# Exploring Fetal and Maternal outcomes in Gestational Diabetes Mellitus: A comprehensive cross-sectional study

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## Abstract

**Background:** Indian females exhibit a significant occurrence of diabetes, with their likelihood of developing gestational diabetes mellitus being 11.3 times higher compared to Caucasian females. Women with GDM are more prone to experiencing complications such as preeclampsia and requiring a cesarean delivery.

**Aim:** This study aims to investigate both antepartum and intrapartum complications in individuals with gestational diabetes mellitus, as well as assess fetal outcomes during pregnancy in these patients.

**Material and methods:** A prospective observational study was done in the department of obstetrics & gynecology, tertiary care hospital. Pregnant women were screened for GDM by GST. 100 women defined as GDM by DIPSI criteria were included in the study. Maternal outcomes and Neonatal outcomes were studied.

**Results:** The peak incidence of gestational diabetes mellitus occurred between ages 26 to 30 (44%), with 70% of the studied population being multigravida. The majority of GDM cases were identified between 34 and 36 weeks of gestation (68%). Mode of delivery was fairly balanced, with 52% through vaginal delivery and 48% through cesarean section. Pre-eclampsia developed in 16% of cases, while 12% experienced preterm labor and 6% had premature rupture of membranes.

**Conclusion:** Gestational diabetes mellitus is linked to a slight elevation in perinatal complications. There is an increased occurrence of neonatal hypoglycemia, respiratory distress syndrome, hyperbilirubinemia, intrauterine death and Neonatal Intensive Care Unit admissions. Despite a notable association with significant macrosomia, the mode of delivery remained unchanged, with vaginal delivery being the most common at 52%.

**Keywords:** Gestational Diabetes Mellitus, Perinatal Complications, Pregnancy Screening, Health Outcomes, Delivery

## Introduction

Carbohydrate intolerance first recognized during pregnancy is known to be gestational diabetes. The prevalence ranges between 11-14% in the world whereas the prevalence of GDM in Indian population was found to be higher, at 16.2%[1]. The reason was higher prevalence amongst pregnant Indian women was attributed to genetics, ethnicity and hereditary components.

Pregnancy is a physiological condition which promotes a hyperglycemic state because of the hormonal changes associated with it. This physiological change can turn pathological leading to the onset and progression of GDM, leading to adverse maternal and perinatal outcomes.

Diabetes in pregnancy has a maze for a history. The different criteria used for the diagnosis of this condition have had a lot of changes over the course of its history. From the time 50-gram glucose was used for a 1-hour screening test in Boston in 1954[2] to the 100g Oral glucose tolerance test. This was developed by O'Sullivan.

From previously being called as meta gestational diabetes mellitus[3], the terminology completely changed to gestational diabetes mellitus after O'Sullivan first debuted the term in 1967 a popular monograph[4] by Jorgen Pederson had the term "Gestational Diabetes". Jorgen also made a note that the glucose intolerance lasted only upto the postpartum period and then normalized.

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Norbert Freinkel and Boyd Metzger brought GDM on the obstetric map and brought out the importance of the condition as an obstetric complication.

GDM during the antenatal period not only has an adverse effect on this pregnancy but also influences subsequent pregnancies. This was first proposed by Peter Damm [5].

**Fetomaternal complications of GDM**

GDM is a condition with increased blood glucose levels in pregnancy characterized, which is a multifactorial and is resultant of underlying proposed mechanisms such as genetic predisposition, insulin resistance, and chronic inflammation.

This condition usually returns to normal after pregnancy, but there is increased risk of progression to type 2 DM eventually. It is also responsible for long-term adverse effects in mother and child [6]

**Maternal complications**

**Hypertensive disorders:**

In GDM, hyperglycemia may damage endothelial cells, which can result in vascular dysfunction associated with hypertension. Because of this, it is having been suggested that GDM increases the incidence of hypertension during pregnancy and the postpartum period.

Both diabetes and hypertension are risk factors for the development of pre-eclampsia, a disorder that affects between 3% and 5% of pregnancies worldwide and is characterized by high blood pressure and proteinuria[6]. Recent ACOG guidelines advocate that proteinuria is not essential for the diagnosis of pre-eclampsia.

**Polyhydramnios:**

Polyhydramnios is osmotic diuresis from hyperglycemic mother to the fetus inside. Significant association was found between amniotic fluid and maternal glucose concentration[7].

