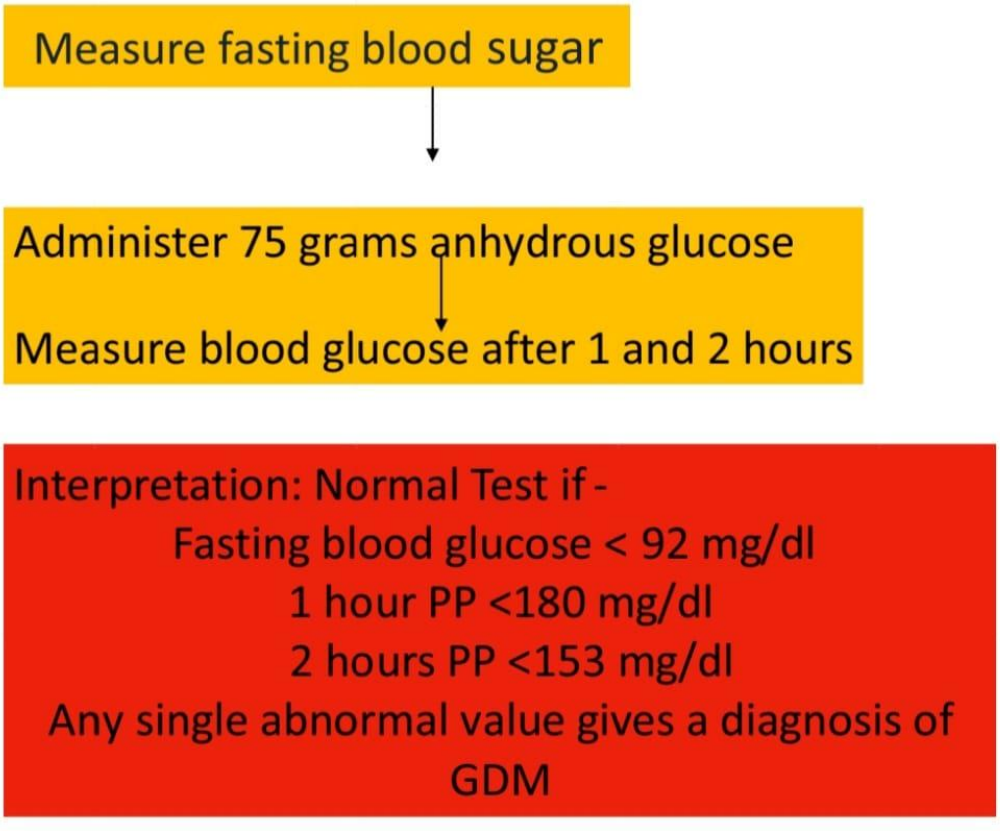


Figure 6: IADPSG criteria for GDM [44]

The critical determinant of fetal outcome in both pregestational and GDM is the level of glucose control achieved prior to and throughout pregnancy. The plasma glucose levels in usual pregnant are below 90 mg% while fasting and below 120 mg% during non-fasting. Therefore, optimal fetal outcomes can be anticipated by sustaining the average glucose at approximately 105 mg. [44]

1. Suitable dietary pattern.
2. Self-monitoring of Blood Glucose (SMBG)
3. Self-administration of insulin and modification of insulin dosages.
4. Management of hypoglycemia
5. Suitable physical activity.
6. Advancement of strategies to mitigate stress.

a. MNT

The dietary regimen must supply sufficient calories and nutrients to fulfil the requirements of pregnancy. The total increase in weight in pregnancy is 10 to 12 kg, i.e., on average, 300 to 400 grams per week. Pregnant women are recommended to carefully divide their caloric intake, particularly at breakfast, into two equal portions, ingesting them with a two-hour interval between each. MNT is recommended for two weeks.[44]

b. Insulin treatment

Insulin is needed if MNT does not attain euglycemia. During a typical pregnancy, the FBG concentration varies from 55 to 70 mg/dl, whereas the one-hour PPBS level is less than 120 mg/dl.

SMBG should be conducted prior to breakfast and 2.5 hours following each meal.[43] Women with gestational diabetes mellitus typically have elevated plasma glucose levels after breakfast in comparison to post-lunch and post-dinner

measurements. Some women with gestational diabetes mellitus exhibit elevated postprandial plasma glucose levels. Insulin is initiated within 1 to 2 weeks if fasting blood sugar exceeds 90 mg/dl despite medical nutrition therapy. The plasma glucose peak is elevated during breakfast, explained by the dawn phenomenon. The plasma glucose value exceeds the whole blood value by 12%. If the FPG concentration during the OGTT exceeds 120 mg/dl, the patient is promptly initiated on insulin alongside a meal plan.[44]

The insulin dosage is customized to the individual. The first dosage should commence at a minimum of 4 units and be subsequently titrated. The addition of fetal growth may reduce glucose monitoring for low-risk pregnancies. Maintaining an average blood glucose level below 105 mg% is optimal for favourable fetal outcomes since it mitigates the risk of hypoglycemia in the infant. While the fetus remains in the uterine environment, it possesses sufficient energy to sustain its blood glucose levels. Upon delivery of the fetus, the hyperactive β cells of the neonate persist in insulin secretion. However, there is an absence of sufficient energy flow from the maternal compartment, resulting in the newborn developing hypoglycemia. Neonatal hypoglycemia results from inadequate management of maternal diabetes. Although oral antidiabetic medications are not advised, evidence indicates favourable fetal outcomes in women with GDM treated with glyburide. Metformin has been beneficial for women with polycystic ovarian syndrome. Self-glucose monitoring is required weekly once the desired result is attained, monthly for FBS and PPBS up to 28 weeks, biweekly from 28 to 32 weeks, and until delivery thereafter.[44] In high-risk cases, the frequency of self-monitoring is increased. Additional assessments comprise fundus examination and microalbuminuria quantification.

Intrapartum Diabetic Management:

Metabolic investigations in non-GDM pregnant during labour indicated that glucose metabolism increases fourfold with minimal alterations in insulin levels during active labour; insulin requirements are negligible but glucose is needed at a rate of 2.6 mg/kg/min. Women with gestational diabetes mellitus do not require insulin after labour starts, and no insulin is necessary following the expulsion of the placenta.

Gestational diabetes mellitus necessitates follow-up and a 75-gram oral glucose tolerance test after 6 weeks, with a repeat if required after 6 months. Gestational diabetes mellitus recurs in around 50% of future pregnancies. Maintaining an optimal body weight significantly mitigates danger. The necessity of insulin alongside dietary measures to sustain euglycemia throughout the first pregnancy is indicative of potential future diabetes.[44]

Table 5: Safe Insulins for using in Pregnancy [44]

Insulin name	Type	Onset	Peak effect	Duration	Dosing interval
Aspart	Rapid acting	15 min	60 min	3-5 hrs	At start of each meal
Lispro	Rapid acting	15 min	60 min	3-5 hrs	At start of each meal
Regular	Short acting	30-60 min	2-4 hr	6-8 hrs	60-90 minutes before meal
NPH	Intermediate Acting	2 hr	4-6 hr	12-20hrs	Every 8-12 hr
Insulin detemir	Long acting	2 hr	-	24 hrs	Every 24 hr
Insulin Degludac	Long acting	-	-	> 24 hrs	> 24 hr

Table 6: Exercise guidelines for GDM [44]

Type of exercise	Intensity	Duration	Frequency
Aerobic (large muscle activities in a rhythmic manner) *e.g*, walking, running, swimming and cycling	Moderate 60%-90% of APHRM RPE 12-14 Previously sedentary Owt/Ob should begin training at 20%-30% of $APVO_2R$ RPE 12-14 Vigorous RPE 14-16	≤ 30 min continuously (up to 45 min if self-paced)	No more than two consecutive days without exercising
Resistance (multi joint exercises, large muscle groups) *e.g*, dumbbells, resistance band and pregnancy Pilates	Moderate 50% 1RM 5-10 exercises 8-15 repetitions 1-2 sets	60 min	At least 2 but ideally 3 times a week

APHRM: Age predicted heart rate maximum; RPE: Rate of perceived exertion; Owt: Overweight; Ob: Obese; $APVO_2R$: Age predicted VO_2 reserve; RM: Repetition maximum.

Table 7: Diet plan for GDM (1800 calories) [44]

Meal	Menu	Amount	Number of carbohydrate serves as per exchange list
Breakfast (7-8 am)	Dalia/Porridge/Oats Milk	½ cup 1 cup	2 Other varieties can be included in meal plan as per the exchange list
Mid- Morning (10-10.30 am)	Mung bean sprouts (ankurit mung)/Roasted Mung	½ cup	1
Lunch (1-1.30 pm)	Chapati Or chapati + Rice	2 1+1/3 cup	2-3
	Vegetables	1 cup	
	Yogurt/Curd	¾ cup	
	Soya nugget (soya badi) curry/Dal	½ cup	
Evening (4.30-5 pm)	Seasonal fruit (medium size)	1	1-2
	Murmura chat with vegetables/idli with sambhar	1 ½ cup/1	
Dinner (8-8.30 pm)	Chapati Or chapati + Rice	2 1+ 1/3 cup	2-3
	Vegetable	1 cup	
	Dal Or Fish (curry/grilled/ steamed)	½ cup ½ cup	
Bed time (10-10.30 pm)	Milk	1 cup	1
	Brown bread	1	
Total fat/d		4 tsp/d	

Table 8: Diet plan for GDM (2000 calories) [44]

Meal	Menu	Amount	Number of carbohydrate serves as per exchange list
Breakfast (7-8 am)	Whole grain Bread (Brown Bread)	2	2
	Egg bhurji/egg omelet	1	
Mid- Morning (10-10.30 am)	Vegetable Dalia	½ cup	1
Lunch (1-1.30 pm)	Chapati	3	3-4
	Or		
	chapati + Rice	2+1/3 cup	
	Vegetables	1 cup	
	Yogurt/Curd	¾ cup	
	Soya nugget curry/Dal	½ cup	
	Or		
	Chicken/fish curry	1 cup	
Evening (4.30-5 pm)	Seasonal fruit (medium size)	1	1-2
	Vegetable Poha/vegetable upma	½ cup	
Dinner (8-8.30 pm)	Chapati	2	2-3
	Or	1+ 1/3 cup	
	chapati + Rice		
	Vegetable	1 cup	
	Dal	½ cup	
Bed time (10-10.30 pm)	Milk	1 cup	1
	A bowl of cut mixed fruits	1	
Total fat/d		5 tsp/d	

Table 9: Food exchange list [44]

Food Groups	Food	Portion
Cereal/Starch Exchange Serving Choose any serving of the food mentioned here,each serving will provide – 75 calories 15 gm carbohydrates 2 gm protein 0-1 gm fat	Bread	1 slice (1oz)
	Idli (plain)	3" round - 1
	Naan	½ of 8"x2"
	Dosa (plain)	1
	Rice white or brown (cooked)	1/3 cup
	Roti (atta, bajra, corn, juwar)	1 (6")
	Murmura (puffed rice)	¾ cup
	Millet (cooked)	1/3 cup
	Museli	¼ cup
	Oats (cooked)	1/3 cup
	Pasta (cooked)	1/3 cup
	Pop-corn (no fat)	3 cups
	Biscuit (2 ½" across)	1
	Chowmein noodles	½ - 1/3 cup
	Muffin (small)	1 piece
	Poha (cooked)	1 cup
	Starchy vegetables:	
	Potato (baked or boiled)	1½ cup
	Potato (mashed)	½ cup
	Yam, sweet potato (plain)	1 small; ½ cup
Fruit Exchange Serving Choose any serving of the fruits mentioned here, each serving will provide – 45 calories 10 grams carbohydrate 1 gm protein Negligible fat	Apple (medium)	1 (4 oz)
	Apricots (dry)	3 pieces
	Cherries	15-20 pieces
	Blueberries	¾ cup
	Dates	3
	Grapes	¾ cups (10-12 nos)
	Guava (medium)	1
	Mango (medium)	½ cup
	Orange (medium)	1
	Papaya (cubes)	1 cup
	Peaches (medium, fresh)	1 (6 oz)

	Pear (medium) Pineapple (fresh) Plums (small) Sapota, Chikoo (medium) Strawberries (whole) Watermelon (cut and diced) Kiwi (medium) Banana	1½ ¾ cup (2 slices) 2 ½ approx 24 in nos. 2 cups 1 ½
Pulse Exchange Serving Choose any serving of the fruits mentioned here, each serving will provide – 100 calories 17 grams carbohydrate 7 gm protein	All lentil/dals cooked Sprouted pulses Soya nuggets	1 cup ¾ cups ¼ cup – 10 chunks
Vegetable Exchange Serving 1 serving = ½ cup cooked (100 gms–150 gms) or 1 cup raw vegetables . Choose any serving of the food mentioned here, each serving will provide- 30 calories 2.5-3.5 gms carbohydrate 2-3 gms protein 0 gm fat	Amaranath (chaulai) Bathua French beans Bean sprouts (moong) Beets (chukander) Bitter gourd (karela) Bottle gourd (lauki) Broad beans (papdi) Broccoli Brussels sprouts Cabbage Carrots Cauliflower Cluster beans (guvar) Cucumber Drumsticks (surgavo) Eggplant (brinjal) Fenugreek leaves Green onion Green papaya Jack fruit (kathal) Lady's finger (bhindi) Mustard leaves (sarson) Onion Parwal Peas	
	Pumpkin Radish Ridge gourd (torai or turia) Salad greens Spinach (physical activity levelak) Tomatoes fresh Zucchini	
Milk Exchange Serving Choose any serving of the food mentioned here, each serving will provide – 180 calories 12 gms carbohydrate 8 gms protein	**Skim and very low fat milk (1-3gm fat)** Skimmed milk powder Non-fat buttermilk (chaaj) Yogurt (plain) Paneer **Whole milk (buffalo)** Whole milk (cow's milk) Goat's milk Lassi, regular	 1 cup ¼ cup 2½ cups 1 cup ¼ cup (40 gms) (200 ml) <1 cup 1 cup (240 ml) 1 cup 1 cup

PREVIOUS STUDIES

Kaveriappan G et al. study (2024) [45] among 112 pregnant women and had found 21 as GDM cases. Among these, 95.2% exhibited HbA1c levels ≥5.7%. Of the cases, 38.4% were aged 21–25 years, and 48.2% were aged 26–30 years. The incidence of GDM was 20% in individuals of 26 years and older, with a HbA1c of ≥5.7%. In total, 86.6% were aged between 21 and 30 years, of which 23.7% of cases had a HbA1c level beyond 5.7; among these, 73.9% of cases had GDM, while none of the women with a HbA1c level below 5.7 acquired GDM. Among primigravida, 24.5% and 28.8% of cases exhibited HbA1c levels over 5.7%. Among these, 42.9% of primi gravida and 57.1% of multi gravida developed GDM during the later stages of pregnancy. Twenty-one participants acquired GDM with HbA1c levels exceeding 5.7%. These individuals had HbA1c assessed only after 9 weeks of gestational age, while women who had HbA1c evaluated before 9 weeks did not develop GDM. Therefore, a gestational period of 9 to 14 weeks is optimum for employing HbA1c in the diagnosis of GDM. Adverse pregnancy outcomes occur when HbA1c levels above 5.7%, but no patients experienced gestational diabetes mellitus or other adverse pregnancy outcomes with HbA1c levels below 5.7%. Consequently, HbA1c exceeds 5.7%.

Infants born to women with HbA1c ≥5.7%, regardless of the development of GDM, experience adverse foetal outcomes. Of the total cases, 51.8% of cases experienced normal vaginal birth, of which 84.4% of cases had HbA1c levels over 5.7%. A total of 25% of cases underwent primary lower segment caesarean delivery, of which 39.3% of cases had HbA1c levels over 5.7%; among these, 33.3% of cases were diagnosed with GDM, while 44.4% were not diagnosed with GDM. Among the subjects, 8.1% of cases underwent instrumental delivery, of whom 7.3% of cases had

HbA1c levels < 5.7%, whereas 27% of cases had HbA1c levels ≥5.7%. Among them, 8% of cases had GDM, while 22% of cases did not have GDM. Consequently, an HbA1c level of ≥5.7% is significant concerning the mode of birth, regardless of gestational diabetes mellitus status.

In the **Parsaei M et al.** study (2024), [46] the mean age of the cases was 32.6 ± 5.7 years, average gravidity was 1.1 ± 1.1, and parity was 0.8 ± 0.8. Of the total, 19% were diagnosed with GDM. The mean difference between GDM and Non GDM was significant regarding maternal age, BMI, nulligravida and nullipara. The early-pregnancy PLT and FBS levels were markedly raised in the GDM than the non-GDM. No notable changes were detected in other clinical or laboratory parameters between the two groups. In the univariate analysis, elevated maternal age and BMI were significant predictors of GDM occurrence. Furthermore, nulliparity was correlated with a reduced risk of GDM development. Moreover, the history of GDM and preeclampsia was predictive of GDM occurrence in the current pregnancy. The elevated PLT and FBS were predictive of the incidence of GDM.

Valadan M et al. study (2022) [47] found GDM in 16.4% of cases. Women with GDM had significantly greater age, elevated pre-gestational BMI, and increased weight gain throughout pregnancy compared to their non-GDM counterparts. In pregnant women with GDM, the mean HbA1c level was 5.45 ± 0.39, but in women without GDM, it was 4.96 ± 0.3. The utilization of HbA1c may reduce the necessity for OGTT in 40.4% of pregnant women, comprising 28.70% with HbA1c < 4.85 and 11.7% with HbA1c ≥ 5.45%. The sensitivity and specificity for diagnosing GDM at a HbA1c threshold of 5.45% were 54.8% and 96.8%, respectively, with a NPV of 91.5% and a PPV of 76.8%.

In the **Tripathy S, and Mohapatra S** study (2022), [48] the hemoglobin levels for non-GDM and GDM patients were 11.09 ±0.07, 11.47 ±0.14, respectively. The hematocrit PCV percentage for non-GDM and GDM was 33.85 ±0.28, 34.85 ±0.37%, respectively, which was significant difference. HbA1c readings for non-GDM were 5.18 ±0.04, while for GDM, they were 5.62 ±0.09. The thyroid levels for non-GDM and GDM were measured at 1.97 ±0.08 and 1.86 ±0.13, respectively. The cutoff value of HbA1c was 5.25%, and the PCV was 34.5%. HbA1c and PCV had a sensitivity of 64.4%, 36.1% and 58.6%, 43.1%, respectively. The combination of HbA1c and PCV demonstrated a sensitivity of 36.8% and a specificity of 85.4%.

