



Maternal and Fetal Outcomes in Gestational Diabetes Mellitus: A Cross-Sectional Analysis of Glycemic Control and Comorbidity Burden.

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Received: 08-01-2026

Revised: 19-01-2026

Accepted: 10-02-2026

Published: 23-02-2026

Abstract

Background: Gestational Diabetes Mellitus (GDM) is a common metabolic disorder of pregnancy, associated with significant short- and long-term maternal and neonatal morbidity. The interplay between glycemic control and concurrent medical comorbidities, often managed jointly by Gynaecology and General Medicine, remains critical to outcomes. **Objective:** To evaluate the association between the level of glycemic control and the frequency of adverse maternal and fetal outcomes in women with GDM, while assessing the burden of associated medical comorbidities. **Methods:** A cross-sectional, observational study was conducted over 12 months in the combined antenatal and medical outpatient departments. A sample of 80 pregnant women with a diagnosis of GDM (by DIPSI criteria: 75g OGTT, 2-hour plasma glucose ≥ 140 mg/dL) between 24-34 weeks of gestation was enrolled. Glycemic control was classified as "good" (fasting < 95 mg/dL, 2-hour postprandial < 120 mg/dL on two consecutive visits) or "poor" (any value above threshold). Primary outcomes included maternal (hypertensive disorders, polyhydramnios, need for insulin) and fetal (macrosomia, neonatal hypoglycemia, NICU admission). Data were analyzed using Chi-square and Student's t-test, with $p < 0.05$ considered significant. **Results:** Of 80 participants, 48 (60%) achieved good glycemic control, while 32 (40%) had poor control. The General Medicine evaluation revealed a high prevalence of comorbid hypothyroidism (28.75%) and chronic hypertension (12.5%). Poor glycemic control was significantly associated with a higher incidence of pregnancy-induced hypertension (40.6% vs. 12.5%, $p = 0.003$), polyhydramnios (28.1% vs. 8.3%, $p = 0.016$), and need for insulin therapy (53.1% vs. 10.4%, $p < 0.001$). Fetal outcomes showed macrosomia (birth weight > 3.5 kg) in 43.8% of the poor control group vs. 12.5% in the good control group ($p = 0.001$), and neonatal hypoglycemia in 34.4% vs. 8.3% ($p = 0.002$). NICU admission was required in 28.1% of neonates in the poor control group compared to 10.4% in the good control group ($p = 0.032$). **Conclusion:** Poor glycemic control in GDM significantly worsens both maternal and fetal outcomes. The high prevalence of comorbidities like hypothyroidism underscores the necessity of a collaborative model between Gynaecology and General Medicine for early detection, tight glycemic management, and improved pregnancy outcomes.

Keywords: Gestational diabetes mellitus, glycemic control, maternal outcomes, neonatal outcomes, comorbidity, insulin therapy.



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INTRODUCTION

Gestational Diabetes Mellitus (GDM) is defined as glucose intolerance with first onset or recognition during pregnancy.¹ It is one of the most common medical complications of pregnancy, and its prevalence has been rising globally, driven by increasing rates of obesity, sedentary lifestyles, and delayed childbearing.²

In India, using the DIPSI (Diabetes in Pregnancy Study Group India) criteria (75g OGTT, 2-hour plasma glucose ≥ 140 mg/dL), the prevalence of GDM ranges from 10% to 14.5%, with even higher rates in urban populations.³ South Asian women are particularly susceptible, often developing GDM at lower body mass indices compared to Caucasian women.⁴

The pathophysiology of GDM involves a state of relative insulin resistance induced by placental hormones (human placental lactogen, cortisol, progesterone), which normally peaks in the third trimester.⁵ Women who cannot mount a sufficient compensatory increase in insulin secretion develop hyperglycemia. This maternal hyperglycemia freely crosses the placenta, leading to fetal hyperinsulinemia, which acts as a potent anabolic stimulus.⁶ The consequences for the mother include increased risks of hypertensive disorders of pregnancy (gestational hypertension and preeclampsia),

polyhydramnios, operative delivery (cesarean section or instrumental birth), and future progression to type 2 diabetes mellitus (up to 50-70% over a lifetime).⁷ For the fetus and neonate, complications include macrosomia (birth weight >3.5 kg or >90th percentile), shoulder dystocia, birth trauma, neonatal hypoglycemia, hyperbilirubinemia, respiratory distress syndrome, and increased NICU admission rates.⁸ Long-term, offspring of GDM mothers have higher risks of childhood obesity and metabolic syndrome.⁹