Strangenberg et al[8] proposed that fetal insulin production and its renal clearance as seen in amniotic fluid concentrations are biologically more related to pathogenic processes producing diabetic fetopathy.

Complications of polyhydramnios are: Premature rupture of membranes, Preterm labor, Malposition and malpresentation, Umbilical cord prolapse, Abruption placentae, Atonic uterus leading to PPH.

**Caesarean Section and Instrumental Deliveries**

Cesarean section and operative vaginal delivery rates are higher in women with GDM.

C D Naylor et al conducted a study at Toronto tri hospital where they studied the relationship between birth weight and mode of delivery among women with untreated borderline GDM, treated overt GDM, and controls. They observed that compared to normoglycemic controls, the untreated borderline GDM group had increased cesarean deliveries (29.6% vs 20.2%). Cesarean delivery rate was about 33% irrespective of whether macrosomia was present or absent.

Operative vaginal deliveries are more in women with GDM. Significant increase in risk was associated with elevated levels of glycemia (FPG>105mg/dl) and maternal weight.

**Fetal And Neonatal Complications**

**Macrosomia:**

RCOG defines macrosomia as fetal weight as 4500g[9]. There are no precise definitions agreed upon by authorities. ACOG mentions fetal weight more than 4000- 4500g as macrosomia[10].

**Malformation:**

Congenital malformations are more prevalent in pregestational diabetes rather than gestational diabetes mellitus.

The risk of congenital anomalies increases by 3 to 10 times in pre-existing diabetics when compared with the general population, both minor and major with incidences reported up to 9.5 – 16.5%[11,12].

**Hypoglycemia:**

Neonatal hypoglycemia is defined as plasma glucose concentration <40 mg/dl or serum glucose concentration <45 mg/dl.

Neonatal hypoglycemia occurs immediately after birth because of hyperinsulinemia that is persisting in newborns. At birth maternal supply of glucose is cut off, but insulin levels are disproportionately high resulting in a fall in blood glucose levels[1].

**Hypocalcemia:**

It is defined as a serum total concentration of <8mg/dl in term infants and <7mg/dl in preterm infants. It is found in around 7% of diabetic pregnancies[1].

**Polycythemia:**

It is defined as venous hematocrit > 65%. Maternal and subsequent fetal hyperglycemia lead to fetal tissue hypoxia which stimulates fetal erythropoietin production.

This causes increased viscosity of the blood leading to poor circulation, vascular sludging, ischemia, and microthrombi leading to infarction in vital organs like kidneys, brain, and adrenals.

**Hyperbilirubinemia:**

It usually manifests from the second day onwards in 20-25% of cases. It is found proportional to maternal

glucose levels during pregnancy. It is attributed to prematurity, immature hepatic bilirubin conjugation enzyme systems, and increased breakdown of red blood cells due to often associated neonatal polycythemia.

**Respiratory Distress Syndrome:**

Poor glycemic control delays fetal lung maturity and manifests as RDS in the newborn. A study conducted by Robert et al[13] concluded that there is 5-6 times more risk of developing RDS in neonates of pregestational diabetes than gestational diabetes.

The International Diabetic Federation alarming statistic reveals that one in every seven births in India is impacted by Gestational Diabetes Mellitus (GDM). Furthermore, a substantial proportion of women diagnosed with GDM progress to overt diabetes, contributing to complications that significantly affect their future health and well-being, resulting in varying degrees of morbidity. In light of these concerning trends, this study was undertaken to meticulously assess the maternal and fetal complications observed in patients diagnosed with Gestational Diabetes Mellitus.

The research was conducted within the Department of Obstetrics and Gynecology at a tertiary care hospital, aiming to provide critical insights into the challenges and complexities associated with GDM during pregnancy. By evaluating the specific complications experienced by both mothers and fetuses, this study seeks to contribute valuable information that can inform healthcare strategies, improve antenatal care protocols, and ultimately enhance the overall health outcomes for individuals grappling with Gestational Diabetes Mellitus in the Indian context.