Fonseca L et al. study (2021)[49] among 1,085 antenatal women assessed the average age as 32.9 ± 5.3 years. Concerning pre-pregnancy BMI, 34.5%, 34.6%, and 29.5% of the women were classified as having normal weight, overweight, and obesity, respectively. Of the total, 7.8% classified as preterm and average birth weight was 3.21 ± 4.95 g. Of these, 4.5% were classified as LGA, while 12.4% were categorized as SGA, and 4.8% were macrosomic. In comparing pregnant women with and without LGA offspring, those with LGA offspring exhibited a significantly higher prevalence of prior macrosomic newborns (24.5% and 4.4%) and an increased rate of excessive gestational weight gain (52.1% and 30.6%). Women with LGA newborns exhibited elevated FBS levels in the first trimester (92.0 and 85.0), increased glucose at baseline in the OGTT (93.5 and 85.0), a higher HbA1c in the third trimester (5.70 and 5.30), and a greater incidence of insulin therapy (59.2 and 53.3%).

Turan H et al. (2021) study [50] had observed that 21 antenatal women had abnormal test findings, had a mean gestational week of 25.4. Among the 21 pregnant women, five had GDM, and 10 showed normal glucose levels. Of the total, during the first trimester 4.5% had GDM and 6.5% during the second trimester, with an overall

prevalence of 11% among the pregnant women in the study. GDM was detected in 41.2% of cases during the first trimester and 58.8% during the second trimester, with a greater prevalence observed in pregnant women over 30 years of age. In the GDM group, a prior history of GDM, a familial history of diabetes mellitus, a previous occurrence of preeclampsia, and fasting blood glucose levels were seen at a significantly elevated rate compared to the non-GDM group. The GDM group had a markedly elevated incidence of large infants (>4000 g) in prior pregnancies compared to the non-GDM group.

In the **Cetin C et al.** study (2021) [51] among 195 pregnant women, 32 women (16.4%) were diagnosed with GDM. The incidence of GDM was high in those aged 35 and older; however. No statistically significant association was found between gravidity, parity, or abortion and the development of GDM. The incidence of GDM was elevated among those who were obese (37.5%) and overweight (25%) prior to pregnancy, compared to those normal (12%). The incidence of GDM was observed to be 3.8 times more in smokers compared to non-smokers. The average HbA1c level was 5.52% in individuals with GDM and 5.21% in those without GDM. Only 3.6% of the women exceeded 1st trimester HbA1c threshold, and all these with prediabetes developed GDM.

Pukale RS et al. study (2019) [52] involved 100 pregnant women with the mean age as 24.6 ± 2.57 years, and the average gestational age was 26.38 ± 1.11weeks. OGTT indicates average FBS was 113.48 ± 10.81, at one hour it was 181.14 ± 49.37, and after 2 hours, it was 183.34 ± 40.78. The average HbA1c level was 4.98 ± 0.8, and the OGCT was 166.87 ± 16.96. Patients screened for GDM with the OGCT resulted in about six cases identified as non-GDM when subsequently evaluated with the OGTT. Of the total cases tested by OGTT, about 99% were diagnosed with GDM

based on the fasting sample, 41% from the first-hour sample, and 81% from the second-hour sample. Compared to the 2nd-hour OGTT (81%), the 2nd-hour OGCT had identified 94% of GDM cases. Consequently, it was determined that approximately 6% of cases would be overlooked if only the DIPSI OGCT were employed. However, the use of both criteria results in a diagnosis of GDM with exceptionally high sensitivity and specificity.

Patil S, Sharma S study (2019), [53] reported the average maternal age as 24.22 ± 3.9 years and the average BMI as 23.7 ± 3.6 kg/m². Of the total cases, 5.71% had a familial predisposition to diabetes mellitus, whereas 64.57% were in the gestational age range of 24 to 32 weeks. The average birth weight of the neonate was 2.65 ± 0.45 kg. Among the total women, 5.71% of cases were positive for OGTT according to DIPSI criteria, of them 3.43% of cases had GDM. The sensitivity, specificity, PPV, and NPV of the DIPSI OGCT are 100%, 97.63%, 60%, and 100%, respectively. The majority of deliveries were via LSCS (60%), followed by vaginal delivery (40%). The average birth weight of neonates from DIPSI positive participants was 2.84 ± 0.79 kg. The occurrence of macrosomic neonates was greater in the DIPSI +ve study group (20%) than in the DIPSI -ve study group (3.57%). A significant association is observed with factors such as age ≥ 25 years, BMI ≥ 25 kg/m², family history of diabetes, polyhydramnios, pregnancy-induced hypertension, and NICU admission. In the GDM group, 66.67% of individuals underwent LSCS. The average birth weight of neonates in the GDM research group was 2.83 ± 0.48 kg. No neonate in the GDM study group exhibited macrosomia. Glycemic control with MNT was achieved in one participant with GDM (16.67%) and in all four subjects with DIPSI positive but OGTT negative results (100%). 66.67% of participants in the GDM research

group necessitated insulin in conjunction with MNT for optimal glycemic control. Metformin was administered to one patient with GDM (16.67%). [53]

Riaza M et al. study (2019) [54] had 11,430 individuals in the study. The average age of the cases was 27.2 ± 5.9 years, and the gestational age was 27.1 ± 6.8 weeks. A total of 29.1% of women were primigravida, and 70% of were multigravida. Of the patients, 4.1% pf cases were in the first trimester, 38% of cases were in the second trimester, and 54.6% of cases were in the third trimester of gestation. Among the total, 1.6% of cases were diagnosed with GDM. The majority of patients in the GDM group were aged 30 years or older. An elevated incidence was observed during the first trimester and among multigravida women.

In the **Desai GG et al.** study (2018),[55] the incidence of GDM by OGTT was 19%, and by DIPSI was 17.5%. The DIPSI test diagnosed a comparable percentage of GDM cases (17.5%) to the more complex and stricter OGTT criteria (19%). DIPSI exhibited a sensitivity of 86.8% and specificity of 98.8%, with a PPV of 94.3% and NPV of 97.0%, resulting in an overall diagnostic accuracy of 96.5%. The sensitivity of DIPSI, which is 86.8% relative to the usual 75 g OGTT, and its specificity, which is 98.8% compared to OGTT, both of which are equivalent. This comparison indicates that DIPSI can reliably substitute the traditional OGTT as a more straightforward, one-step screening method, alleviating concerns of underdiagnosing or over diagnosing GDM in the population.

In the **Sujithra D et al.** study (2018),[56] the mean POG was 7 ± 0.85 weeks. The average age and weight gain was 27.6 ± 4.67 years, and 14.12 ± 2.99 kg, respectively. A strong statistical correlation between HbA1c and GTT. The sensitivity and specificity of HbA1c was 70.4%, and 93.2%, respectively. Women with elevated HbA1c (≥5.7) were more predisposed to developing GDM compared to those with

lower HbA1c (≤5.7). This study found that advancing maternal age correlates with elevated HbA1C levels during pregnancy (≥5.7) and the onset of GDM. HbA1C can serve as a dependable instrument for forecasting GDM as early as 12 weeks of gestation.

In the **Amreen S et al.** study (2018),[57] 76 cases were found to be GDM, whereas the remaining 75 were classified as non-GDM based on the 75g OGTT. The average age of GDM cases was 30.4 ± 4.8 years, in contrast to 28.9 ± 4.1 years in the non-GDM. The current study reported the mean HbA1C to be 5.49 ± 0.5 in GDM and 5.1 ± 0.43 in non-GDM. The HbA1c level of 5.5% had a sensitivity of 80% and a specificity of 55.3%. Using a cutoff of 5.5% for screening, there were 57 positive cases and 94 negative cases. Among the screened patients, 42 were accurately diagnosed as GDM. The mean RBS level in women with GDM was 112±0.77 mg/dl, whereas the mean RBS in women without GDM was 91 ± 1.48 mg/dl. The screening in the first trimester yielded a sensitivity of 35.5% and a specificity of 94.7%. There were 31 screen-positive cases and 120 screen-negative cases. Of those screened, only four were inaccurately diagnosed with GDM. Among the screen-negative cohort, 49 women were not diagnosed.

In the **Gayam S et al.** study (2015),[58] the mean age was 24.07 ± 3.77 years. The average BMI was 25.5 ± 2.74 kg/m². As per DIPSI, the incidence of GDM in the first trimester was 4.6%, and overt diabetes was 0.7%. In the second trimester, 16 cases identified GDM positive during the first trimester became positive. Out of the remaining cases, 14.9% of cases were newly identified as GDM positive. Based on WHO criteria, 4% of cases of GDM occur in the first trimester, and 0.7% of cases are overt diabetes. During the second trimester, 23 women were lost to follow-up; of the remaining 263 women 12.5% of were newly diagnosed with GDM. In the third

trimester, 27 women were lost to follow-up, and 15.3% of cases were newly diagnosed with GDM. No significant link was observed between advancing age and the incidence of GDM across all three trimesters. A positive significant association was seen between elevated BMI and the incidence of GDM across all three trimesters.

MATERIALS AND METHODS

- **Study design:** Prospective Observational study
- **Study setting**: The current research was conducted at B.M. Patil Medical College and Hospital, Karnataka.
- **Duration of the study**: from JULY 2023 to APRIL 2025.
- **Study population**: Antenatal women with confirmed intrauterine pregnancy of gestational age who are attending to the Obstetrics and Gynecology OPD.
- **Sample size -**123

Sample size (n) = $(1.96)^2pq/d^2$

P= Prevalence = prevalence of GDM was 17.5% according to Desai GG et al. study[55]

q=100-p=100-17.5= 82.5%

d=absolute precision=7%

n= $(1.96)^2pq/d^2 = (1.96)^2\times17.5\times82.5/7^2$

=5546.31/49=113.19, which is taken as 114, but during the study period 123 antenatal came, all of them were considered as sample.

Inclusion criteria:

1. Antenatal women with a singleton pregnancy
2. Pregnant women of aged ≥18 years
3. Women in the first trimester
4. Not known diabetics
5. Not a known GDM cases in the previous pregnancy

Exclusion criteria:

1. Women with known Hypertension, diabetes, and GDM
2. Known systemic disease such as coronary heart diseases, liver diseases and renal diseases
3. Pregnant women age less than 18 years

Methodology

The current research was conducted after obtaining Institutional Ethics Committee permission (CTRl/2024/07/071464) and consent from the antenatal women after clearly explaining the study purpose and procedure.

Pregnant women attending the antenatal outpatient department or maternity ward during the 1st trimester with elevated risk factors for gestational diabetes mellitus were incorporated. Patients with a history of GDM in prior pregnancies, a family history of diabetes mellitus, a history of pre-mature delivery, fetal loss or unexplained neonatal demise, previous intrauterine death, polycystic ovarian syndrome, or anomalous infants in past pregnancies were classified as possessing high-risk factors for GDM. Antenatal women with a history of chronic renal disease, Hb variants, anaemia, cardiac and respiratory diseases, HbA1c levels exceeding 6.5, and those with a known diagnosis of DM were excluded. Venous blood was collected from all pregnant women in the first trimester with high-risk factors for fasting blood glucose, HbA1c, and all standard investigations. If the FBS exceeded 92 mg/dl or the HbA1c was below 6.5% during the first trimester, these values were deemed abnormal, prompting further evaluation in the second trimester with a repeat HbA1c and a 75 gm 2-hour DIPSI as a single-step protocol, regardless of the last meal. After giving 75 grams of anhydrous glucose dissolved in 250-300 millilitres of water, and plasma glucose levels were measured after two hours.[55] A number below 140 mg/dl was classified as normal, whereas a value beyond 200 mg/dl was diagnosed as overt diabetes. Any number ranging from 140 to 200 mg/dl was classified as impaired glucose tolerance.

Patients with HbA1c levels below 6.5% and a DIPSI value ranging from 140 to 200 mg/dl underwent a 75g OGTT to validate the diagnosis of GDM [53-55] based on

WHO 2013 criteria.[55] A comprehensive clinical history was documented, and an extensive physical examination was conducted at the time of presentation, focusing on the risk factors of GDM. All pregnant women received routine prenatal care, and treatment for gestational diabetes mellitus commenced solely in confirmed cases. The birth weight, NICU admission was also considered. The FBS level was measured to assess the continuation of elevated blood sugar.[53]

Statistical analysis

Data was analyzed using Microsoft Excel 11.0 and SPSS software version 26.0. The mean and SD of the quantitative variables were measured. The association was estimated using the chi-square or Fisher's exact test for categorical variables. P value ≤ 0.05 was taken as significant.

RESULTS

This study was conducted with total 123 antenatal cases with confirmed intrauterine pregnancy of gestational age who are attending to the Obstetrics and Gynecology OPD.

Table 10: Age (in years)

Age (in years)	Frequency	Percentage (%)
≤20	13	10.6
21-25	71	57.7
26-30	33	26.8
>30	6	4.9
Total	123	100

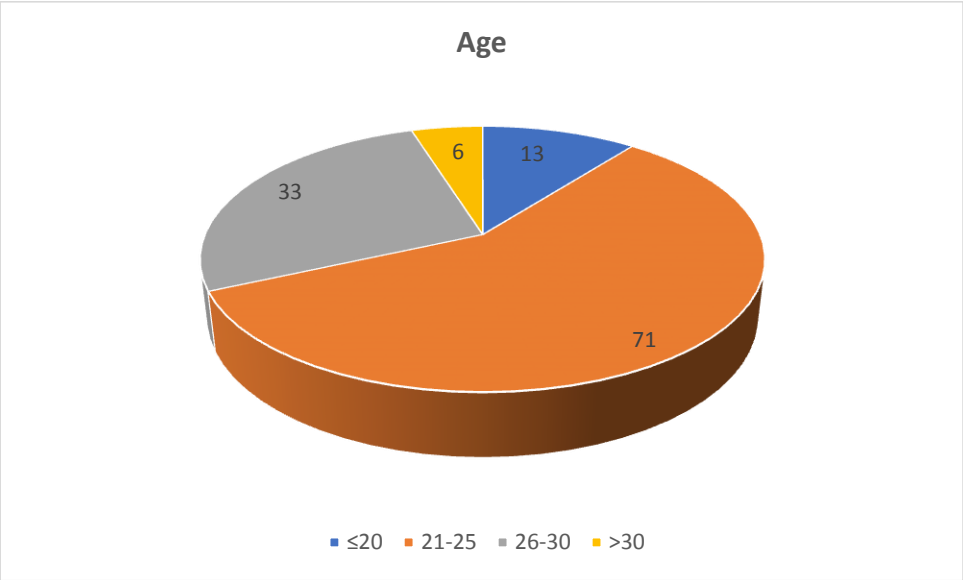


Figure 7: Age (in years)

Of the total cases, 13(10.6%) cases belonged to ≤20 years, 71(57.7%) cases were of 21-25 years, 33(26.8%) cases were of 26-30 years, and 6(4.9%) cases were of >30 years.

Table 11: BMI

BMI	Frequency	Percentage (%)
18.5-22.9	51	41.5
23-24.9	19	15.4
25-29.9	53	43.1
Total	123	100.0

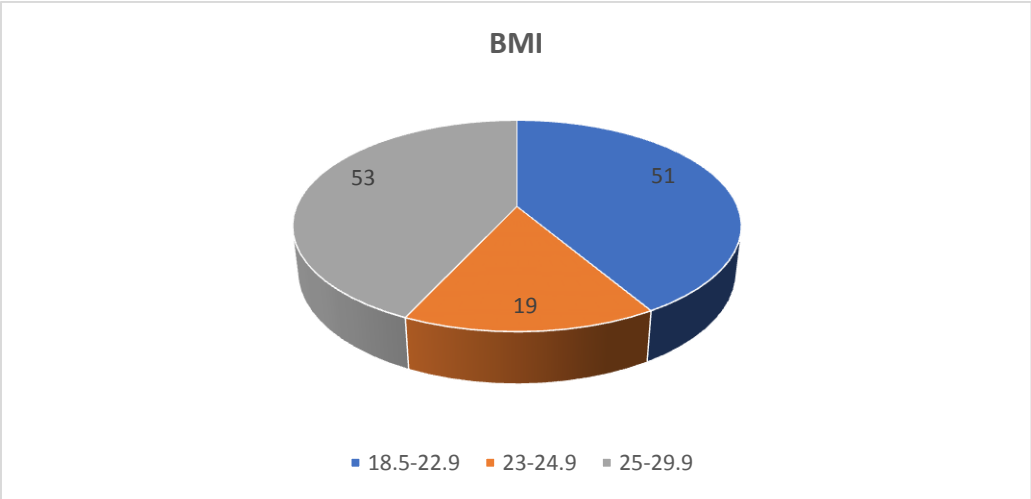


Figure 8: BMI

Of the total cases, BMI of 18.5-22.9 was seen in 51(41.5%) cases, BMI of 23-24.9 was seen in 19(15.4%) cases, and BMI of 25-29.9 was seen in 53(43.1%) cases.

Table 12: Mean of age, height, weight and BMI

Parameter	Mean	S.D
Age	24.42	3.38
Height	152.13	3.41
Weight	57.88	9.06
BMI	24.79	4.07

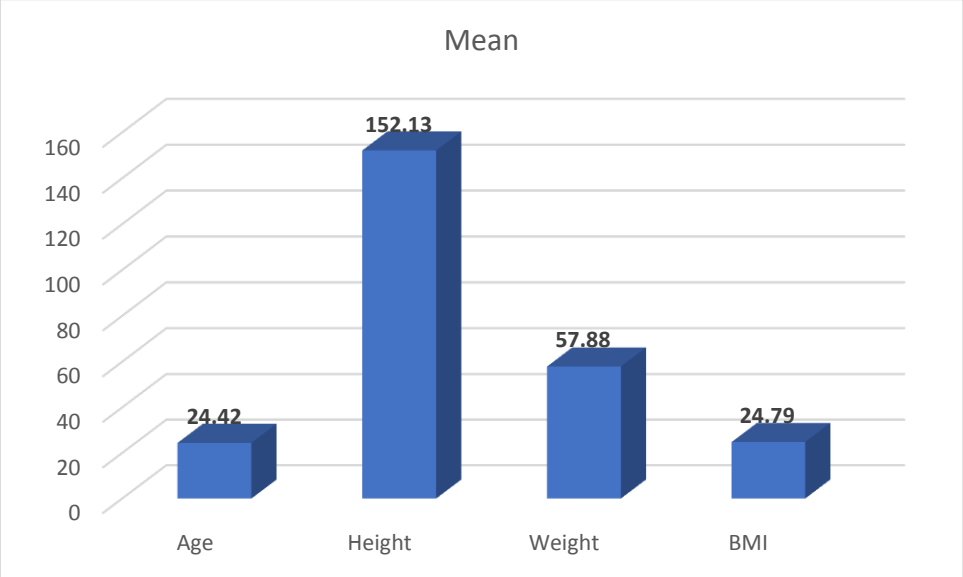


Figure 9: Mean of age, height, weight and BMI

The mean age of the total cases was 24.42 ± 3.38 years, height was 152.13 ± 3.41, weight was 57.88 ± 9.06, and BMI was 24.79 ± 4.07.