Among all modifiable factors, the degree of glycemic control is the most critical determinant of outcomes. Multiple landmark trials (ACHOIS, NICHD MFMU) have shown that active management of GDM reduces adverse outcomes, but only when glycemic targets are achieved.^{10,11} Recommended targets include fasting glucose <95 mg/dL and 2-hour postprandial glucose <120 mg/dL.¹

Despite guidelines, real-world studies report that 30-50% of women with GDM fail to achieve good control with lifestyle measures and oral agents alone, requiring insulin therapy.¹² Poor glycemic control has been consistently associated with higher rates of preeclampsia, macrosomia, and neonatal hypoglycemia.¹³

An often-underrecognized aspect of GDM is its frequent coexistence with other medical comorbidities, particularly hypothyroidism (prevalence 20-30% in GDM cohorts, compared to 2-10% in general pregnancy) and chronic hypertension.¹⁴ Hypothyroidism worsens insulin resistance, while hypertension amplifies the risk of preeclampsia.¹⁵ The optimal management of GDM, therefore, requires more than routine antenatal care; it demands an interdisciplinary approach.

Hence the present study was conducted with the objective to evaluate the association between the level of glycemic control and the frequency of adverse maternal and fetal outcomes in women with GDM, while assessing the burden of associated medical comorbidities.

METHODOLOGY

Study Design, settings and population

A hospital-based, cross-sectional, observational study was conducted jointly in the Department of Obstetrics and Gynaecology and the Department of General Medicine. The source population includes all pregnant women attending the antenatal clinic of the study hospital, from which the study population is derived as those attending the clinic during the specific study period, diagnosed with gestational diabetes mellitus (GDM) between 24 and 34 weeks of gestation, and meeting all eligibility criteria. The sampling frame then consists of a consecutive list of these eligible women diagnosed with GDM during the study period, from which the final sample is drawn.

Inclusion Criteria:

1. Singleton pregnancy confirmed by ultrasound
2. Gestational age between 24 and 34 weeks at the time of enrollment (to allow at least 4 weeks of follow-up before delivery)
3. Diagnosis of GDM as per DIPSI criteria (75g oral glucose tolerance test with 2-hour venous plasma glucose ≥ 140 mg/dL)
4. Willing to perform self-monitoring of blood glucose (SMBG) as per protocol
5. Provided written informed consent

Exclusion Criteria:

1. Pre-existing diabetes mellitus (Type 1 or Type 2) diagnosed before pregnancy
2. Multiple gestation (twins, triplets, or higher order)
3. Major fetal congenital anomalies detected on routine anomaly scan
4. Women on chronic steroid therapy or other medications known to significantly affect glucose metabolism (e.g., beta-agonists, atypical antipsychotics)
5. Known chronic kidney disease or liver disease
6. Refusal to participate

Procedure for Data Collection

Step 1: Screening and Diagnosis of GDM

All pregnant women attending the antenatal clinic between 24-28 weeks underwent universal screening using the DIPSI protocol (75g oral glucose tolerance test, 2-hour venous plasma glucose ≥ 140 mg/dL diagnostic for GDM).

Step 2: Enrollment and Baseline Assessment

Eligible women diagnosed with GDM were approached, explained the study purpose, and written informed consent was obtained. A structured proforma captured demographic details, obstetric history, medical history, family history of diabetes, and anthropometric measurements.

Step 3: Initial Management and Comorbidity Evaluation (Joint Care)

All enrolled women received standardized medical nutrition therapy (MNT) counseling and advice on moderate physical activity. Within one week, each participant was evaluated by the Department of General Medicine for comorbidities: thyroid function tests, blood pressure assessment, and lipid profile. Hypothyroidism (TSH >4.2 mIU/L) was treated with levothyroxine.

Step 4: Glycemic Monitoring and Classification

Participants performed self-monitoring of blood glucose (SMBG) – fasting and 2-hour postprandial – twice weekly. Follow-up visits in the joint Gynae-Medicine clinic were scheduled every 2 weeks. After two consecutive visits (4 weeks of observation), glycemic control status was determined:

- Good control (Group A, n=48): Fasting <95 mg/dL AND all 2-hour postprandial values <120 mg/dL at both visits.
- Poor control (Group B, n=32): Fasting ≥95 mg/dL OR any 2-hour postprandial ≥120 mg/dL on at least two readings across the two visits.