Factors like maternal age, diet, obesity, lifestyle and artificial reproduction techniques are significantly contributing to the increasing prevalence of Diabetes and GDM in India. The geographical differences in the prevalence is attributed to the differences in the maternal age and socioeconomic status of the pregnant women in the different regions. Effective screening method, lifestyle modification and pharmacological therapies wil reduce the burden of the disease on maternal and neonatal health. Many associations have recommended different criteria to diagnose Diabetes in pregnancy. We adopted the DIPSI criteria as a norm to diagnose GDM. The prevalence data on GDM is influenced by the criteria used for diagnosis. It is important to define when to treat which was possible by doing the present study in our tertiary referral centre.

The various interventions we adopted in our centre provided us opportunities to improve the lives of mother and child in our region of North karnataka**.**

**Material and Methods:**

It was a cross sectional study. Data was collected from the Department of Obstetrics and Gynecology, in tertiary care hospital, with informed consent fitting the inclusion and exclusion criteria. After getting approval from the Institutional Ethical Committee, this study was started. A sample of 100 Pregnant women diagnosed with GDM was taken. Sample size was chosen with 95% Confidence interval, 5% level of significance and 10% relative error of prevalence.

The inclusion criteria encompass gestational diabetes mellitus patients who have undergone screening by GST following the DIPSI guidelines within the Department of Obstetrics and Gynecology at a tertiary care hospital. Exclusion criteria include individuals with pre-existing diabetes mellitus, hypertension, renal diseases, liver diseases, and autoimmune diseases.

**DIPSI** criteria: DIPSI (Diabetes In Pregnancy Study Group India) introduced in 2005 by V. Seshiah and colleagues proposes a streamlined approach to screening and diagnosing gestational diabetes mellitus (GDM) regardless of the last meal. Approved by the Ministry of Health, Government of India, this method involves administering 75 g of glucose in 250-350 ml of water to pregnant women, irrespective of their meal status. Plasma glucose levels are then measured after 2 hours, with a reading of ≥140 mg/dl indicating GDM.

Furthermore, this method provides a spectrum of glucose levels:

- 121-139 mg/dl: Impaired glucose tolerance
- 140–199 mg/dl: Gestational diabetes
- >200 mg/dl: Overt diabetes

The advantages of the DIPSI approach are noteworthy. Fasting status is not a prerequisite, allowing the test to be conducted during the first visit and easily repeated in the second and third trimesters. Importantly, it seamlessly integrates into a woman's routine, serving both as a screening and diagnostic procedure. This simplicity and practicality make it particularly well-suited for implementation in low-resource settings.

**Statistical analysis**

Data was entered into Microsoft excel data sheet and was analyzed using SPSS 22 version software. Categorical data was represented in the form of Frequencies and proportions.

Pearson's chi-square was used as test of significance for qualitative data.

**p value** (Probability that the result is true) of <0.05 was considered as statistically significant after assuming all the rules of statistical tests.

**Results:**

**Statistical software:** MS Excel**,** SPSS version 22 **(**IBM SPSS Statistics, Somers NY, USA) was used to analyze data.

**Table 1: Age-wise distribution of patients**

| Age groups | No of patients | % of patients |
|---|---|---|
| 21 - 25yrs | 24 | 24.00 |
| 26 - 30yrs | 44 | 44.00 |
| 31 - 35yrs | 24 | 24.00 |
| 36 -40yrs | 8 | 8.00 |
| Total | 100 | 100.00 |
| Mean±SD | 28.75±4.50 | |

Table 1 shows that the maximum population of pregnant women with GDM were found to be in the age group 26-30 years (44%). Elderly gravid pregnancies covered 8%. In this study occurrence of GDM was found to be less in extremes of age group. The lower incidence in the higher age group can be attributed to the factor that they would have been diagnosed with pregestational diabetes.

**Table 2: Mode of delivery-wise distribution of patients**

| Mode of delivery | No of patients | % of patients |
|---|---|---|
| Elective LSCS | 9 | 9.00 |
| Emergency LSCS | 39 | 39.00 |
| Vacuum-assisted | 4 | 4.00 |
| Vaginal | 48 | 48.00 |
| Total | 100 | 100.00 |

Table 2 shows that 52% of the study population underwent vaginal delivery.48% of these vaginal deliveries were natural birth and 4% were instrumental delivery. Macrosomia was found to be the indication in most of these instrumental deliveries.

48% of the study population delivered by lower segment cesarean section. Elective LSCS was the mode of delivery in 9% and emergency LSCS in 39%.