Table 13: Parity

Parity	Frequency	Percentage (%)
Primi	46	37.4
Multi	77	62.6
Total	123	100

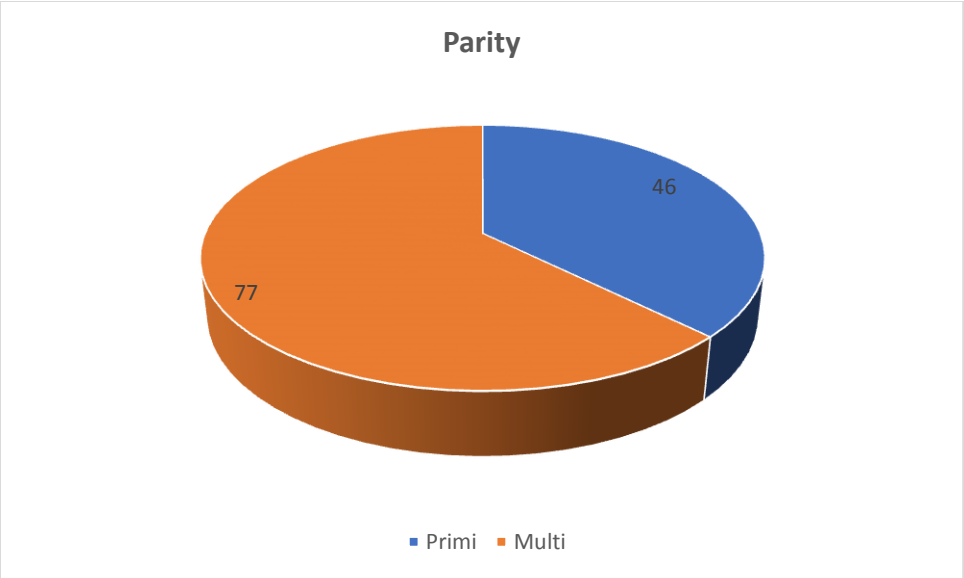


Figure 10: Parity

Of the total cases, 46(37.4%) cases were primis and 77(62.6%) cases were multi paras.

Table 14: FBS

FBS	Frequency	Percentage (%)
Normal	101	82.1
Abnormal	22	17.9
Total	123	100

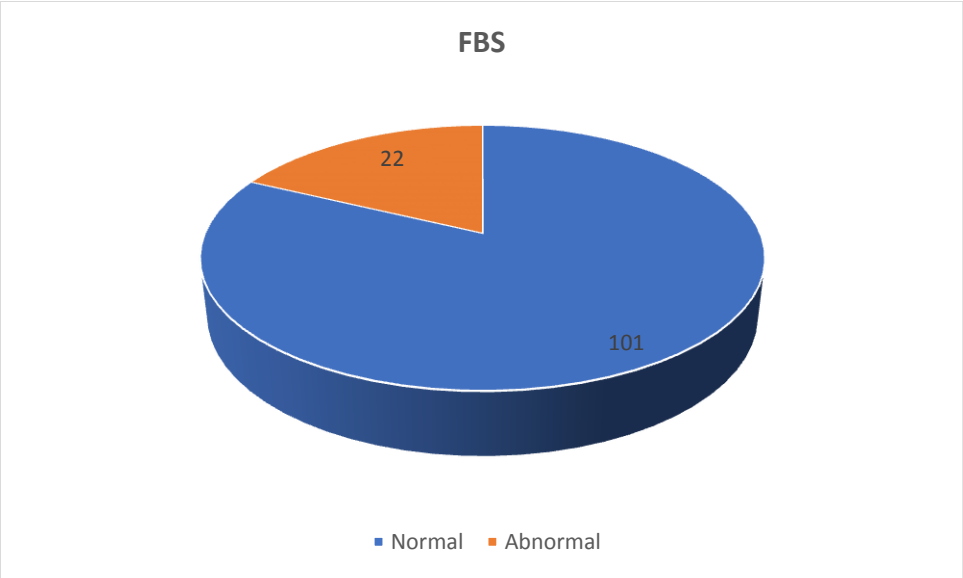


Figure 11: FBS

Of the total cases, 101 (82.1%) cases had normal and 22(17.9%) cases had abnormal FBS levels.

Table 15: Mean HbA1C (1st trimester)

Group	Mean HbA1C (1st trimester)	S.D	P value
Non GDM	4.99	0.46	**0.0001(significant)**
GDM	5.64	0.32	

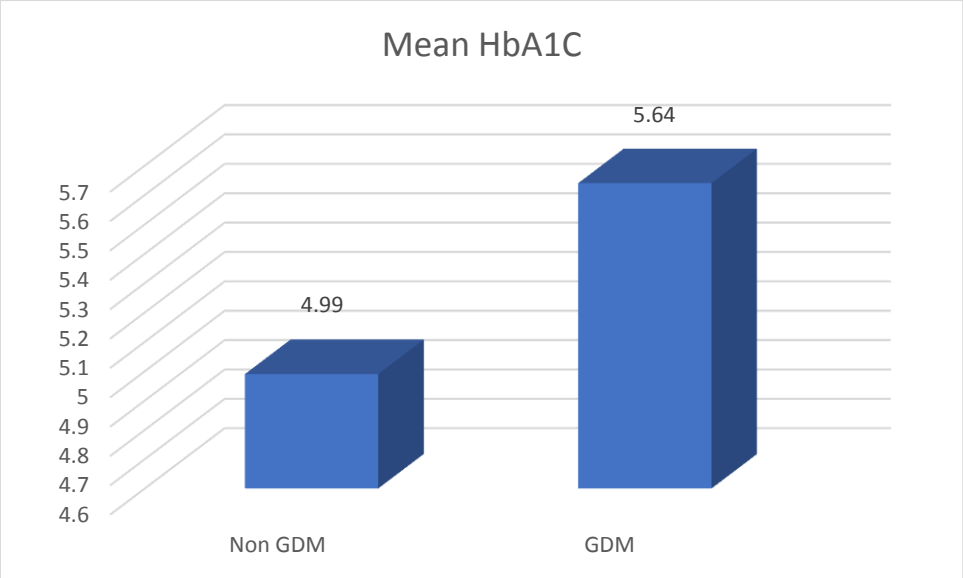


Figure 12: Mean HbA1C (1st trimester)

In the current research, mean HbA1C of non-diabetic was 4.99 ± 0.46, and diabetic was 5.64 ± 0.32 and this mean difference was significant.

Table 16: OGTT (2nd Trimester)

OGTT	Frequency	Percentage (%)
Normal	107	86.9
GDM	16	13.1
Total	123	100

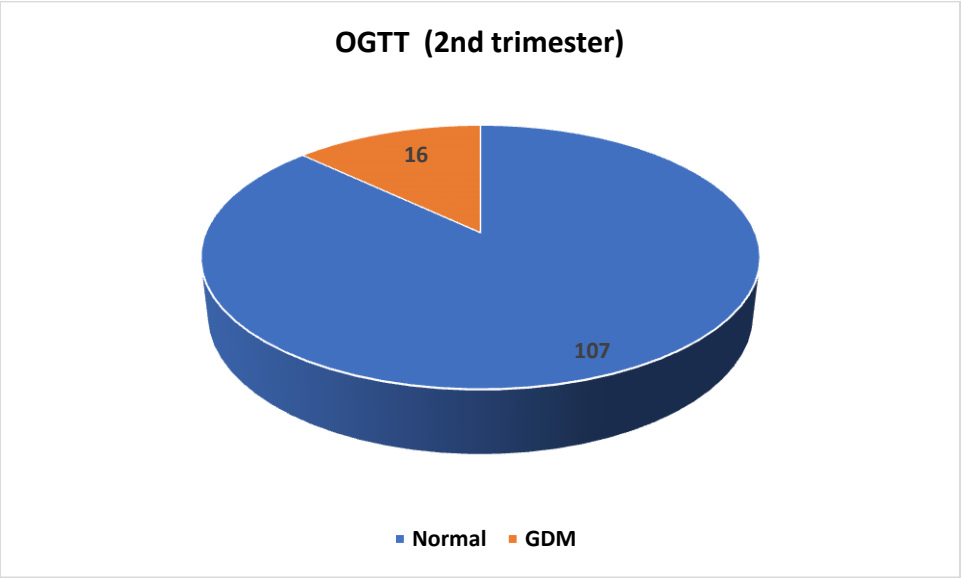


Figure 13: OGTT (2nd Trimester)

Of the total cases, based on 2nd Trimester OGTT, 107 (86.9%) cases were normal and 16 (13.1%) cases were GDM.

Table 17: Mean HbA1C (2nd trimester)

Group	Mean HbA1C (2nd trimester)	S.D	P value
Non GDM	5.19	0.41	**0.0001(significant)**
GDM	6.36	0.39	

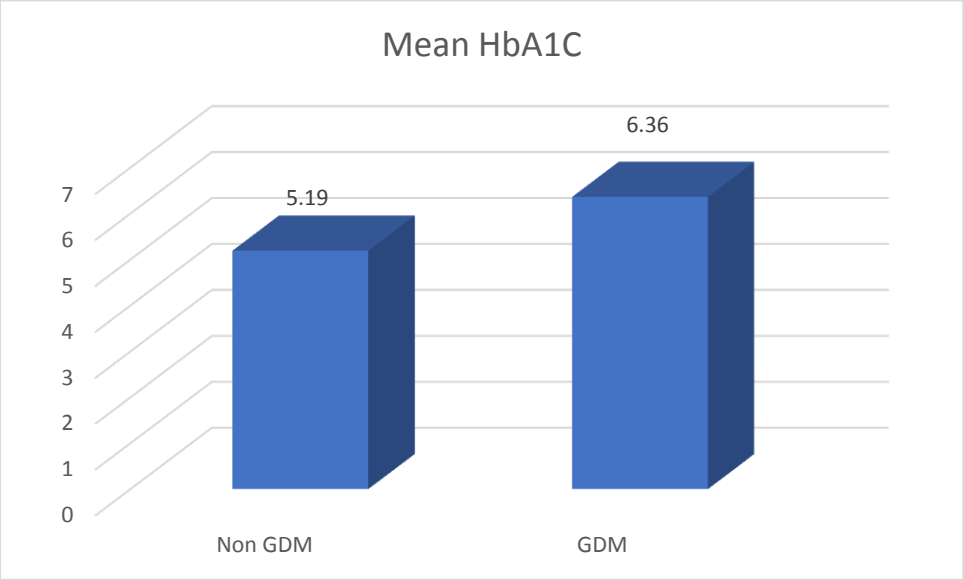


Figure 14: Mean HbA1C (2nd trimester)

In the current research, mean 2nd trimester HbA1C of diabetic was 6.36 ± 0.39, and non-diabetic was 5.19 ± 0.41, and this mean difference was significant.

Table 18: OGTT (3rd trimester)

OGTT	Frequency	Percentage (%)
Normal	107	86.9
GDM	16	13.1
Total	123	100

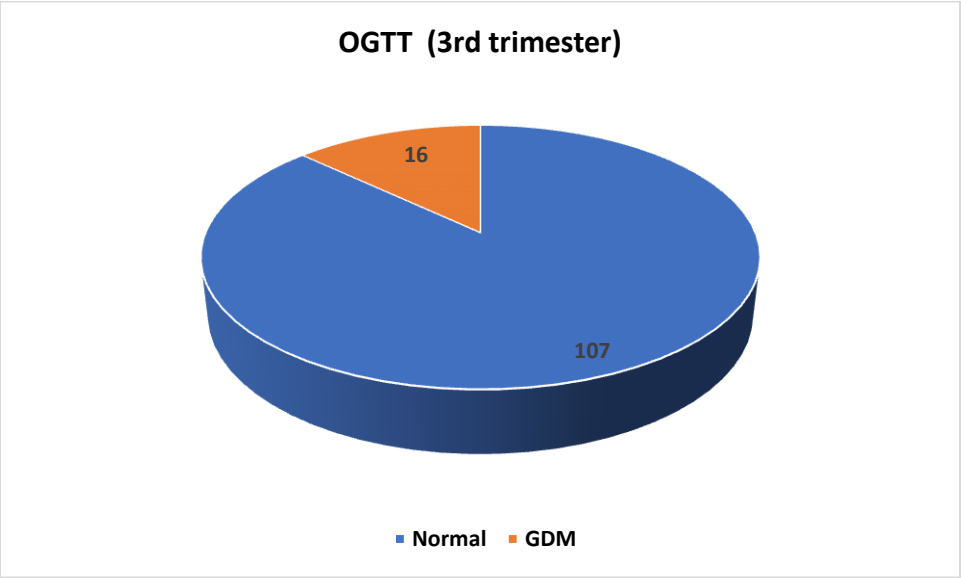


Figure 15: OGTT (3rd trimester)

OGTT (3rd trimester) was normal in 107(86.9%) cases, and GDM in 16(13.1%) cases.

Table 19: Mode of delivery

Mode of delivery		Frequency	Percentage (%)
Vaginal delivery	FTND	29	23.6
	FTVD	48	39
	PTVD	3	2.4
LSCS		43	35.0
Total		123	100.0

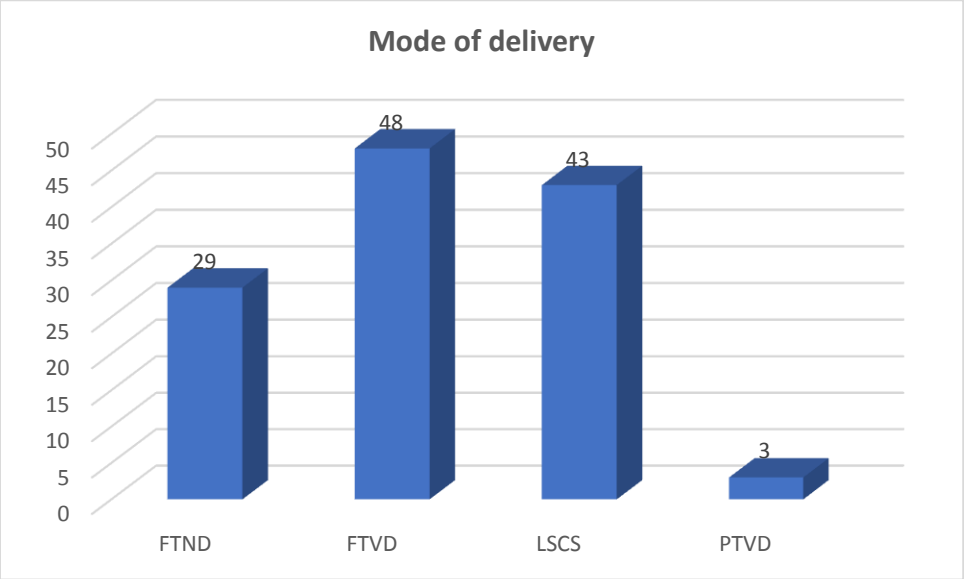


Figure 16: Mode of delivery

Of the total cases, FTND was seen in 29(23.6%) cases, FTVD was seen in 48(39%) cases, PTVD was seen in 3(2.4%) cases, and LSCS was seen in 43(35%) cases.

Table 20: Birth weight

Birth weight (in kgs)	Frequency	Percentage (%)
<2.5	4	3.3
≥2.5	119	96.7
Total	123	100.0

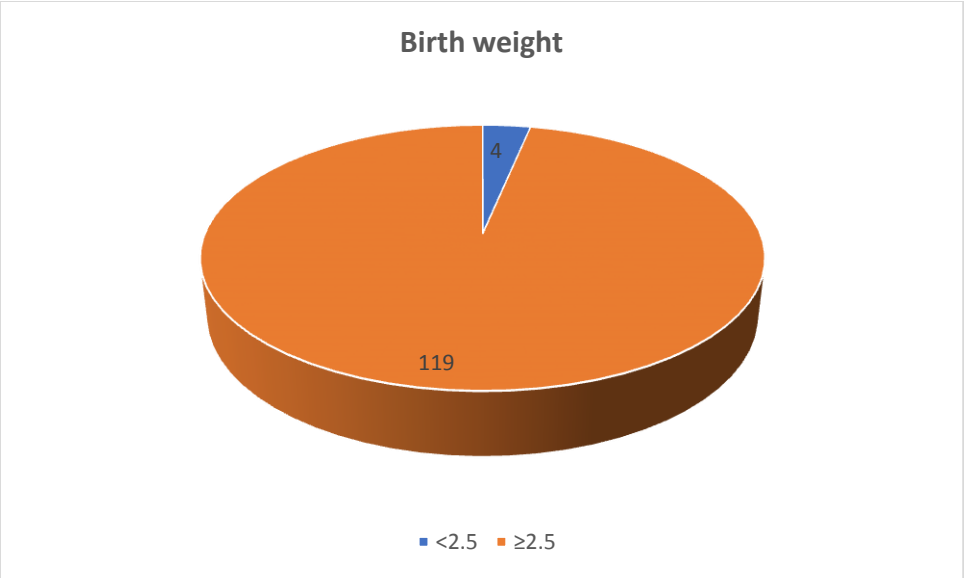


Figure 17: Birth weight

Of the total cases, 4(3.3%) cases had a birth weight of <2.5 kgs, and 119(96.7%) cases had a birth weight of ≥2.5 kgs.

Table 21: Maturity

Maturity	Frequency	Percentage (%)
Preterm	3	2.4
Term	120	97.6
Total	123	100.0

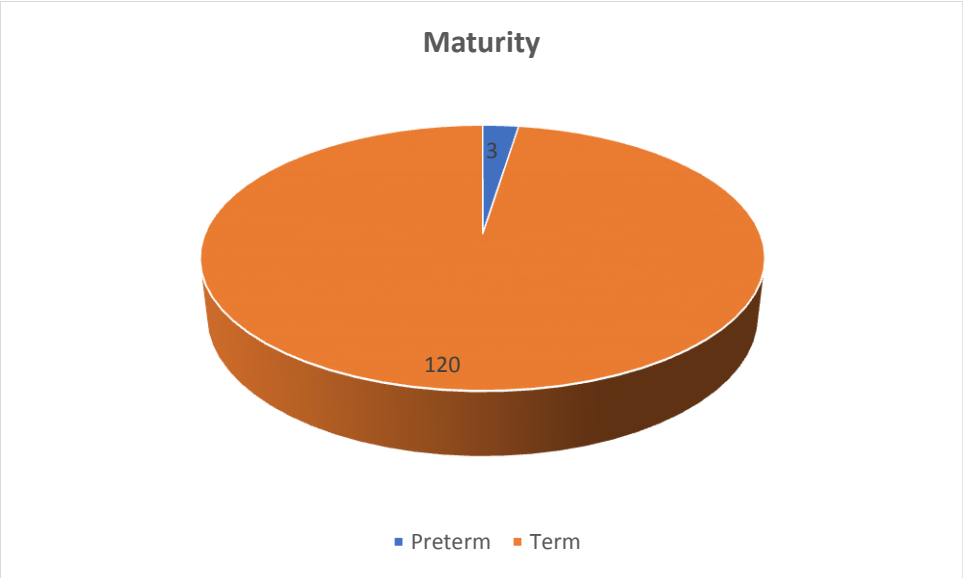


Figure 18: Maturity

Regarding maturity, 3(2.4%) cases had pre term, and 120(97.6%) cases.