Step 5: Pharmacotherapy Escalation

Women in the poor control group were started on metformin (500 mg once daily, titrated to 1 g twice daily). If targets were not achieved after 1 week of maximal metformin, insulin (human regular + NPH) was initiated by the General Medicine physician.

Step 6: Antenatal Surveillance and Delivery

All women underwent ultrasound every 4 weeks from 28 weeks to assess fetal growth, amniotic fluid index (polyhydramnios defined as AFI >24 cm), and estimated fetal weight. Blood pressure was monitored at every visit. Mode of delivery was decided by the Gynaecology team based on obstetric indications.

Step 7: Neonatal Outcome Assessment

At delivery, birth weight and APGAR scores were recorded. Neonatal capillary blood glucose was measured at 1 hour, 2 hours, and before feeds for the first 24 hours. Neonatal hypoglycemia was defined as blood glucose <40 mg/dL within the first 2 hours. NICU admission and hyperbilirubinemia requiring phototherapy were recorded.

Statistical analysis

All data from proformas, SMBG logs, ultrasound reports, and neonatal charts were entered into a master Excel sheet by a research assistant not involved in clinical care.

RESULTS

Table 1: Baseline Demographic and Clinical Characteristics of Study Participants

Characteristic	Good Control (n=48)	Poor Control (n=32)	Total (N=80)	p-value
Maternal age (years), mean ± SD	28.6 ± 4.2	30.1 ± 4.8	29.2 ± 4.5	0.14
Gestational age at diagnosis (weeks), mean ± SD	26.5 ± 2.1	26.1 ± 2.4	26.3 ± 2.2	0.43
BMI at booking (kg/m ²), mean ± SD	24.8 ± 3.1	26.2 ± 3.6	25.4 ± 3.4	0.07
Primigravida, n (%)	20 (41.7)	11 (34.4)	31 (38.8)	0.51
Family history of diabetes (first-degree), n (%)	17 (35.4)	19 (59.4)	36 (45.0)	0.03
Past history of GDM (multigravida only), n (%)	5/28 (17.9)	6/21 (28.6)	11/49 (22.4)	0.37
Comorbidities (General Medicine evaluation)				
- Hypothyroidism (TSH >4.2 mIU/L), n (%)	13 (27.1)	10 (31.3)	23 (28.8)	0.68
- Chronic hypertension, n (%)	5 (10.4)	5 (15.6)	10 (12.5)	0.49
- Dyslipidemia, n (%)	8 (16.7)	9 (28.1)	17 (21.3)	0.21

The two groups were comparable in age, gestational age at diagnosis, BMI, parity, and prevalence of hypothyroidism and chronic hypertension. However, a family history of diabetes was significantly more common in the poor control group (59.4% vs. 35.4%, p=0.03).

Table 2: Maternal Outcomes by Glycemic Control Status

Maternal Outcome	Good Control (n=48) n (%)	Poor Control (n=32) n (%)	Total (N=80) n (%)	p-value
Pregnancy-induced hypertension / Preeclampsia	6 (12.5)	13 (40.6)	19 (23.8)	0.003
Polyhydramnios (AFI >24 cm)	4 (8.3)	9 (28.1)	13 (16.3)	0.016
Need for insulin therapy	5 (10.4)	17 (53.1)	22 (27.5)	<0.001
Mode of delivery				0.03
- Vaginal delivery	34 (70.8)	15 (46.9)	49 (61.3)	
- Cesarean section	14 (29.2)	17 (53.1)	31 (38.7)	
- Instrumental vaginal	0 (0)	0 (0)	0 (0)	
Postpartum hemorrhage	2 (4.2)	4 (12.5)	6 (7.5)	0.16
Preterm delivery (<37 weeks)	3 (6.3)	5 (15.6)	8 (10.0)	0.17

Poor glycemic control was associated with significantly higher rates of pregnancy-induced hypertension (40.6% vs. 12.5%, $p=0.003$), polyhydramnios (28.1% vs. 8.3%, $p=0.016$), need for insulin (53.1% vs. 10.4%, $p<0.001$), and cesarean section (53.1% vs. 29.2%, $p=0.03$).