**Table 3: Treatment types wise distribution of patients**

| Treatment types | No of patients | % of patients |
|---|---|---|
| Diet | 62 | 62.00 |
| Insulin | 9 | 9.00 |
| OHA | 29 | 29.00 |
| Total | 100 | 100.00 |

**Table 4: Association between Modes of delivery and treatment types**

| Mode of delivery | Diet | Insulin | OHA | Total | Chi-square | p-value |
|---|---|---|---|---|---|---|
| Elective LSCS | 2 | 4 | 3 | 9 | | |
| Emergency LSCS | 23 | 2 | 14 | 39 | | |
| Vacuum-assisted | 3 | 1 | 0 | 4 | 18.2001 | 0.0011, S |
| Vaginal | 34 | 2 | 12 | 48 | | |
| Total | 62 | 9 | 29 | 100 | | |

Table 4 highlights that in the study population, the majority of women, constituting 62%, achieved glycemic control solely through a dietary plan. 9% were on insulin and 29% of the women were treated with OHA. 52% underwent vaginal delivery. A significant association was found between mode of delivery and treatment plan.

Cesarean section rate was found to be higher in those GDM women who were treated with insulin compared to those on the meal plan with p-value = 0.0011 which was statistically significant. This is attributed to the higher association of fetal macrosomia in these mothers.

Among the 52 women who delivered vaginally, 37 were on the diet plan, 3 were on insulin and 12 were on OHA.

**Table 5: Gestational age-wise distribution of patients at diagnosis**

| Gestational age | No of patients | % of patients |
|---|---|---|
| 32.0-33.0 | 12 | 12.00 |
| 33.1-34.0 | 14 | 14.00 |
| 34.1-35.0 | 52 | 52.00 |
| 35.1-36.0 | 16 | 16.00 |
| 36.1-37.0 | 5 | 5.00 |
| >37 | 1 | 1.00 |
| Total | 100 | 100.00 |

**Table 6: Polyhydramnios wise distribution of patients**

| Polyhydramnios | No of patients | % of patients |
|---|---|---|
| No | 83 | 83.00 |
| Yes | 17 | 17.00 |
| Total | 100 | 100.00 |

Table 6 shows that 17 women were found to have associated polyhydramnios in this study group. Of these women 46% were on a diet plan, 49% were on OHA, and the remaining 5% were on insulin. 15 out of 17 women with polyhydramnios had term delivery whereas only 2 women with polyhydramnios underwent preterm labor

**Table 7: Preeclampsia wise distribution of patients**

| Preeclampsia | No of patients | % of patients |
|---|---|---|
| Absent | 84 | 84.00 |
| NSPE | 13 | 13.00 |
| SPE | 3 | 3.00 |
| Total | 100 | 100.00 |

Table 7 highlights that Preeclampsia was noted in 16% of the study population. Severe pre-eclampsia was found in 3% and non-severe pre-eclampsia was noted in 13%.

Among the 3 women who developed severe pre-eclampsia 1 was on OHA and the remaining 2 were on a diet plan. Among women with non-severe pre-eclampsia, 4 were on OHA,1 was on insulin and 8 were managed by diet plan alone.

No significant association was found between treatment plan and occurrence of preeclampsia.

**Table 8: Association between polyhydramnios and Preterm labor**

| Polyhydramnios | Preterm | | | Chi-square | p-value |
|---|---|---|---|---|---|
| | Preterm - No | Preterm -Yes | Total | | |
| No | 73 | 10 | 83 | 0.0010 | 0.974 ko0 |
| Yes | 15 | 2 | 17 | | |
| Total | 88 | 12 | 100 | | |

Table 8 shows that There was no significant association found between polyhydramnios and preterm labor, p value- 0.9740.

**Table 9: Fetal outcome wise distribution of patients**

| Fetal outcome | No of patients | % of patients |
|---|---|---|
| Live birth | 96 | 96.00 |
| IUD | 3 | 3.00 |
| Both (Live birth and IUD)/twins | 1 | 1.00 |
| Total | 100 | 100.00 |

**Table 10: APGAR score-wise distribution of patients**

| APGAR score | No of patients | % of patients |
|---|---|---|
| 5min <3 | 5 | 4.9 |
| 5 min >3 | 96 | 95.1 |

Table 10 shows that All babies with 5 min APGAR Score less than 3 were said to have birth asphyxia, 4.9% had APGAR at 5min < 3 out of which 4 were IUD. Two of the babies had congenital anomalies. 96 babies had a 5 min APGAR > 3.