Table 22: NICU admission

NICU admission	Frequency	Percentage (%)
Yes	35	28.5
No	88	71.5
Total	123	100.0

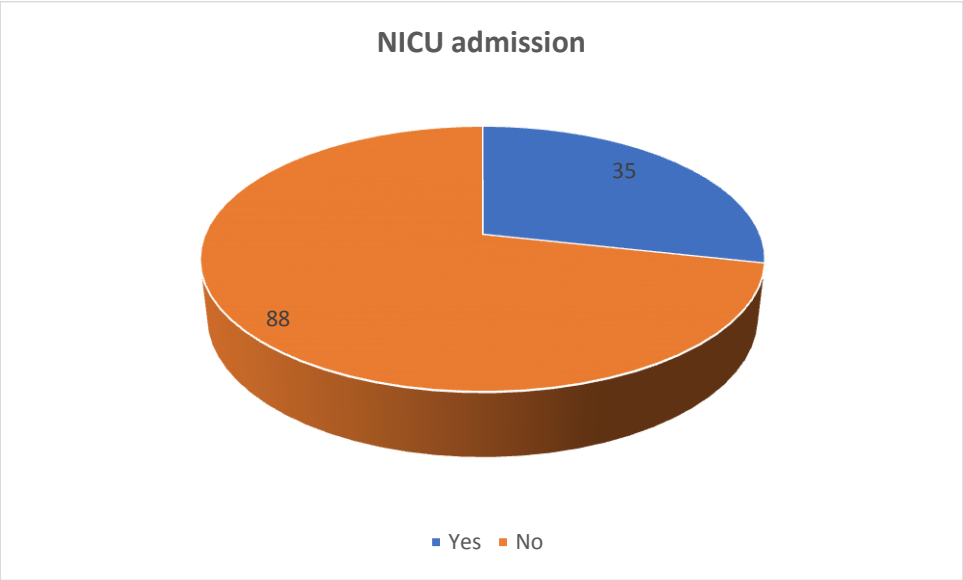


Figure 19: NICU admission

In this study, 35(28.5%) cases needed NICU admission.

Table 23: Comparison of mean age

Group	Mean age (years)	S.D	P value
Non GDM	24.35	3.4	0.76(non-significant)
GDM	24.63	3.54	

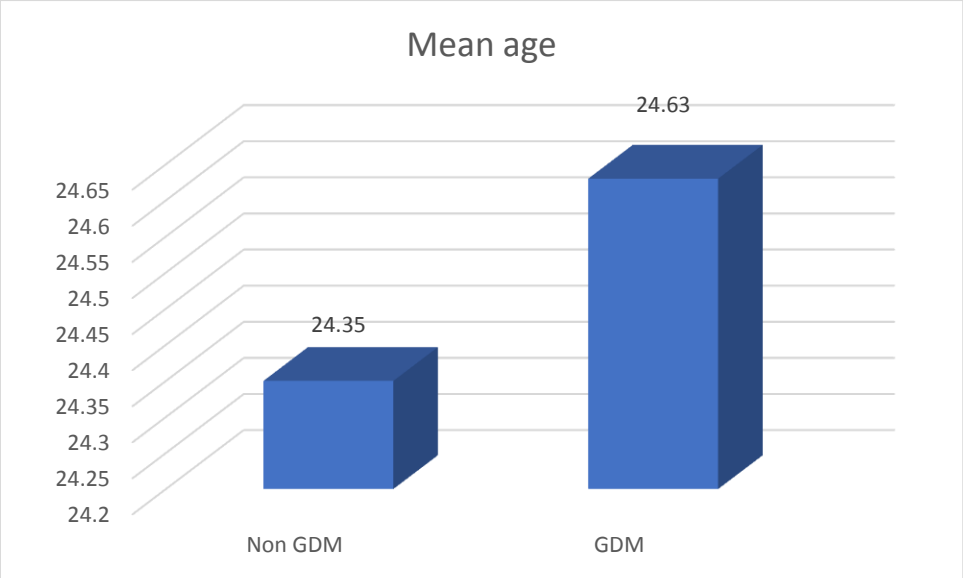


Figure 20: Comparison of mean age

In the current research, mean age of diabetic was 24.35 ± 3.4 years, and non-diabetic was 24.63 ± 3.54 years, and this mean difference was non-significant.

Table 24: Comparison of mean BMI (kg/m^2)

Group	Mean BMI	S.D	P value
Non GDM	24.58	3.89	0.14(non-significant)
GDM	26.18	5.02	

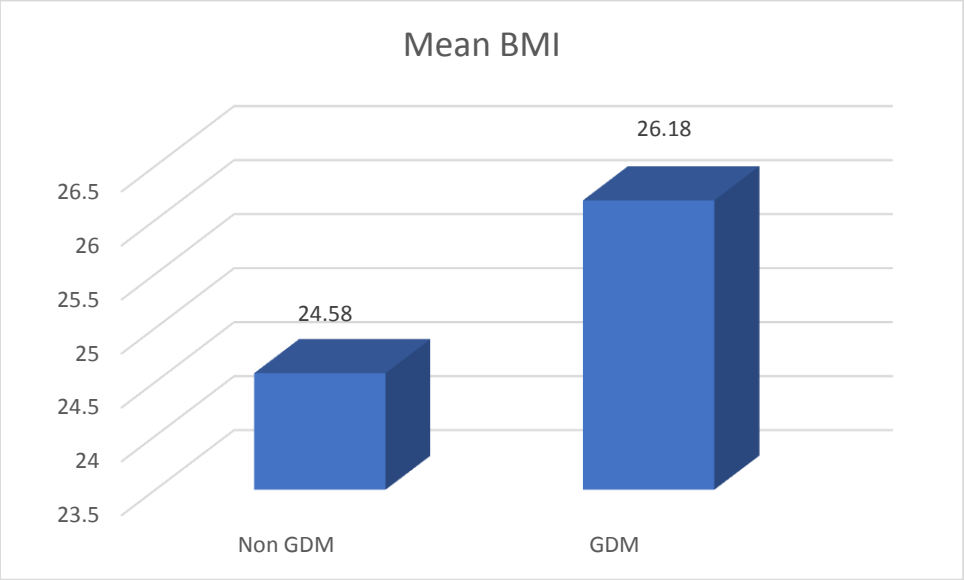


Figure 21: Comparison of mean BMI

In the current research, mean BMI of diabetic was 24.58 ± 3.89, and non-diabetic was 26.18 ± 5.02 and this mean difference was non-significant.

Table 25: Comparison of mean 2nd trimester HbA1C

Group	Mean 2nd trimester HbA1C	S.D	P value
Non GDM	5.19	0.41	**0.0001(significant)**
GDM	6.36	0.39	

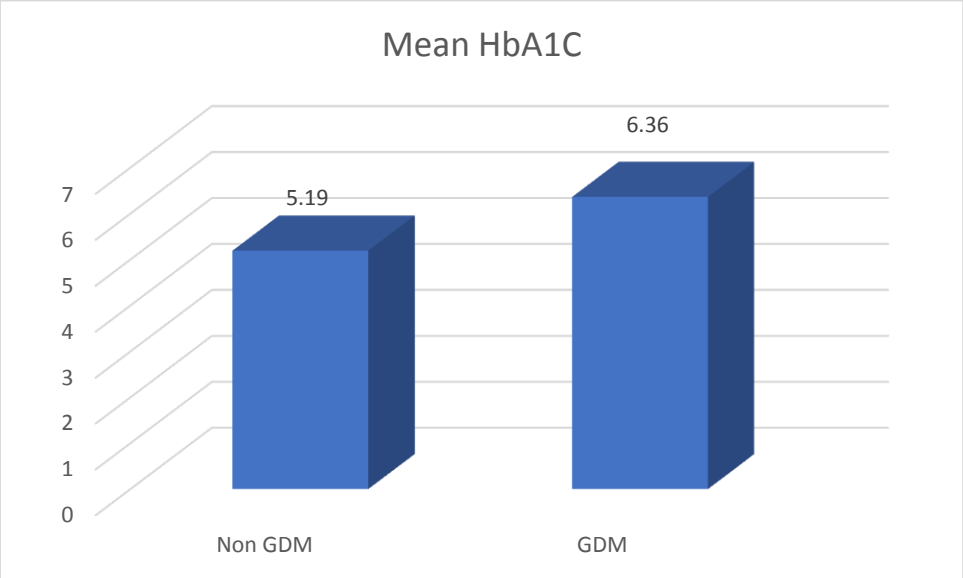


Figure 22: Comparison of mean 2nd trimester HbA1C

In the current research, mean 2nd trimester HbA1C of diabetic was 6.36 ± 0.39, and non-diabetic was 5.19 ± 0.41, and this mean difference was significant.

Table 26: Comparison of mean OGTT (3rd trimester)

Group	Mean OGTT	S.D	P value
Non GDM	100.47	12.38	**0.0001(significant)**
GDM	152.00	7.63	

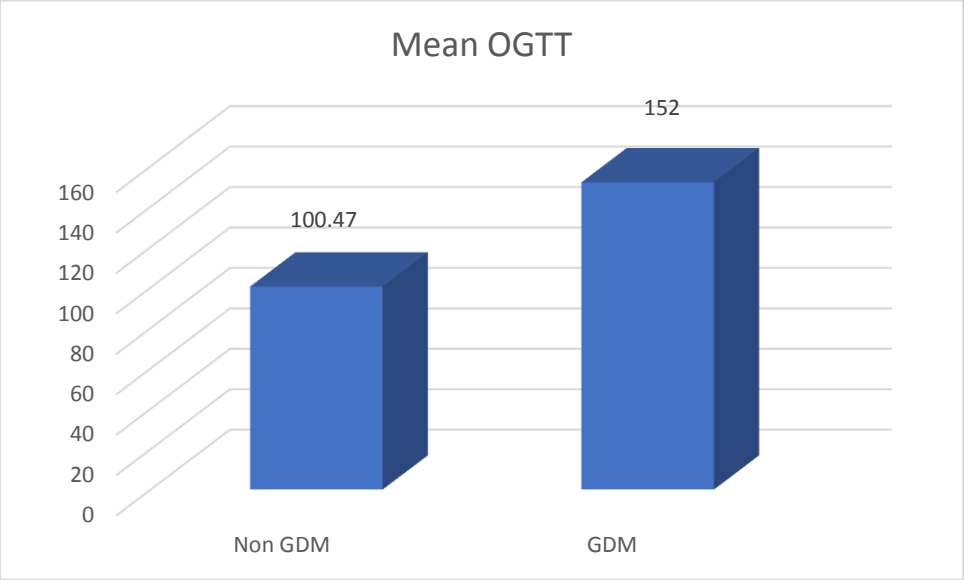


Figure 23: Comparison of mean OGTT (3rd trimester)

In the current research, mean OGTT of diabetic was 152 ± 7.63, and non-diabetic was 100.47 ± 12.38, and this mean difference was significant.

Table 27: Comparison of mean birth weight

Group	Mean birth weight (kg)	S.D	P value
Non GDM	2.94	0.26	**0.0001(significant)**
GDM	3.39	0.11	

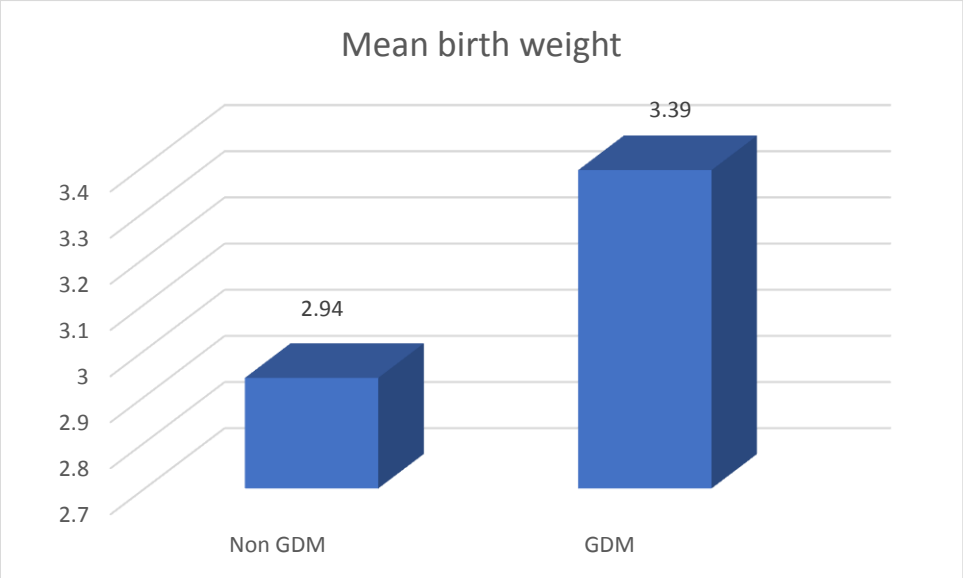


Figure 24: Comparison of mean birth weight

In the current research, mean birth weight of diabetic was 3.39 ± 0.11kgs and non-diabetic was 2.94 ± 0.26 kgs and this mean difference was significant.

Table 28: Age and OGTT (2nd trimester)

Age	OGTT (2nd trimester)		Total
	Non diabetic	Diabetic	
≤20	11 (10.3%)	2 (12.5%)	13 (10.6%)
21-25	64 (59.8%)	7 (43.8%)	71 (57.7%)
26-30	27 (25.2%)	6 (37.5%)	33(26.8%)
>30	5 (4.7%)	1 (6.3%)	6(4.9%)
Total	107 (87%)	16 (13%)	123(100%)

Fishers exact test 1.54; P value 0.67 (non-significant)

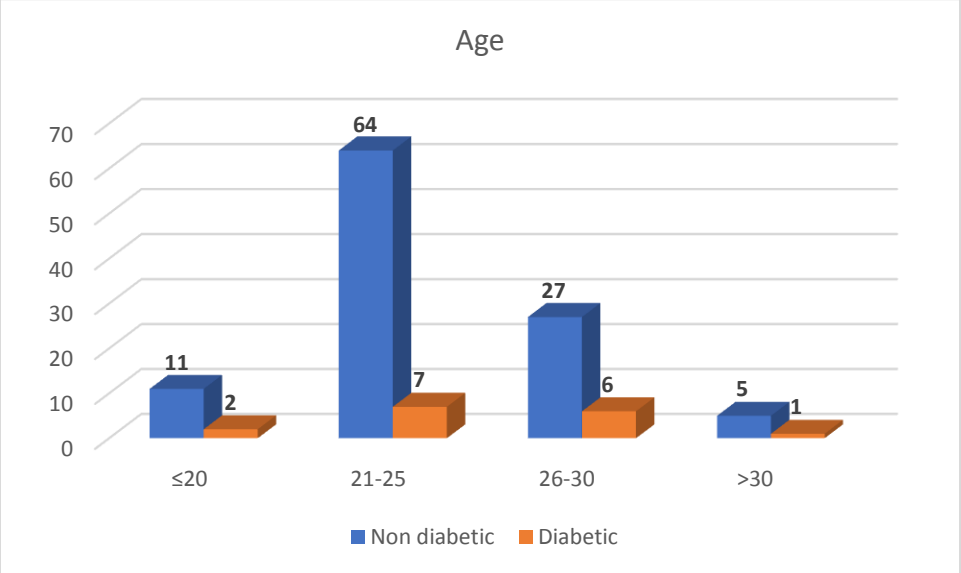


Figure 25: Age and OGTT (2nd trimester)

Of the non-diabetic, 11 (10.3%) cases were of ≤20 years, 64 (59.8%) cases were of 21-25 years, 27 (25.2%) cases were of 26-30 years, and 5 (4.7%) cases were of >30 years. Among the diabetic, 2 (12.5%) cases were of ≤20 years, 7 (43.8%) cases were of 21-25 years, 6 (37.5%) cases were of 26-30 years, and 1 (6.3%) case was of >30 years.

Table 29: BMI and OGTT (2nd trimester)

BMI	OGTT (2nd trimester)		Total
	Non diabetic	Diabetic	
18.5-22.9	44 (41.1%)	7 (43.8%)	51 (41.5%)
23-24.9	19 (17.8%)	0	19 (15.4%)
25-29.9	44 (41.1%)	9 (56.2%)	53 (43.1%)
Total	107 (87%)	16 (13%)	123(100%)

Fishers exact test 3.61; P value 0.17 (non-significant)

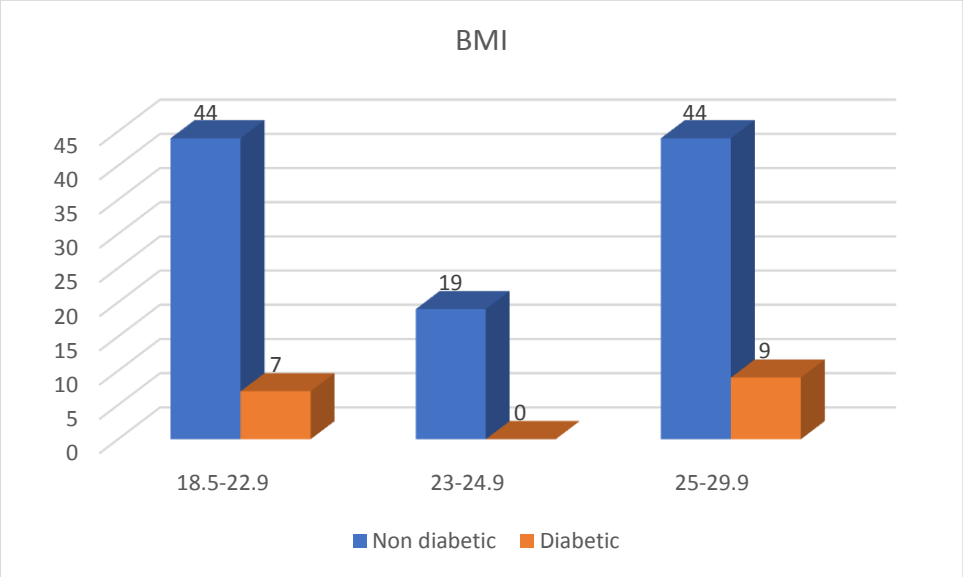


Figure 26: BMI and OGTT (2nd trimester)

Of the cases belonged to 18.5-22.9 BMI, 44 (41.1%) cases were non-diabetics, and 7 (43.8%) cases were diabetic; Of the 23-24.9 BMI, 19 (17.8%) cases were non-diabetic, and none of the cases were diabetic; and of the BMI of 25-29.9, 44 (41.1%) cases were non-diabetic and 9 (56.2%) cases were diabetic.