**Table 11: Birth weight wise distribution of patients**

| Birth weight | No of patients | % of patients |
|---|---|---|
| 2.0-2.5kg | 20 | 20.00 |
| 2.6-3.0kg | 35 | 35.00 |
| 3.1-3.5kg | 26 | 26.00 |
| 3.6-4.0kg | 13 | 13.00 |
| >=4.1kg | 9 | 9.00 |
| Mean±SD | 3.11±0.58 | |

**Table 12: Association between Macrosomia and treatment**

| Macrosomia | Diet | Insulin | OHA | Total | Chi-square | p-value |
|---|---|---|---|---|---|---|
| Macrosomia | 1 | 6 | 4 | 11 | 34.2990 | 0.0001 |
| Nil | 61 | 3 | 25 | 89 | | |
| Total | 62 | 9 | 29 | 100 | | |

Table 11 and 12 highlights the birth weight wise distribution of patients and association between macrosomia and treatment respectively. Most of the babies i.e, 35% had birth weight ranging from 2.6 to 3.5kg. 20% had low birth weight out of which 4 were preterm births.26% of babies weighed between 3.1 to 3.5kg.

11% had a birth weight of 3.6-4kg. Fetal macrosomia (4 kg and more) was found in 11% of babies. Out of these, 6 women were treated with insulin, 1 was on diet and 4 women were treated with OHA.

Significant association i.e., p-value of 0.0001 was found with women treated with insulin & fetal macrosomia. Thus, women treated with insulin had a higher risk of fetal macrosomia.

**Table 13: Hypoglycemia wise distribution of patients**

| Hypoglycemia | No of patients | % of patients |
|---|---|---|
| No | 92 | 92.00 |
| Yes | 8 | 8.00 |
| Total | 100 | 100.00 |

**Table 14: Hyperbilirubinemia wise distribution of patients**

| Hyperbilirubinemia | No of patients | % of patients |
|---|---|---|
| No | 83 | 83.00 |
| Yes | 17 | 17.00 |
| Total | 100 | 100.00 |

**Table 15: Association between NICU admission and treatment**

| NICU admission | Diet | Insulin | OHA | Total | Chi-square | p-value |
|---|---|---|---|---|---|---|
| No | 49 | 7 | 21 | 77 | 0.4920 | 0.7820 |
| Yes | 13 | 2 | 8 | 23 | | |
| Total | 62 | 9 | 29 | 100 | | |

**Discussion:**

GDM has been diagnosed as a clinical entity for the past 50 years. Early studies have strongly indicated untreated carbohydrate intolerance during pregnancy is associated with higher rates of maternal mortality and morbidity.

The purpose of screening, treatment, and management of GDM is to prevent stillbirth, congenital anomalies, pre-eclampsia, intrauterine death and decrease the incidence of macrosomic babies and cesarean section rates thereby reducing maternal and perinatal morbidity and mortality. The findings of the present study confirmed that GDM patients are liable to have adverse pregnancy outcomes.

The maximum incidence of GDM occurred between 26 to 30 years of age (44%). A higher incidence was noted in higher parity (70%). The maximum number of GDM cases was detected between 34 and 36weeks of gestation (68%), which is attributed to the fact that the maximum insulin resistance occurs at this age which was also reinforced by Rajesh kumara et al [14].

Rajesh kumari et al studied maternal and fetal outcome in GDM women for 5 years in tertiary care hospital, Delhi. The prevalence of GDM was 5.72%, maximum cases were found between 32-36 weeks of gestation (62%). Mode of delivery was not different in different treatment modalities. Incidence of macrosomia was 21%. Among neonatal complications, hypoglycemia was significantly higher (32%)

Mutum Matouleibi et al studied that cesarean section rates were higher in women with GDM (52%)[15]. It was also associated with an increased frequency of preterm labor and polyhydramnios in GDM patients. In present study, there was no increase in caesarean section (48%) in relation to vaginal deliveries (52%).

Ameya R et al conducted a study on maternal and neonatal outcome in GDM and found out that 26% of GDM mothers were complicated with pre-eclampsia[16]. This study showed 16% of GDM mothers developing pre-eclampsia. Ingrid Ostlund et al[17] studied association of pre-eclampsia in gestational diabetes mellitus where rate of pre-eclampsia was higher in the GDM group than in the non GDM (6.1% vs 2.8%) and adjusted odd's ratio for GDM as a risk factor for pre-eclampsia was 1.61

The occurrence of pre-eclampsia in GDM was found to be 30% by Krishnamoorthy et al[18], 9% was the incidence of preterm labor and 8% had PROM [18]. In the present study, preterm labor was observed in 8% and PROM in 6% of GDM.

Monique M Hedderson et al investigated whether different degrees of maternal glucose tolerance are associated with the risk of spontaneous pre term births. Incidence of spontaneous preterm birth was 6.7% in GDM[19].

In regards to fetal complications, the incidence of macrosomia was 11% in this study whereas higher incidence was noted in the other studies (40% in the study by Ameya et al[16] and 23% in the study by Mutum Matouleibi et al[15]). In the present study significant association i.e., p-value of 0.0001 was found with women treated with insulin & fetal macrosomia. Thus, women treated with insulin had a higher risk of fetal macrosomia.

Michael Lynge et al observed 24.7% of macrosomia in GDM. Macrosomia was found to be associated with high BMI in mother in pre-pregnancy stage[20].

Hypoglycemia was noted in 8% of the study population and 6 of these required NICU admissions which was lower compared to a prospective cohort study conducted by Daphne N which found 20% incidence of hypoglycemia in neonates of GDM[21].

Adverse fetal outcome (stillborn, intrauterine death) was seen in 4% of the study population. Study conducted by BA Girz et al found stillbirth rate for women with gestational diabetes was 7.7 per 1000[22].

Isabelle Mortier et al observed 20% incidence of respiratory distress syndrome in neonates of GDM mothers[23]. In this study 9% neonates developed RDS and required NICU admissions.

23% in this study required NICU admission which is similar in a study conducted by Diana Watson et al where NICU admissions rate was 29%. Diana Watson et al conducted 2-year study to observe NICU admissions in neonates born to GDM and pre gestational diabetes women. Admission to NICU occurred in 29% of GDM and 40% of type 2 DM pregnancies. Median gestation was 37 weeks with 46% preterm delivery[24].

**Conclusion:**

Though GDM is a transient condition, its sequelae are long-lasting. Therefore, all antenatal women attending the OPD should be offered a simple glucose screening test and with this intervention we can reduce the complications.

Strict glycemic control is necessary for pregnancy to minimize complications. Dietary and lifestyle modifications can reduce both maternal and neonatal

complications. Patient needs to be counseled regarding proper follow up and blood sugar levels evaluation in the postpartum period. Early detection and prompt management of GDM significantly decreases the complication in both the mother and neonate.

## Recommendation

In light of the research findings it is recommended that all practicing obstetricians consider adopting the DIPSI criteria for all the pregnant women as a screening method of GDM. The MNT (medical nutritional therapy or dietary management) and home glucose monitoring becomes increasingly important. These interventions have been recommended to ensure that there are lesser complications in the mother and the neonate.