Table 30: Parity and OGTT (2nd trimester)

Parity	OGTT (2nd trimester)		Total
	Non diabetic	Diabetic	
Primi	43 (40.2%)	3 (18.7%)	46 (37.4%)
Multi	64 (59.8%)	13 (81.3%)	77 (62.6%)
Total	107 (87%)	16 (13%)	123(100%)

Fishers exact test 2.73; P value 0.99 (non-significant)

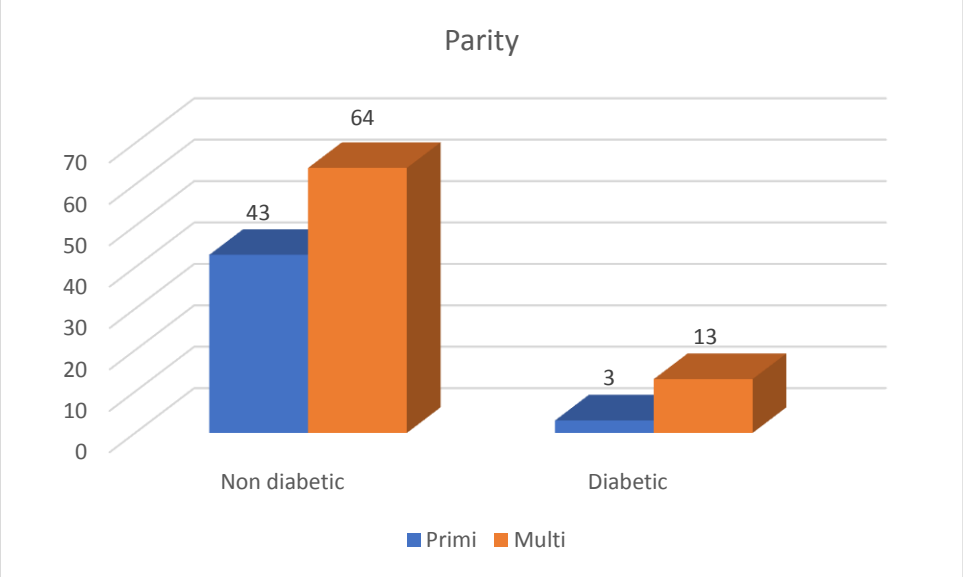


Figure 27: Parity and OGTT (2nd trimester)

Of the cases belonged to primi para, 43 (40.2%) cases were non-diabetic and 3 (18.7%) cases were diabetic, while among multi para cases, 64 (59.8%) cases were non-diabetic and 13 (81.3%) cases were diabetic.

Table 31: FBS and OGTT (2nd trimester)

FBS	OGTT (2nd trimester)		Total
	Non diabetic	Diabetic	
Abnormal	10 (9.3%)	12 (75%)	22 (17.9%)
Normal	97 (90.7%)	4 (25%)	101 (82.1%)
Total	107 (87%)	16 (13%)	123(100%)

Fishers exact test 40.85; **P value 0.0001 (Significant)**

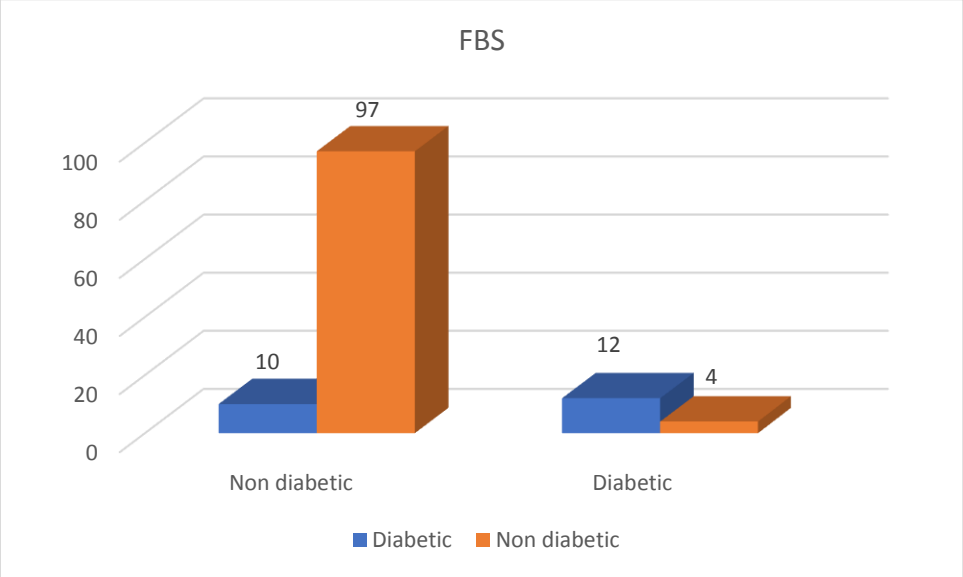


Figure 28: FBS and OGTT (2nd trimester)

Of the total cases identified as abnormal values for FBS, 10 (9.3%) cases were non-diabetic and 12 (75%) cases were diabetic as per HBA1C, and among the cases identified as normal values for FBS, 97 (90.7%) cases were non-diabetic and 4 (25%) cases were diabetic as per HBA1C. The association between FBS and HbA1C was significant.

Table 32: Mode of delivery and OGTT (2nd trimester)

Mode of delivery	OGTT (2nd trimester)		Total
	Non diabetic	Diabetic	
LSCS	28 (26.2%)	15 (93.7%)	43 (35%)
Vaginal delivery	79 (73.8%)	1 (6.3%)	101 (82.1%)
Total	107 (87%)	16 (13%)	123(100%)

Fishers exact test 27.96; **P value 0.002 (Significant)**

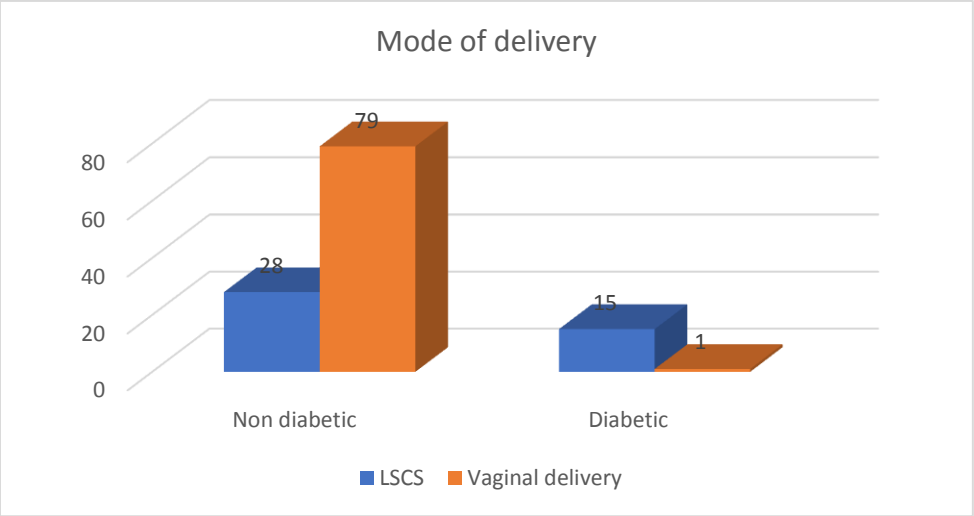


Figure 29: Mode of delivery and OGTT (2nd trimester)

Of the total cases, 28 (26.2%) non diabetic cases and 15 (93.7%) diabetic cases had LSCS deliveries, while 79 (73.8%) non diabetic cases and 1 (6.3%) diabetic case had vaginal deliveries. The association between mode of deliveries and HbA1C was significant.

Table 33: Birth weight and OGTT (2nd trimester)

Birth weight	OGTT (2nd trimester)		Total
	Non diabetic	Diabetic	
<2.5	4 (3.7%)	0	4 (3.3%)
≥2.5	103 (96.3%)	16 (100%)	119 (96.7%)
Total	107 (87%)	16 (13%)	123(100%)

Fishers exact test 0.62; P value 0.43 (non-significant)

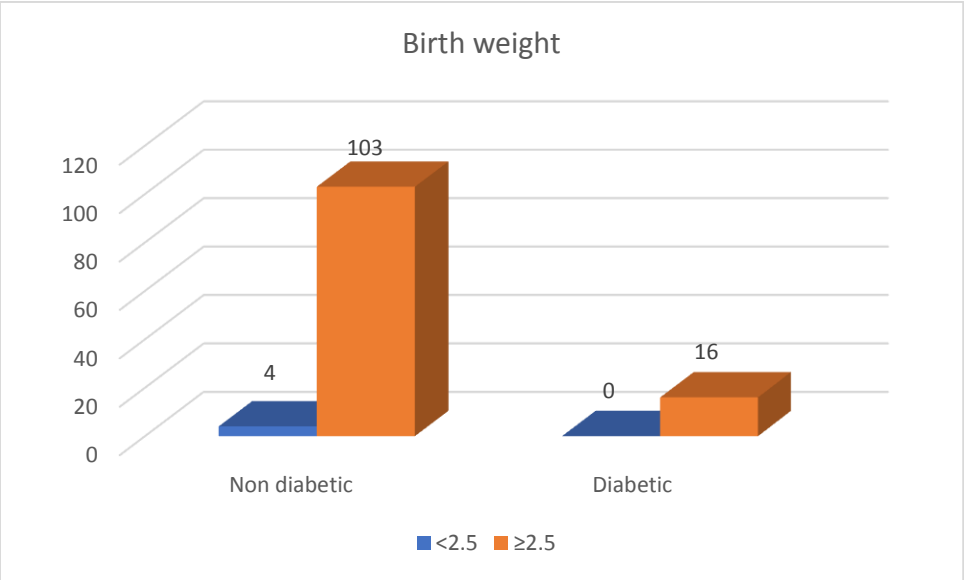


Figure 30: Birth weight and OGTT (2nd trimester)

Of the total, 4 (3.7%) non diabetic cases and none of the diabetic cases had birth weight of <2.5 kgs, while 103 (96.3%) non diabetic cases, and 16 (100%) diabetic cases were of birth weight of ≥2.5kgs.

Table 34: Maturity and OGTT (2nd trimester)

Maturity	OGTT (2nd trimester)		Total
	Non diabetic	Diabetic	
Pre term	3 (2.8%)	0	3 (35%)
Term	104 (97.2%)	16 (100%)	120 (82.1%)
Total	107 (87%)	16 (13%)	123(100%)

Fishers exact test 0.46; P value 0.49 (non-significant)

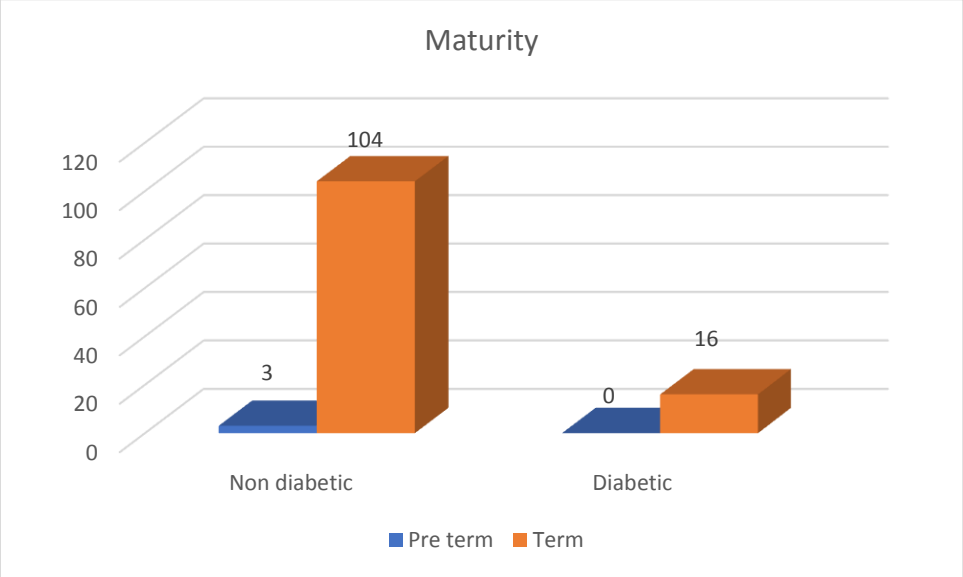


Figure 31: Maturity and OGTT (2nd trimester)

Of the total cases, 3 (2.8%) non diabetic cases and none of the diabetic cases had pre term deliveries, while 104 (97.2%) non diabetic cases and 16 (100%) diabetic cases had term deliveries.

Table 35: NICU and OGTT

NICU	OGTT (2nd trimester)		Total
	Non diabetic	Diabetic	
Yes	21 (19.6%)	14 (87.5%)	35 (28.5%)
No	86 (80.4%)	2 (12.5%)	88 (71.5%)
Total	107 (87%)	16 (13%)	123(100%)

Fishers exact test 31.49; P value 0.0001 (Significant)

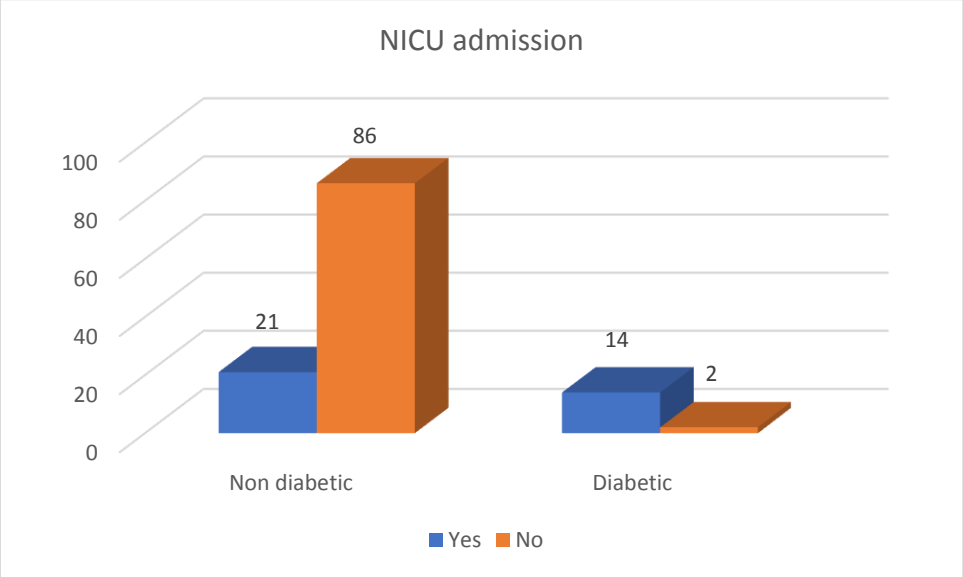


Figure 32: NICU and OGTT (2nd trimester)

Regarding NICU admission, 21 (19.6%) non diabetic cases and 14 (87.5%) diabetic cases needed NICU admission.

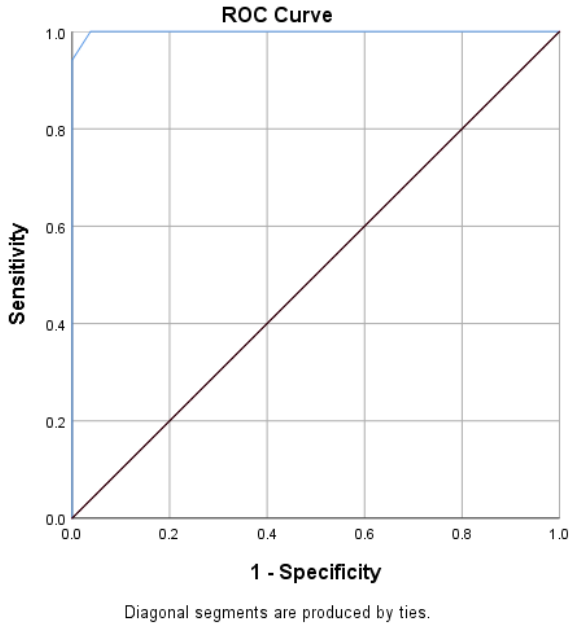


Figure 33: ROC curve of 1st trimester HbA1C for diagnosing GDM

Regarding diagnosing GDM, 1st trimester HbA1C had a sensitivity of 97.1%, and specificity of 100% with 5.61 as a cut off level of HbA1C to diagnose as diabetic.

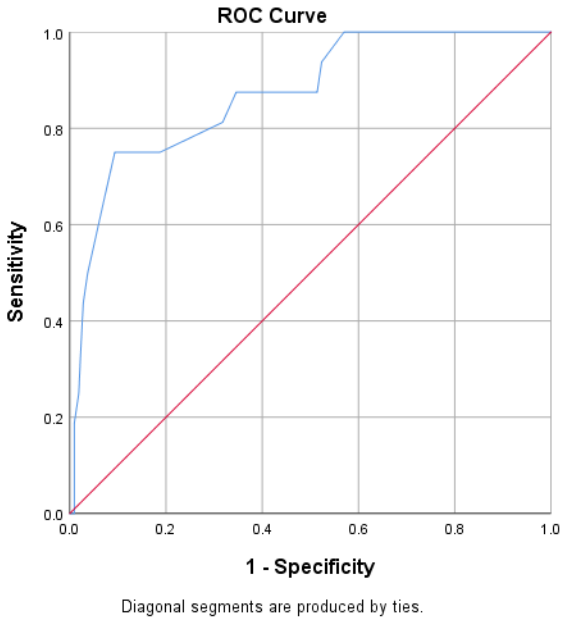


Figure 34: ROC curve of FBS for diagnosing GDM

Regarding diagnosing GDM, FBS had a sensitivity of 93.8%, and specificity of 52.8% with 86.5 as a cut off level of FBS to diagnose as diabetic.

Table 36: Correlation between FBS and 1st Trimester HbA1C

	FBS (mg/dL)	1st Trimester HbA1C
Pearson Correlation	1	0.518
P-value		0.0001(significant)

There was a significant correlation between FBS and 1st trimester HbA1C.

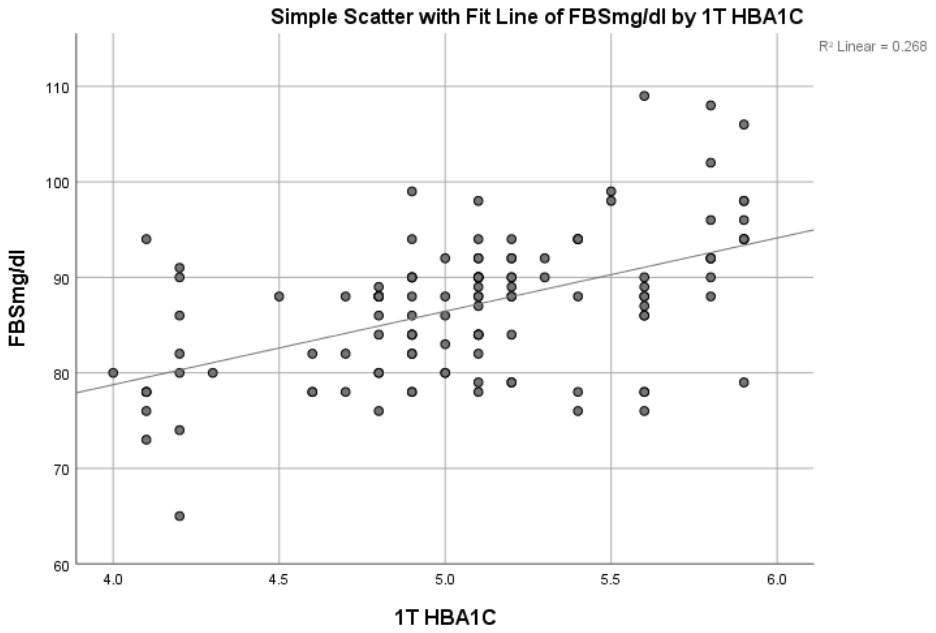


Figure 35: Correlation between FBS and 1st Trimester HbA1C

Table 37: Correlation between FBS and 2nd Trimester HbA1C

	FBS (mg/dL)	2nd Trimester HbA1C
Pearson Correlation	1	0.523
P-value		0.0001(significant)

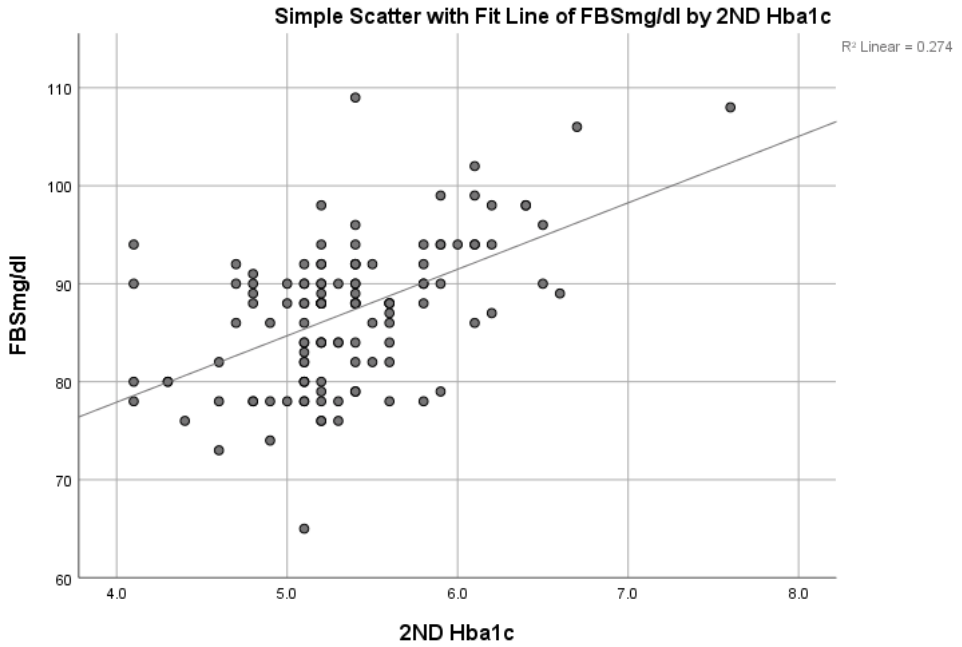


Figure 36: Correlation between FBs and 2nd Trimester HbA1C

There was a significant correlation between FBS and 2nd Trimester HbA1C.

DISCUSSION

The present observational study was done among 123 Antenatal women attended Obstetrics and Gynecology OPD in S.H.R.I. B.M. PATIL Medical College Hospital and Research Centre, Vijayapura to determine the efficacy of HbA1c as a predictor for early diagnosis of gestational diabetes mellitus.

Incidence of GDM

Of the total cases, 13% of cases were diabetic, similar to the studies by Shrivastava N et al.[59] (13.6%), Balaji V et al.[60] (13.4%), Sridhar GR et al.[61] (12.7%), and Tong JN et al.[62] (14.4%).

The variation in the findings was due to the various GDM thresholds for diagnosis, and varied screening OGTTs used in the above studies.

Table 38: Incidence of GDM

Research name	Incidence
Shrivastava N et al. [59]	13.6%
Balaji V et al. [60]	13.4%
Sridhar GR et al. [61]	12.7%
Tong JN et al. [62]	14.4%
Current research	13%

Age

Of the total cases, 10.6% cases belonged to ≤20 years, 57.7% of cases were of 21-25 years, 26.8% of cases were of 26-30 years, and 4.9% of cases were of >30 years.

In the study by Rajputa R et al.,[63] 18.1% of cases were of 16-20 years, 58.2% of cases were of 21-25 years, 19.9% of cases were of 26-30 years, and 3.8% of cases were of >30 years.

Desai GG et al. study [55] had 49.5% of cases in 20-25 years, 26.5% of cases in 26-30 years, 18.5% of cases in 31-35 years, and 5.5% of cases in >35 years.

Shrivastava N et al. study[59] had 67.8% of cases in 18-25 years, 26% of cases in 26-30 years, and 6.2% of cases in >30 years.

In the current research, the mean age of the total cases was 24.42 ± 3.38 years, which was similar to the studies by Shrivastava N et al. [59] (24.34 ± 3.7), Pukale RS et al. [52] (24.6±2.57), Singh A et al.[64] (25.71 ± 3.39), and Sujithra D et al. [56] (27.6 ± 4.67).

Table 39: Average age

Research name	Average age
Shrivastava N et al. [59]	24.34 ± 3.7
Pukale RS et al. [52]	24.6±2.57
Singh A et al. [64]	25.71 ± 3.39
Sujithra D et al. [56]	27.6 ± 4.67
Current research	24.42 ± 3.38

This variation in the findings was because of differences in sample sizes, and demographic differences of the above studies.

Average age and GDM

In the current research, mean age of diabetic was 24.35 ± 3.4, and non-diabetic was 24.63 ± 3.54, which was in accordance with the studies by Singh A et al. [64] (25.52 ± 3.19, 25.82 ± 3.52), Tripathy S, and Mohapatra S[48] (27.0 ± 2.87, 26.44 ± 2.34), lesser than the studies by Valadan M et al. [47] (32.64 ± 5.49, 30.64 ± 5.17), Tong JN et al. [62] (32.52 ± 4.22, 30.57 ± 3.94), and Turan H et al. [50] (33.0 ± 5.5, 27.9 ± 5.2).

Table 40: Average age of diabetic and non-diabetic

Research name	Average age of diabetic	Non diabetic
Singh A et al. [64]	25.52 ± 3.19	25.82 ± 3.52
Tripathy S, and Mohapatra S[48]	27.0 ± 2.87	26.44 ± 2.34
Valadan M et al. [47]	32.64 ± 5.49	30.64 ± 5.17
Tong JN et al. [62]	32.52 ± 4.22	30.57 ± 3.94
Current research	24.35 ± 3.4	24.63 ± 3.54

The mean age difference was non-significant in the current research, similar to the studies by Singh A et al., [64] Tripathy S, and Mohapatra S, [48] but contrast to the studies by Valadan M et al., [47] and Tong JN et al. [62]

Different findings regarding mean age of this study than the other studies were because of difference inclusion criteria of the above studies.

BMI

Of the total cases, normal BMI was seen in 41.5% of cases, over weight was seen in 15.4% of cases, and Obesity was seen in 43.1% of cases.

In the Fonseca L et al. study, [49] underweight was seen in 1.5% of cases, 34.5% of cases in normal BMI, 34.6% of cases were seen in over-weight, and 29.5% of cases were seen in obese.

In the Singh A et al. study, [64] 11.3% of cases had underweight, 68.8% of cases were of normal BMI, 19.4% of cases were of overweight and 0.6% of cases were obese.

In the Parsaei M et al. study, [46] 4.3% of cases were under weight, 45.8% of cases were of normal BMI, 32.8% of cases were of overweight, and 15.9% of cases were obese.

In the study by Rajputa R et al.,[63] 38.2% of cases were underweight, 53.6% were of normal BMI, and 8.2% of cases were of obese.

Table 41: BMI

Research name	BMI of Under-weight, Normal, over-weight, Obese
Fonseca L et al. [49]	1.5%, 34.5%, 34.6%, 29.5
Singh A et al. [64]	11.3%, 68.8%, 19.4%, 0.6%
Parsaei M et al. [46]	4.3%, 45.8%, 32.8%, 15.9%
Rajputa R et al.[63]	38.2%, 53.6%, 0% 8.2%
Current research	41.5%, 15.4%, 43.1%

The mean BMI of the cases in the current research was 24.79 ± 4.07, similar to the studies by del Val TL et al. [65] (24.9 ± 4.6), Fonseca L et al. [49] (26.5 ± 5.6), and Haddad AS et al.[66] (36.2 ± 7.7).

Table 42: Average BMI

Research name	Average BMI
Fonseca L et al. [49]	26.5 ± 5.6
del Val TL et al. [65]	24.9 ± 4.6
Haddad AS et al.[66]	36.2 ± 7.7
Current research	24.79 ± 4.07

Average BMI comparison

In the current research, mean BMI of diabetic was 24.58 ± 3.89, and non-diabetic was 26.18 ± 5.02, which was in accordance with the studies by Singh A et al. [64] (22.81 ± 3.51, 22.03 ± 2.57), Valadan M et al. [47] (27.23 ± 3.93, 24.89 ± 2.93), Turan H et al. [50] (26.0 ± 4.1, 24.7 ± 4.2), Tripathy S, and Mohapatra S[48] (30.10 ± 2.33, 26.54 ± 3.45), and Tong JN et al. [62] (21.69 ± 3.01, 20.5 ± 2.56).

Table 43: Comparison of Average BMI in diabetic and non-diabetic

Research name	Average BMI	
	Diabetic	Non diabetic
Singh A et al. [64]	22.81 ± 3.51	22.03 ± 2.57
Valadan M et al. [47]	27.23 ± 3.93	24.89 ± 2.93
Turan H et al. [50]	26.0 ± 4.1	24.7 ± 4.2
Tripathy S, and Mohapatra S[48]	30.10 ± 2.33	26.54 ± 3.45
Current research	24.58 ± 3.89	26.18 ± 5.02

The mean BMI difference was non-significant in the current research, contrast to the studies by Valadan M et al., [47] and Turan H et al., [50] Singh A et al., [64] and Tripathy S, and Mohapatra S. [48]

Parity

Of the total cases, 37.4% of cases were primis and 62.6% of cases were multi paras, which was in accordance with the studies by Shrivastava N et al. [59] (39%, 61%), Singh A et al. [64] (45%, 55%), del Val TL et al.[65] (42.5%, 57.5%), and Parsaei M et al. [46] (38%, 60.2%).

Table 44: Parity

Research name	Parity (Primi, Multi)
Shrivastava N et al. [59]	39%, 61%
Singh A et al. [64]	45%, 55%
del Val TL et al. [65]	42.5%, 57.5%
Parsaei M et al. [46]	38%, 60.2%
Current research	37.4%, 62.6%

FBS

Of the total cases, 82.1% of cases had normal and 17.9% of cases had abnormal FBS levels, similar to the study by Desai GG et al. [55] (82.5%, 17.5%), respectively.

Average FBS comparison

In the current research, average FBS of diabetic was 85.72 ± 6.52 and non-diabetic was 95.81 ± 6.23, that showed higher FBS among GDM than Non GDM, similar to the studies by Valadan M et al. [47] (92.01 ± 7.79, 82.61 ± 6.46), Parsaei M et al. [46] (94.1 ± 17.3, 85.2 ± 9.1), and Eidgahi ES et al.[68] (92.04 ± 12.01, 83.19 ± 11.75).

Table 45: Average FBS comparison

Research name	Average FBS	
	Diabetic	Non diabetic
Valadan M et al. [47]	92.01 ± 7.79	82.61 ± 6.46
Parsaei M et al. [46]	94.1 ± 17.3	85.2 ± 9.1
Eidgahi ES et al.[68]	92.04 ± 12.01	83.19 ± 11.75
Current research	85.72 ± 6.52	95.81 ± 6.23

The mean FBS difference was significant in the current research, similar to the studies by Valadan M et al., [47] Tong JN et al., [62] Parsaei M et al., [46] and Eidgahi ES et al.[68]

Average 1st trimester HbA1C comparison

In the current research, average HbA1C of diabetic was 5.64 ± 0.32, and non-diabetic was 4.99 ± 0.46that means higher HbA1C was reported among GMD than Non GDM, which was in accordance with the studies by Tripathy S, and Mohapatra S[48] (5.62 ± 0.09, 5.18 ± 0.04), Balaji V et al. [60] (5.96 ± 0.63, 5.36 ± 0.36), Rajput R et al.[63] (5.73 ± 0.34, 5.34 ± 0.35), and Valadan M et al. [47] (5.45 ± 0.39, 4.96 ± 0.30).

Table 46: Average 1st trimester HbA1C comparison

Research	Average 1st trimester HbA1C	
	Diabetic	**Non diabetic**
Valadan M et al. [47]	5.45 ± 0.39	4.96 ± 0.30
Rajput R et al.[63]	5.73 ± 0.34	5.34 ± 0.35
Balaji V et al. [60]	5.96 ± 0.63	5.36 ± 0.36
Tripathy S, and Mohapatra S[48]	5.62 ± 0.09	5.18 ± 0.04
Current research	5.64 ± 0.32	4.99 ± 0.46

The mean HbA1C difference was significant in the current research, similar to the studies by Valadan M et al., [47] Tripathy S, and Mohapatra S, [48] and Balaji V et al. [60]

Mode of delivery

Of the total cases, FTND was seen in 23.6% of cases, FTVD was seen in 40% of cases, PTVD was seen in 2.4% of cases and LSCS was seen in 35% of cases, i.e., vaginal delivery was seen in 65% of cases, and LSCS was seen in 35% of cases.

In the study by Rupala T et al., [67] 3.8% of cases had FTND, 41.5% of cases had pre term LSCS, and 54.7% of cases had term LSCS. In the Tong JN et al. study, [62] caesarean section was observed among 42.16% of cases, and 57.84% cases had normal vaginal delivery.

Table 47: Mode of delivery

Research name	NVD, LSCS
Tong JN et al. [62]	42.16%, 57.84%
Rupala T et al.[67]	3.8%, 96.2%
Current research	65%, 35%

Maturity

Regarding maturity, 2.4% of cases had pre term, and 97.6% of cases were term, contrast to the study by Rupala T et al. [67] (41.5%, 58.5%), Fonseca L et al. [49] (7.8%, 92.2%), and Singh A et al. [64] (8.3%, 91.7%), respectively.

Table 48: Maturity

Research name	Pre term, Term
Singh A et al. [64]	8.3%, 91.7%
Rupala T et al. [67]	41.5%, 58.5%
Fonseca L et al. [49]	7.8%, 92.2%
Current research	2.4%, 97.6%

Birth weight

Of the total cases, 3.3% of cases had a birth weight of <2.5 kgs, and 96.7% of cases had a birth weight of ≥2.5 kgs.

The mean birth weight in the current research was 3.01± 0.29 kgs, similar to the studies by Rupala T et al. [67] (3.07± 0.49), Fonseca L et al. [49] (3.12 ± 0.49), and Tong JN et al. [62] (3.28 ± 4.4).

Table 49: Average birth weight

Research name	Average birth weight
Tong JN et al. [62]	3.28 ± 4.4
Rupala T et al. [67]	3.07± 0.49
Fonseca L et al. [49]	3.12 ± 0.49
Current research	3.01± 0.29

Average birth weight comparison between diabetic and non-diabetic

In the current research, mean birth weight of GDM was 3.39 ± 0.11 and non GDM was 2.99 ± 0.28, that showed higher mean birth weight among new born of GDM than non-GDM, which was in concordance with the studies by Valadan M et al. [47] (3.52 ± 3.01, 3.31 ± 1.92), Tong JN et al. [62] (3.3 ± 4.34, 3.27 ± 4.71), and Mane L et al.[69] (3.2 ± 7.07, 3.22 ± 6.67).

Table 50: Average birth weight comparison between diabetic and non-diabetic

Research name	Average birth weight	
	Diabetic	Non diabetic
Valadan M et al. [47]	5.45 ± 0.39	4.96 ± 0.30
Rajput R et al.[63]	5.73 ± 0.34	5.34 ± 0.35
Balaji V et al. [60]	5.96 ± 0.63	5.36 ± 0.36
Tripathy S, and Mohapatra S[48]	5.62 ± 0.09	5.18 ± 0.04
Current research	3.39 ± 0.11	2.99 ± 0.28

The mean birth weight difference was significant in the current research, similar to the studies by Valadan M et al., [47] Tong JN et al., [62] and Mane L et al.[69]

NICU admission

In this study, 28.5% of cases needed NICU admission, which was similar to the studies by Rupala T et al. [67] (22.2%), and Tong JN et al. [62] (10.49%).

Table 51: NICU admission

Research name	NICU admission
Tong JN et al. [62]	10.49%
Rupala T et al. [67]	22.2%
Current research	28.5%

Cut off level of 1st trimester HbA1C

In the current research, cut off level of 1st trimester HbA1C to diagnose as diabetic was 5.95, similar to the studies by Sujithra D et al. [56] (≥5.7), Shashikala et al. [70] (5.6), Huges RC et al. [71] (5.9), and Renz PB et al.[72] (5.8).

Table 52: Cut off level of 1st trimester HbA1C

Research	Cut off level of 1st trimester HbA1C
Sujithra D et al. [56]	≥5.7
Shashikala et al. [70]	5.6
Huges RC et al. [71]	5.9
Renz PB et al. [72]	5.8
Current research	5.95

Sensitivity and specificity of 1st trimester HbA1C

In the current research, 1st trimester HbA1C had a sensitivity of 94.1%, and specificity of 86.5%, similar to the studies by Shrivastava N et al. [59] (98.6%, 84.9%), Sujithra D et al. [56] (70.4%, 93.2%), Shashikala et al. [70] (55.7%, 83.6%), and Poo ZX et al.[73] (82%, 72%), respectively.

Table 53: Sensitivity and specificity of 1st trimester HbA1C

Research	Sensitivity	Specificity
Shrivastava N et al. [59]	98.6%	84.9%
Sujithra D et al. [56]	70.4%	93.2%
Shashikala et al. [70]	55.7%	83.6%
Poo ZX et al.[73]	82%	72%
Current research	94.1%	86.5%

PPV and NPV of 1st trimester HbA1C

In the current research, 1st trimester HbA1C had a PPV of 98.1% and NPV of 85.2%, similar to the studies by Sujithra D et al. [56] (79.2%, 89.5%), Shashikala et al. [70] (41.5%, 83.7%), Arbib N et al.[74] (53%, 90.8%), and Wu K et al.[75] (83.3%, 85%), respectively.