## References


1. *Verma NP, Mehta SP, Madhu S, Mather HM, Keen H. Prevalence of known diabetes in an urban Indian environment: the Darya Ganj diabetes survey. Br Med J (Clin Res Ed). 1986 Aug 16;293(6544):423-4.*
2. *Willkerson HLC, Remeien. QR. Studies of abnormal Carbohydrate Metabolism in Pregenancy. Diabetes 1957; 6:324-9*
3. *O'Sullivan JB. Unsuspected Asymptomatic Diabetes in Pregnancy.1961:264:1082-5*
4. *Pederson J. The Pregnancy Diabetic and her new born problems and Management. Copenhagen 1946.*
5. *Damm P. Gestational Diabetes Mellitus and Subsequent Development of Over diabetes – a Clinical, Metabolic and Epidemiology Study. University of Copenhagen 1998*
6. *Crowther CA, Hiller JE, Moss JR, McPhee AJ, Jeffries WS, Robinson JS; Australian Carbohydrate Intolerance Study in Pregnant Women (ACHOIS) Trial Group. Effect of treatment of gestational diabetes mellitus on pregnancy outcomes. N Engl J Med. 2005 Jun 16;352(24):2477-86.*
7. *Weiss PA, Hofmann H, Winter R, Pürstner P, Lichtenegger W. Amniotic fluid glucose values in normal and abnormal pregnancies. Obstet Gynecol. 1985 Mar;65(3):333-9. PMID: 3883262.*
8. *Strangenberg N – Amniotic fluid volume and concentration of C. Peptide in diabetic pregnancies. ObstetGynecol 1982; 89:536-42*
9. *Diagnosis and Treatment of Gestational Diabetes Scientific Impact Paper No. 23, January 2011, RCOG*
10. *ACOG GDM practice bulletin: no 190; feb 2018*
11. *Aberg A, Westbom L, Källén B. Congenital malformations among infants whose mothers had gestational diabetes or preexisting diabetes. Early Hum Dev. 2001 Mar;61(2):85-95.*
12. *Towner D, Kjos SL, Leung B, Montoro MM, Xiang A, Mestman JH, Buchanan TA. Congenital malformations in pregnancies complicated by NIDDM. Diabetes Care. 1995 Nov;18(11):1446-51.*
13. *Robert MF, Neff RK Association between Maternal Diabetes and Respiratory Distress Syndrome in the new born. N E Engle J Med 1976; 294:357-60*
14. *Kumari R, Dalal V, Kachhawa G, Sahoo I, Khadgawat R, Mahey R, Kulshrestha V, Vanamail P, Sharma JB, Bhatla N, Kriplani A. Maternal and Perinatal Outcome in Gestational Diabetes Mellitus in a Tertiary Care Hospital in Delhi. Indian J Endocrinol Metab. 2018 Jan-Feb;22(1):116-120. doi: 10.4103/ijem.IJEM_582_17. PMID: 29535949; PMCID: PMC5838890.*
15. *Mutum Matouleibi Chanu. Alisha June. Clinical study on fetomaternal outcome in GDM.IOSR Journal.vol14. issue 4 (April 2015)53-56q*
16. *Ameya R Dudhwadhkar, Michelle N Fonseca. Maternal and fetaloutcome in Gestational Diabetes Mellitus. Indian journal of Reproduction andContraception.2016 oct 5(10)3317-3321*
17. *Ostlund I, Haglund B, Hanson U. Gestational diabetes and preeclampsia. Eur J ObstetGynecolReprod Biol. 2004 Mar 15;113(1):12-6.*
18. *Maternal diabetes in pregnancy: Is it time for meaningful research to inform preventive and management strategies? U Krishnamoorthy et AL: November 2006.BJOG,113(10):1134-40*
19. *Association with increased risk of spontaneous preterm birth. Obstet Gynecol. 2003 Oct;102(4):850-6. Hedderson MM, Ferrara A, Sacks DA. Gestational diabetes mellitus and lesser degrees of pregnancy hyperglycemia*
20. *Pedersen ML, Lind O, Abelsen T, Olesen J, Jørgensen ME. Gestational diabetes and macrosomia among Greenlanders. Time to change diagnostic strategy? Int J Circumpolar Health. 2018 Dec;77(1):1528126.*
21. *Following Diet-Controlled and Insulin-Treated Gestational Diabetes Mellitus. Diabetes Care. 2018 Jul;41(7):1385-1. Voormolen DN, de Wit L, van Rijn BB, DeVries JH, Heringa MP, Franx A, Groenendaal F, Lamain-de Ruiter M. Neonatal Hypoglycemia 390.*
22. *Girz BA, Divon MY, Merkatz IR. Sudden fetal death in women with well-controlled, intensively monitored gestational diabetes. J Perinatol. 1992 Sep;12(3):229-33. PMID: 1432278.*
23. *Mortier I, Blanc J, Tosello B, Gire C, Bretelle F, Carcopino X. Is gestational diabetes an independent risk factor of neonatal severe respiratory distress syndrome after 34 weeks of gestation? A prospective study. Arch Gynecol Obstet. 2017 Dec;296(6):1071-1077.*
24. *Watson D, Rowan J, Neale L, Battin MR. Admissions to neonatal intensive care unit following pregnancies complicated by gestational or type 2 diabetes. Aust N Z J ObstetGynaecol. 2003 Dec;43(6):429-32.*


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