Table 54: PPV and NPV of 1st trimester HbA1C

Research	PPV	NPV
Sujithra D et al. [56]	79.2%	89.5%
Shashikala et al. [70]	41.5%	83.7%
Arbib N et al. [74]	53%	90.8%
Wu K et al. [75]	83.3%	85%
Current research	98.1%	85.2%

CONCLUSION

This study highlights the potential of HbA1c as a reliable and practical early screening tool for GDM. HbA1c demonstrated higher sensitivity, specificity, positive predictive value, and negative predictive value, making it a more feasible option for early GDM detection, especially in resource-limited settings. Its non-fasting requirement and single sample collection make it a convenient alternative for both antenatal care providers and patients. Early identification of borderline GDM cases using HbA1c in the first trimester offers the advantage of timely intervention, potentially reducing adverse maternal and neonatal outcomes. Further studies with larger sample sizes across multiple centres are needed to confirm these findings and establish HbA1c as a standard screening tool for GDM.

Strength of the study

In the current study, first time HbA1C was done in the 1st trimester, which helped to identify the at-risk cases of GDM. These cases were counselled and advised regarding dietary as well as life style modification, which helped to reduce the complications in the latter half of pregnancy. This was the major strength of the study.

Limitations of the study

Single-centre study was one limitation of the study. Among anaemic, as glucose uptake by RBC reduces, correct measurement of HbA1C cannot be done.

SUMMARY

The present observational study was done among 123 Antenatal women who attended Obstetrics and Gynecology OPD in S.H.R.I. B.M. PATIL Medical College Hospital and Research Centre, Vijayapura, to determine the efficacy of HbA1c as a predictor for early diagnosis of gestational diabetes mellitus.

The salient features of the current research are mentioned below.

- The current research has diagnosed 13.8% of cases as diabetic.
- Of the cases, the majority (57.7%) were 21-25 years, followed by 26-30 years (26.8%).
- Of the total cases, the majority (62.6%) were multi-paras, and 37.4% were primis.
- Of the total cases, overweight was seen in 15.4% of cases, and obesity was seen in 43.1% of cases.
- Vaginal delivery was seen in 65% of cases, and LSCS was seen in 35% of cases.
- Of the total cases, 2.4% had preterm deliveries.
- Low birth weight (<2.5 kgs) was seen in 3.3% of cases.
- NICU admission was seen in 28.5% of cases.
- 19.6% of non-diabetic cases and 87.5% of diabetic cases needed NICU admission.
- In the current research, the mean age of GDM and non-GDM was similar.
- The mean BMI, FBS, HbA1C, and birth weight were higher in GDM than in non-GDM mothers.

- Regarding diagnosing GDM, HbA1C had a sensitivity of 94.1%, specificity of 86.5%, PPV of 98.1% and NPV of 85.2%, with 5.65 as a cut-off level of HbA1C to diagnose as diabetic.
- Regarding diagnosing GDM, FBS had a sensitivity of 88.2%, specificity of 50.9%, PPV of 96%, and NPV of 59.1%, with 87.5 as a cut-off level of FBS to diagnose as diabetic.

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ANNEXURE I

CONSENT FORM

B.L.D.E. (DEEMED TO BE UNIVERSITY)

SHRI. B. M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTER, VIJAYAPURA-586103

INFORMED CONSENT FOR PARTICIPATION IN

DISSERTATION/RESEARCH

I, the undersigned, , D/O or W/O , aged years, ordinarily resident of do hereby state/declare that Dr. PALETI LEELA LAVANYA of Shri. B. M. Patil Medical College Hospital and Research Centre has examined me thoroughly on at(place) and it has been explained to me in my own language that I am suffering from disease (condition) and this disease/condition mimic the following diseases.

Dr. PALETI LEELA LAVANYA informed me that she is conducting dissertation/research titled "**EARLY DIAGNOSIS OF GESTATIONAL DIABETES MELLITUS BY HbA1C AS A PREDICTOR - PROSPECTIVE OBSERVATIONAL STUDY**", under the guidance of DR. SHOBHA SHIRAGUR requesting my participation in the study. Apart from routine treatment procedures, the preoperative, operative, postoperative, and follow-up observations will be utilized for the study as reference data. The doctor has also informed me that during the conduct of this procedure adverse results may be encountered. Among the above complications, most of them are treatable but are not anticipated hence there is a chance of aggravation of my condition and in rare circumstances, it may prove fatal despite the anticipated diagnosis and best treatment made available. Further Doctor has informed me that my participation in this study would help in the evaluation of the results of the study which is a useful reference to the treatment of other similar

cases in the near future, and I may be benefited in getting relieved of suffering or cure of the disease I am suffering.

The Doctor has also informed me that information given by me, observations made photographs and video graphs are taken upon me by the investigator will be kept secret and not assessed by a person other than me or my legal hirer except for academic purposes.

The Doctor did inform me that though my participation is purely voluntary, based on the information given by me, I can ask for any clarification during treatment/ study related to diagnosis, the procedure of treatment, result of treatment, or prognosis. At the same time, I have been informed that I can withdraw from my participation in this study at any time if I want or the investigator can terminate me from the study at any time from the study but not the procedure of treatment and follow-up unless I request to be discharged.

After understanding the nature of the dissertation or research, diagnosis made, and mode of treatment, I the undersigned Smt. ---------- ----- under my full conscious state of mind agree to participate in the said research/dissertation.

Signature of the patient:

Signature of the doctor:

Date:

Place

B.L.D.E (DEEMED TO BE UNIVERSITY) ಶ್ರೀ ಬಿ.ಎಂ.ಪಟ್ಟೀಲ್ ಮೆಡಿಕಲ್ ಕಾಲೇಜು, ಆಸ್ಪತ್ರೆ ಮತ್ತು ಸಂಶೋಧನಾ ಕೇಂದ್ರ, ವಿಜಯಪುರ-**586103**

ಪ್ರಬಂಧ/ಸಂಶೋಧನೆಯಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ಮಾಹಿತಿ ಪಡೆದ ಸಮ್ಮತಿ

ನಾನು, ಕೆಳಗಿನವರು_____ ಸಹಿಯಿಟ್ಟವರು, ಮಗ/ಮಗಳು/ಪತ್ನಿಯ _____ ವಯಸ್ಸು _____ವರ್ಷಗಳು, ಸಾಮಾನ್ಯವಾಗಿ ನಿವಾಸಿಸುವ ಸ್ಥಳದ ಹೆಸರು_____, ಇಲ್ಲಿ ಹೇಳಿದ್ದೇನೆ/ಘೋಷಿಸುತ್ತೇನೆ ಡಾಕ್ಟರ್ ಹೆಸರು_____ ಅವರು ಆಸ್ಪತ್ರೆ ಹೆಸರು_____ ಅವರು ನನ್ನನ್ನು ಪೂರ್ಣವಾಗಿ ಪರೀಕ್ಷಿಸಿದರು ದಿನಾಂಕದಲ್ಲಿ_____ ಸ್ಥಳ ಹೆಸರು_____ ಮತ್ತು ನನಗೆ ನನ್ನ ಭಾಷೆಯಲ್ಲಿ ವಿವರಿಸಲಾಗಿದೆ ನಾನು ಒಂದು ರೋಗ (ಸ್ಥಿತಿ) ಅನುಭವಿಸುತ್ತಿದ್ದೇನೆ. ಮುಂದುವರಿದು ಡಾಕ್ಟರ್ ನನಗೆ ತಿಳಿಸಿದ್ದಾರೆ ಅವರು ಒಂದು ಪದ್ಧತಿ/ಸಂಶೋಧನೆ ನಡೆಸುತ್ತಿದ್ದಾರೆ ಶೀರ್ಷಿಕೆಯುಳ್ಳ_____ ಡಾಕ್ಟರ್_____ ಮಾರ್ಗದರ್ಶನದಲ್ಲಿ ನನ್ನ ಪಾಲ್ಗೊಳ್ಳುವಿಕೆಯನ್ನು ಕೇಳಿದ್ದಾರೆ ಅಧ್ಯಯನದಲ್ಲಿ.

ಡಾಕ್ಟರ್ ನನಗೆ ಇದನ್ನು ಕೂಡಾ ತಿಳಿಸಿದ್ದಾರೆ ಈ ಕ್ರಮದ ನಡೆವಲ್ಲಿ ಪ್ರತಿಕೂಲ ಫಲಿತಾಂಶಗಳನ್ನು ಎದುರಿಸಬಹುದು. ಮೇಲೆ ಹೇಳಿದ ಪ್ರಕಟಣೆಗಳಲ್ಲಿ, ಅಧಿಕಾಂಶವು ಚಿಕಿತ್ಸಿಸಬಹುದಾದರೂ ಅದನ್ನು ನಿರೀಕ್ಷಿಸಲಾಗುತ್ತಿಲ್ಲ ಆದ್ದರಿಂದ ನನ್ನ ಸ್ಥಿತಿಯ ಹಿರಿದಾಗುವ ಅವಕಾಶವಿದೆ ಮತ್ತು ಅಪರೂಪದ ಸಂದರ್ಭಗಳಲ್ಲಿ ಅದು ಮರಣಕಾರಕವಾಗಿ ಪರಿಣಮಿಸಬಹುದು ಹೊಂದಿದ ರೋಗನಿರ್ಧಾರ ಮತ್ತು ಯಥಾಶಕ್ತಿ ಚಿಕಿತ್ಸೆ ಮಾಡಲು ಹೊಂದಿದರೂ. ಮುಂದುವರಿದು ಡಾಕ್ಟರ್ ನನಗೆ ತಿಳಿಸಿದ್ದಾರೆ ನನ್ನ ಪಾಲ್ಗೊಳ್ಳುವಿಕೆ ಈ ಅಧ್ಯಯನದ ಫಲಿತಾಂಶಗಳ ಮೌಲ್ಯಮಾಪನದಲ್ಲಿ ಸಹಾಯಕವಾಗುತತ್ತದೆ ಇತರ ಸಮಾನ ಪ್ರಕರಣಗಳ ಚಿಕಿತ್ಸೆಗೆ ಉಪಯುಕ್ತ ಉಲ್ಲೇಖವಾಗಿದೆ, ಮತ್ತು ನಾನು ಅನುಭವಿಸುವ ರೋಗದಿಂದ ವಿಮುಕ್ತಿ ಅಥವಾ ಗುಣಮುಖಗೊಳ್ಳುವಲ್ಲಿ ನನಗೆ ಪ್ರಯೋಜನವಾಗಬಹುದು.

ಡಾಕ್ಟರ್ ನನಗೆ ಇದನ್ನು ಕೂಡಾ ತಿಳಿಸಿದ್ದಾರೆ ನನ್ನಿಂದ ನೀಡಿದ ಮಾಹಿತಿ, ಮಾಡಿದ ಪರಿಶೀಲನೆಗಳು / ಫೋಟೋಗ್ರಾಫ್‌ಗಳು / ವೀಡಿಯೋ ಗ್ರಾಫ್‌ಗಳು ನನ್ನ ಮೇಲೆ ತೆಗೆದುಕೊಳ್ಳಲಾಗುವ ಅನ್ವೇಷಕರು ರಹಸ್ಯವಾಗಿ ಇಡುವರು ಮತ್ತು ನಾನು ಅಥವಾ ನನಗೆ ಕಾನೂನು ದೃಷ್ಟಿಯಲ್ಲಿ ಸಂಬಂಧಿತrannu ಹೊರತುಪಡಿಸಿ ಇತರ ವ್ಯಕ್ತಿಯಿಂದ ಮೌಲ್ಯಮಾಪನ ಮಾಡಲಾಗುವುದಿಲ್ಲ. ಡಾಕ್ಟರ್ ನನಗೆ ತಿಳಿಸಿದ್ದಾರೆ ನನ್ನ ಪಾಲ್ಗೊಳ್ಳುವಿಕೆ ಶುದ್ಧವಾಗಿ ಸ್ವೇಚ್ಛಾಯಿತ, ನನ್ನಿಂದ ನೀಡಿದ ಮಾಹಿತಿಯ ಆಧಾರದ ಮೇಲೆ, ಚಿಕಿತ್ಸೆ / ಅಧ್ಯಯನದ ಸಂಬಂಧದಲ್ಲಿ ರೋಗನಿರ್ಧಾರ, ಚಿಕಿತ್ಸೆಯ ವಿಧಾನ, ಚಿಕಿತ್ಸೆಯ ಫಲಿತಾಂಶ ಅಥವ ಆ ಭವಿಷ್ಯದ ಪ್ರವೃತ್ತಿಗಳು ಬಗ್ಗೆ ಯಾವುದೇ ಸ್ಪಷ್ಟತೆ ಕೇಳಬಹುದು. ಅದೇ ಸಮಯದಲ್ಲಿ ನನಗೆ ತಿಳಿಸಲಾಗಿದೆ ನಾನು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನನ್ನ ಪಾಲ್ಗೊಳ್ಳುವಿಕೆಯನ್ನು ನಿಲ್ಲಿಸಬಹುದು ನಾನು ಬಯಸಿದರೆ ಅಥವಾ ಅನ್ವೇಷಕರು ಅಧ್ಯಯನದಿಂದ ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ನನ್ನನ್ನು ನಿಲ್ಲಿಸಬಹುದು.

ಪ್ರಬಂಧ ಅಥವಾ ಸಂಶೋಧನೆಯ ಸ್ವಭಾವ, ಮಾಡಿದ ರೋಗನಿರ್ಧಾರ ಮತ್ತು ಚಿಕಿತ್ಸೆಯ ವಿಧಾನವನ್ನು ಅರ್ಥಮಾಡಿಕೊಂಡು, ನಾನು ಕೆಳಗಿನ ಶ್ರೀ / ಶ್ರೀಮತಿ_____ ನನ್ನ ಪೂರ್ಣವಾದ ಪ್ರಜ್ಞೆಯ ಸ್ಥಿತಿಯಲ್ಲಿ ಹೇಳಿದ ಸಂಶೋಧನೆ / ಪ್ರಬಂಧದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ಒಪ್ಪುತ್ತೇನೆ.

ರೋಗಿಯ ಸಹಿ

ಡಾಕ್ಟರನ ಸಹಿ

ಸಾಕ್ಷಿಗಳು

1)

2)

ANNEXURE II

PROFORMA

A Prospective Clinical Study at a Tertiary Care Hospital

TITLE: EARLY DIAGNOSIS OF GESTATIONAL DIABETES MELLITUS BY HbA1C AS A PREDICTOR - PROSPECTIVE OBSERVATIONAL STUDY

NAME	
AGE/SEX	
ADMISSION NUMBER (O.P. NO)	
DATE OF ADMISSION	
DATE OF DISCHAGE	
ADDRESS AND PHONE NUMBER	

CHIEF COMPLAINTS:

HISTORY OF PRESENT ILLNESS:

HISTORY OF PRESENT PREGNANCY:

A.N.C.:

1ST TRIMESTER:

2NDTRIMESTER:

3RD TRIMESTER:

MARITAL HISTORY:

OBSTETRIC HISTORY: G: P: L: A: D:

L.M.P.:

E.D.D.:

P.O.G.:

TREATMENT HISTORY:

DURATION:

PERSONAL HISTORY:

GENERAL PHYSICAL EXAMINATION:

HEIGHT

WEIGHT

BMI

PULSE:

BLOOD PRESSURE:

RESPIRATORY RATE:

TEMPERATURE:

HEAD-TO-TOE EXAMINATION:

PALLOR

ICTERUS:

CYANOSIS:

CLUBBING:

LYMPHADENOPATHY:

OEDEMA:

THYROID:

BREAST:

SPINE:

CARDIOVASCULAR SYSTEM:

RESPIRATORY SYSTEM:

PER ABDOMEN:

INVESTIGATIONS:

GESTATIONAL AGE	FBS	HbA1C	2HR OGTT

CBC

URINE ROUTINE

BLOOD GROUP AND RH TYPING THYROID PROFILE

HIV

HBsAg

OBSTETRIC SCAN DELIVERY DATE

MATERNAL OUT COME

RISK OF PREECLAMPSIA

GDM IN FUTURE PREGNANCY

TYPE 2 DIABETES MILLETUS

POSTPARTUM FACTORS

RISK OF CESAREAN SECTION

CARDIOVASCULAR RISK IN POSTPARTUM

PERINATAL OUTCOME

FETAL MACROSOMIA

HYPOGLYCIMEIA

SHOULDER DYSTOCIA

RESPIRATORY DISTRESS

NICU ADMISSION

TYPE 2 DM LATER IN LIFE

FETAL DEATH

ANNEXURE III

ETHICAL CLEARENCE

BLDE
(DEEMED TO BE UNIVERSITY)
Declared as Deemed to be University u/s 3 of UGC Act, 1956
Accredited with 'A' Grade by NAAC (Cycle-2)
The Constituent College
SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPURA

BLDE (DU)/IEC/ 878/2022-23 10/4/2023

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this University met on **Saturday, 18th March, 2023 at 11.30 a.m. in the CAL Laboratory, Dept. of Pharmacology,** scrutinizes the Synopsis/ Research Projects of Post Graduate Student / Under Graduate Student /Faculty members of this University /Ph.D. Student College from ethical clearance point of view. After scrutiny, the following original/ corrected and revised version synopsis of the thesis/ research projects has been accorded ethical clearance.

TITLE: "EARLY DIAGNOSIS OF GESTATIONAL DIABETES MELLITUS BY HBA1C AS A PREDICTOR-PROSPECTIVE OBSERVATIONAL STUDY".

NAME OF THE STUDENT/PRINCIPAL INVESTIGATOR: DR.PALETI LEELA LAVANYA

NAME OF THE GUIDE: DR.SHOBHA SHIRAGUR, PROFESSOR, DEPT. OF OBSTETRICS AND GYNAECOLOGY.

Dr. Santoshkumar Jeevangi
Chairperson
IEC, BLDE (DU),
VIJAYAPURA

Chairman,
Institutional Ethical Committee,
BLDE (Deemed to be University)
Vijayapura

Dr.Akram A. Naikwadi
Member Secretary
IEC, BLDE (DU),
VIJAYAPURA

MEMBER SECRETARY
Institutional Ethics Committee
BLDE (Deemed to be University)
Vijayapura-586103. Karnataka

Following documents were placed before Ethical Committee for Scrutinization.

- Copy of Synopsis/Research Projects
- Copy of inform consent form
- Any other relevant document

Smt. Bangaramma Sajjan Campus, B. M. Patil Road (Sholapur Road), Vijayapura - 586103, Karnataka, India
BLDE (DU): Phone: +918352-262770, Fax: +918352-263303, Website: www.bldedu.ac.in, E-mail:office@bldedu.ac.in
College: Phone: +918352-262770, Fax: +918352-263019, E-mail: bmpmc.principal@bldedu.ac.in

ANNEXURE IV

MASTER CHART

Sl.no	ipno	NAME	AGE	OBSHISTO	POG	HT	WT	BMI	FBS	1T HBA1C	2ND OGTT	2ND Hba1c	MODE OF DELIVERY	term/ pre	Bwt kg	NICU	3T OGTT
1	449661	AISHWARYA28	22	G2P1L1	8wks 0 day	150	48	21.3	89	5.2	160	6.6	LSCS	TERM	3.5	no	174
2	190673	paraven	30	G4P2L2A1 9 WKS 1D	11wks 5 d	158	68	27.2	109	5.6	110	5.4	FTVD	TERM	3	no	
3	207859	LAXMI	23	G3P2L2	11wks 4d	154	56	23.6	76	4.8	120	5.2	FTVD	TERM	2.9	no	
4	390476	satyamma	19	G2P1L1	10wks6d	154	48	21.3	78	4.7	78	5.1	FTVD	TERM	2.9	no	
5	390486	kaveri	23	PRIMI	9wks 0d	160	62	24.2	88	4.8	110	5.1	LSCS	TERM	3.2	no	
6	248921	swapna	24	G2P1L1	6wks6d	158	68	27.2	79	5.1	120	5.2	FT VD	TERM	2.8	no	
7	212445	pooja	27	G5P4L4	10wks6d	150	48	21.3	90	5.1	95	4.1	FTVD	TERM	3.1	no	
8	215168	sudharani	24	G4p3L3	9WKS6D	152	65	28.1	82	4.7	124	5.4	FTVD	TERM	3.5	YES	
9	292989	SHRUTI	28	G2P1L1	11WKS2D	154	56	23.6	84	5.1	110	5.2	FTVD	TERM	2.9	no	
10	272258	PARVATI	22	G2A1	9WKS4D	148	60	27.4	87	5.6	156	6.2	LSCS	TERM	3.4	YES	168
11	206313	MADAKINI	28	PRIMI	10WKS2D	150	48	21.3	79	5.2	106	5.4	FTVD	TERM	2.8	no	
12	212445	BHARTI	27	G5P4L4	10WKS0D	154	56	23.6	90	5.1	95	5.1	FTVD	TERM	3.2	no	
13	55953	KAVERI	26	PRIMI	7WKS3D	154	48	21.3	89	5.1	78	4.8	FTVD	TERM	2.8	no	
14	200584	LAXMI	22	G3P2L2	10WKS0D	150	48	21.3	80	4	90	4.3	LSCS	TERM	3.2	no	
15	85290	USHA	21	G3P2L2	11WKS6D	165	68	25	78	4.6	94	4.9	FTVD	TERM	3.1	no	
16	206513	NAKSHATRA	25	PRIMI	11WKS5D	154	56	23.6	91	4.2	120	4.8	LSCS	TERM	3.3	no	
17	209231	SASHIKALA	26	G2P1L1	11WKS2D	156	60	24.7	90	4.2	79	4.7	FTND	TERM	3.2	no	

18	213314	PRABHAVTI 29	29	G2P1L1	9WKS6D	150	48	21.3	74	4.2	110	4.9	LSCS	TERM	3.2	no	
19	209321	LAXMI	22	PRIMI	9WKS0D	154	48	21.3	88	4.8	110	5.1	FTVD	TERM	2.5	no	
20	151783	SALMA	30	G2P1L1	9WKS4D	154	56	23.6	87	5.1	124	5.6	LSCS	TERM	3.4	YES	
21	212448	AKSHTA	24	G3P1L1A1	8WKS2D	152	65	28.1	79	5.9	124	5.9	LSCS	TERM	3.4	YES	
22	212449	AISHWARYA	21	PRIMI	11WKS3D	152	68	29.4	94	4.9	110	5.2	LSCS	TERM	3.2	no	
23	2322215	LAXMI	23	PRIMI	11WKS 3D	154	56	23.6	78	5.6	90	5.2	FTVD	TERM	2.9	no	
24	220618	TANUJA	27	PRIMI	9WKS0D	150	48	21.3	78	5.6	110	5.8	LSCS	TERM	3.4	YES	
25	200583	LAXMI A	22	G2P1L1	8WKS0D	158	68	27.2	99	4.9	157	6.1	LSCS	TERM	3.4	YES	164
26	200585	LAXMI K	22	G3P2L2	10WKS0D	154	48	21.3	80	4.3	90	4.3	FTVD	TERM	2.8	no	
27	212915	POOJA K	24	PRIMI	10WK6D	150	48	1.3	79	5.2	98	5.4	FTVD	TERM	2.7	no	
28	390161	SHEWTA	28	G2P1L1	9WKS6D	150	48	21.3	88	4.8	102	5.2	FTVD	TERM	3	no	
29	217304	POOJA B	24	G3P1L1A1	10WKS2D	154	56	23.6	78	5.1	108	5.6	LSCS	TERM	3.3	YES	
30	220617	SHAILA	35	G3P2L2	9WKS6D	150	48	21.3	82	4.9	78	5.1	FTVD	TERM	2.9	no	
31	118964	IRAMMA	24	G2P1L1	10WKS2D	152	65	28.1	94	4.1	96	4.1	LSCS	TERM	3.1	no	
32	151457	SAHERA	23	G3P1L1A1	11WKS3D	156	60	24.7	90	4.9	100	5.4	FTVD	TERM	2.8	no	
33	231472	SAVITRI	22	PRIMI	11WKS2D	148	50	22.8	88	4.5	74	4.8	FTVD	TERM	2.6	no	
34	232214	POOJA	20	PRIMI	10WKS0D	152	65	28.1	94	5.4	111	5.8	LSCS	TERM	3.2	no	
35	86735	LAXMI	29	G4P1L1A2	11WKS2D	150	48	21.3	78	4.6	110	4.8	LSCS	TERM	2.9	no	
36	224016	RESHMA	28	G3P2L2	11WKS4D	152	68	29.4	98	5.5	150	6.4	LSCS	TERM	3.45	YES	172
37	16645	SUSHMA	26	PRIMI	11WKS4D	158	68	27.2	82	4.6	100	5.1	LSCS	TERM	3.4	YES	
38	234837	POOJA	24	PRIMI	10WKS0D	148	60	27.4	90	4.9	93	5.2	FTVD	TERM	2.8	no	
39	410801	NEELA	26	G3A2	10WKS2D	150	48	21.3	86	4.8	98	5.1	FTVD	TERM	2.9	no	
40	231458	AMBIKA	20	G2A1	9WKS5D	154	56	23.6	90	5.1	94	4.8	FTVD	TERM	3	no	
41	240182	JYOTI	26	G2P1L1	10WKS3D	165	68	25	89	4.8	96	5.2	FTVD	TERM	2.6	no	
42	236198	ANITA	33	G6P2L2A4	11WKS0D	154	56	23.6	82	4.2	96	4.6	FTVD	TERM	3.2	no	
43	254242	PAPI RATHOD	26	G4P3L3	9WKS6D	152	65	28.1	80	4.2	88	4.1	FTVD	TERM	2.8	no	
44	194105	ASHA	25	G3A2	10WKS5D	150	48	21.3	78	4.1	81	4.8	FTVD	TERM	2.7	no	

45	257336	POOJA	22	PRIMI	10WKS0D	152	68	29.4	78	4.1	87	4.1	LSCS	TERM	2.9	no	
46	225576	SUMA	23	G2P1L1	10WKS6D	150	48	21.3	90	5.1	89	5.2	LSCS	TERM	3.1	no	
47	252967	PRIYANKA	29	G3P2L2	9WKS4 D	150	59	26.2	88	4.8	104	5	FTVD	TERM	2.8	no	
48	259131	LAXMI	26	G3A2	9wks 0d	150	48	21.3	65	4.2	87	5.1	FTVD	TERM	3.1	no	
49	267267	GEETA	29	G2P1L1	10WKS2D	158	68	27.2	73	4.1	92	4.6	LSCS	TERM	3.2	no	
50	201453	PARVATI	21	PRIMI	11WKSOD	148	50	22.8	88	5	92	5.2	PTVD	PRE TERM	2.3	YES	
51	250902	YASHODA	26	G2A1	9WKS6D	150	48	21.3	98	5.9	160	6.2	LSCS	TERM	3.5	YES	175
52	250903	SAVITA	20	PRIMI	9WK4D	152	65	28.1	76	4.1	64	4.4	FTVD	TERM	3.1	no	
53	270640	sweta	34	G3P2L2	10WKS2D	156	72	29.6	92	5.2	97	5.4	FTVD	TERM	3.3	YES	
54	163427	PRIYANKA	22	PRIMI	11WKSO D	148	60	27.4	90	5.1	110	5.3	LSCS	TERM	3.4	YES	
55	19704	SHIVALEELA	32	G4P2L1D1A 1 9WKS5D	10WKS4D	154	56	23.6	80	5	106	5.2	PTVD	PRE TERM	2.2	YES	
56	184034	PRIYANKA	23	primi	10WK6D	152	68	29.4	92	5	98	4.7	FTVD	TERM	2.7	no	
57	259131	LAXMI	24	PRIMI	9WKS6D	152	65	28.1	80	4.8	87	5.1	LSCS	TERM	3.1	no	
58	297944	GOURAMMA	26	G2A1	10WK4D	150	48	21.3	88	4.8	110	5.6	LSCS	TERM	2.9	no	
59	299236	MEGHA	22	G4A3	9WKW5D	148	50	22.8	94	5.2	147	6.1	LSCS	TERM	3.4	YES	178
60	198569	JYOTHI	28	G3P2L2	10WK2D	158	68	27.2	86	4.2	104	4.7	FTVD	TERM	3.1	no	
61	236198	ANITA	32	G6P2L2A3	9WK6D	154	56	21.3	78	4.1	61	4.6	FTVD	TERM	2.9	no	
62	208974	bhagyashree	28	PRIMI	10WK5D	150	48	21.3	108	5.8	160	7.6	LSCS	TERM	3.3	YES	180
63	210279	TASLEEM	25	G3P1L1A1	9WK2D	148	60	27.4	94	5.4	110	5.4	LSCS	TERM	3.2	YES	
64	19537	SUMA	29	PRIMI	9WKS5D	150	48	21.3	82	5.1	104	5.6	FTVD	TERM	2.8	no	
65	20213	REHAN	25	PRIMI	10WK2D	148	62	20.1	88	4.9	98	5.4	FTVD	TERM	2.9	no	
66	20916	RESHMA	21	PRIMI	10WK3D	152	65	28.1	92	5.3	104	5.8	FTVD	TERM	3	no	
67	244953	REKHA	25	PRIMI	9WK3D	150	48	21.3	94	5.1	117	5.9	FTVD	TERM	2.9	no	
68	259029	GOUSIYA	27	G2P1L1	8WKS6D	156	72	29.6	98	5.9	144	6.4	LSCS	TERM	3.4	YES	164
69	259429	NETRA	25	primi	9WKS6D	152	68	29.4	94	5.9	119	5.9	FTVD	TERM	3.2	no	
70	259847	MUSKAN	19	PRIMI	1OWK2D	150	48	21.3	84	4.9	103	5.1	FTND	TERM	2.8	no	
71	259895	NAJMA	24	G2P1L1	10WK4D	152	65	28.1	88	5.1	102	5.4	FTND	TERM	3	no	

72	262112	KAVYA	23	PRIMI	10WK4D	152	65	28.1	92	5.1	109	5.4	FTND	TERM	3	no	
73	276541	AISHWARYA	25	PRIMI	9WKS7D	158	58	27.2	90	5.2	120	5.9	FTND	TERM	3.1	no	
74	278565	POOJA	23	PRIMI	10WK2D	150	48	21.3	84	5.1	95	5.3	FTVD	TERM	2.9	YES	
75	278881	SHOBHA	25	G2P1L1	9WK4D	150	88	39.1	86	5.6	148	6.1	LSCS	TERM	3.6	YES	168
76	60268	PRATHIMA	30	G2P1L1	10WK1D	152	68	29.4	99	5.5	127	5.9	LSCS	TERM	3.1	YES	
77	283084	MAHESHWARI	23	PRIMI	9WK3D	148	60	27.4	80	4.8	87	5.1	FTVD	TERM	2.9	no	
78	29813	RENUKA	31	G5P3L3A1	9WK6D	150	48	21.3	94	5.4	142	6	FTVD	TERM	3.25	YES	138
79	304186	PAYAL	22	PRIMI	10WK1D	148	50	22.8	78	4.9	102	5	FTVD	TERM	2.8	YES	
80	306401	BHAGYASREE	20	G2P1L1	9WK5D	150	48	21.3	94	5.9	144	6.2	LSCS	TERM	3.1	YES	142
81	306792	SUNITA	23	PRIMI	9WK3D	156	72	29.6	90	5.2	120	5	FTVD	TERM	3.1	no	
82	315833	AKSHATA	25	PRIMI	9WK4D	150	48	21.3	83	5	102	5.1	FTVD	TERM	3.1	no	
83	279083	PAVITRA	20	PRIMI	10WK3D	152	68	29.4	90	5.1	110	5.1	FTVD	TERM	2.8	no	
84	360611	LAXMI	28	G3P1L1A1	9WK2D	158	58	27.2	92	5.2	104	5.2	FTVD	TERM	3.1	no	
85	245929	SURABHI	21	PRIMI	9WK3D	150	48	21.3	80	5	88	5.1	FTVD	TERM	2.9	no	
86	249078	SHREEDEVI	21	G2A1	8WK6D	148	50	22.8	78	4.9	90	5.1	FTVD	TERM	2.8	no	
87	249083	ALFIYA	22	G3P2L2	9WK1D	148	50	22.8	98	5.1	103	5.2	LSCS	TERM	3.1	no	
88	249086	BAGAMMA	25	G4P2L2A1	9WK2D	148	60	27.4	102	5.8	144	6.1	LSCS	TERM	3.4	YES	158
89	255288	MALAMMA	22	PRIMI	10WK1D	154	56	23.6	88	4.8	92	5.2	LSCS	TERM	2.7	no	
90	166124	SRAVYA	21	PRIMI	9W2D	150	48	21.3	96	5.9	156	6.5	LSCS	TERM	3.4	YES	160
91	107068	KHAIRUNBI	24	G2P1L1	9W3D	154	56	23.6	89	5.6	110	5.4	FTND	TERM	2.8	YES	
92	360867	SAROJINI	22	G2P1L1	8W6D	152	68	29.4	92	5.8	106	5.1	FTND	TERM	2.6	no	
93	203771	PRATIMA	26	G3P2L2	10W1D	156	72	29.6	84	4.8	98	5.1	FTND	TERM	2.6	no	
94	203769	SAMATA	25	PRIMI	9W5D	148	50	22.8	78	5.4	102	5.3	FTND	TERM	2.8	no	
95	203767	ALIFA	21	G2P1L1	9W8D	150	48	21.3	86	5	92	5.5	LSCS	TERM	2.9	no	
96	203727	SANVI	25	G2P1L1	8W4D	154	56	23.6	90	5.6	98	5.4	FTND	TERM	2.8	no	
97	203773	PRATIKA	25	G3P2L2	8W3D	150	48	21.3	88	5.1	100	5.2	FTND	TERM	2.6	no	
98	123845	NIRMALA	21	G2A1	10W2D	148	60	27.4	84	4.9	96	5.4	FTND	TERM	2.9	no	

99	338023	BHAGYASREE	24	G2P1L1	10W1D	152	68	29.4	92	5.8	104	5.2	LSCS	TERM	3	YES	
100	196775	PARVEEN	22	PRIMI	11W0D	156	72	29.6	88	5.2	106	5.8	LSCS	TERM	3.1	YES	
101	196776	MUSKAN	28	PRIMI	10W7D	152	68	29.4	94	5.9	148	6.1	LSCS	TERM	3.4	YES	174
102	196504	SAVITA	22	G2A1	10W5D	154	56	23.6	84	5.2	98	5.6	FTND	TERM	2.9	YES	
103	155308	AARTI	28	G3P2L2	9W4D	148	50	22.8	96	5.8	98	5.4	FTND	TERM	2.8	no	
104	155305	SANDYA	24	G3P2L2	9W3D	156	72	29.6	82	4.9	104	5.5	FTND	TERM	3.1	no	
105	348685	HASINA	25	G2P1L1	8W8D	150	48	21.3	88	5.6	110	5.6	FTND	TERM	2.9	no	
106	194801	NAGAMMA	19	G3P2L2	11W0D	152	68	29.4	106	5.9	168	6.7	LSCS	TERM	3.45	no	180
107	194015	ASHA RANI	20	G3P2L2	10W1D	148	50	22.8	76	5.6	108	5.3	FTND	TERM	2.6	no	
108	242447	SAVITRI	21	PRIMI	9W3D	156	72	29.6	84	4.9	98	5.2	FTND	TERM	2.1	no	
109	193566	PARIMALA	22	G2P1L1	9W5D	152	68	29.4	88	4.7	106	5.4	FTND	TERM	2.8	no	
110	193565	PRATIKA	25	G2P1L1	10W3D	150	48	21.3	84	5.1	98	5.3	LSCS	TERM	3.1	no	
111	193564	SANVI	22	G2A1	11W1D	148	50	22.8	76	5.4	96	5.2	FTND	TERM	3.2	no	
112	158666	NIVEDITA	25	G3P2L2	10W8D	152	68	29.4	90	5.3	108	5.8	FTND	TERM	2.9	no	
113	127390	RENUKA	25	G3P2L2	9W9D	148	50	22.8	86	4.9	98	5.6	FTND	TERM	3.1	no	
114	166127	PREETI	28	G2P1L1	9W1D	152	68	29.4	90	5.8	148	6.5	LSCS	TERM	3.35	YES	168
115	123853	SUPRIYA	28	PRIMI	9W7D	156	72	29.6	88	5.4	94	5.2	LSCS	TERM	2.8	YES	
116	32817	POOJA	19	PRIMI	11W1D	148	50	22.8	86	5.6	89	4.9	PTVD	PRE TERM	2.1	YES	
117	49236	VIJAYALAXMI	20	PRIMI	10W2D	152	68	29.4	84	5.1	96	5.2	FTND	TERM	2.9	no	
118	103151	GURUDEVI	21	G2A1	10W6D	156	72	29.6	92	5.8	102	5.4	FTND	TERM	2.8	no	
119	91536	SWATI	20	PRIMI	9W7D	154	56	23.6	88	5.2	94	5.2	FTND	TERM	2.8	no	
120	101487	PALLAVI	24	G2A1	9W4D	148	50	22.8	92	5.1	98	5.5	FTND	TERM	2.9	no	
121	111011	SHILPA	20	G2P1L1	8W3D	154	56	23.6	90	4.9	100	5.8	FTND	TERM	3.1	YES	
122	8189	NAFISHA	21	PRIMI	8W3D	150	48	21.3	88	5.8	106	5.4	FTND	TERM	3.2	no	
123	1110001	SHILPA BASAVARAJ K	20	G2P1L1	8W8D	150	48	21.3	88	5.6	110	5.6	FTND	TERM	2.9	no	

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