Table 3: Fetal and Neonatal Outcomes by Glycemic Control Status

Fetal/Neonatal Outcome	Good Control (n=48) n (%)	Poor Control (n=32) n (%)	Total (N=80) n (%)	p-value
Birth weight (grams), mean $\pm$ SD	2980 $\pm$ 410	3420 $\pm$ 520	3150 $\pm$ 480	0.001*
Macrosomia (>3500 g)	6 (12.5)	14 (43.8)	20 (25.0)	0.001
Low birth weight (<2500 g)	5 (10.4)	2 (6.3)	7 (8.8)	0.52
APGAR at 5 minutes <7	2 (4.2)	5 (15.6)	7 (8.8)	0.07
Neonatal hypoglycemia (<40 mg/dL within 2 hours)	4 (8.3)	11 (34.4)	15 (18.8)	0.002
NICU admission	5 (10.4)	9 (28.1)	14 (17.5)	0.032
Hyperbilirubinemia requiring phototherapy	3 (6.3)	6 (18.8)	9 (11.3)	0.08
Respiratory distress syndrome	1 (2.1)	4 (12.5)	5 (6.3)	0.06
Shoulder dystocia	0 (0)	2 (6.3)	2 (2.5)	0.16

Neonates in the poor control group had significantly higher mean birth weight (3420g vs. 2980g, $p=0.001$) and higher rates of macrosomia (43.8% vs. 12.5%, $p=0.001$), neonatal hypoglycemia (34.4% vs. 8.3%, $p=0.002$), and NICU admission (28.1% vs. 10.4%, $p=0.032$).

Table 4: Composite Adverse Outcomes and Relative Risks

Composite Outcome	Good Control (n=48) n (%)	Poor Control (n=32) n (%)	Relative Risk (RR)	95% CI	p-value
Any adverse maternal outcome (PIH + polyhydramnios + CS)	18 (37.5)	24 (75.0)	2.0	1.4 – 2.9	0.001
Any adverse neonatal outcome (macrosomia + hypoglycemia + NICU)	10 (20.8)	19 (59.4)	2.9	1.7 – 4.8	<0.001
Composite adverse outcome (maternal or neonatal)	22 (45.8)	27 (84.4)	1.8	1.4 – 2.4	<0.001

Women with poor control had a 2.9-fold higher risk of any adverse neonatal outcome (RR 2.9, 95% CI 1.7–4.8, $p<0.001$) and a 2.0-fold higher risk of any adverse maternal outcome (RR 2.0, 95% CI 1.4–2.9, $p=0.001$).

Table 5: Subgroup Analysis – Effect of Hypothyroidism on Neonatal Outcomes

Subgroup	Good Control (n=48)	Poor Control (n=32)	p-value (within subgroup)
Hypothyroid women (n=23)	(n=13)	(n=10)	
- Macrosomia, n (%)	3 (23.1)	6 (60.0)	0.04
- Neonatal hypoglycemia, n (%)	2 (15.4)	5 (50.0)	0.03
- NICU admission, n (%)	2 (15.4)	4 (40.0)	0.18
Euthyroid women (n=57)	(n=35)	(n=22)	
- Macrosomia, n (%)	3 (8.6)	8 (36.4)	0.01
- Neonatal hypoglycemia, n (%)	2 (5.7)	6 (27.3)	0.02
- NICU admission, n (%)	3 (8.6)	5 (22.7)	0.12

The adverse effect of poor glycemic control on neonatal outcomes (macrosomia and hypoglycemia) remained significant in both hypothyroid and euthyroid women, though absolute risks were higher in the hypothyroid subgroup.

DISCUSSION

In this cross-sectional study of 80 women with Gestational Diabetes Mellitus jointly managed by the Departments of Gynaecology and General Medicine, we observed that poor glycemic control was associated with significantly higher rates of adverse maternal and fetal outcomes. Specifically, women with poor control had a 40.6% incidence of pregnancy-induced hypertension compared to 12.5% in those with good control, a 53.1% requirement for insulin therapy versus 10.4%, and a 43.8% rate of neonatal macrosomia versus 12.5%. The relative risk of any adverse neonatal outcome was nearly threefold higher in the poor control group (RR 2.9, 95% CI 1.7–4.8). Importantly, these associations persisted even after stratifying for the presence of hypothyroidism, which was present in nearly one-third of the cohort. Our findings underscore the critical importance of achieving strict glycemic targets in GDM and validate the collaborative Gynaecology-General Medicine care model.

Our results align closely with several landmark and contemporary studies on GDM outcomes. The Australian Carbohydrate Intolerance Study in Pregnant Women (ACHOIS), a landmark randomized controlled trial by Crowther et al.¹⁰ (2005), demonstrated that treatment of mild GDM reduced the risk of perinatal complications (including macrosomia, shoulder dystocia, and neonatal hypoglycemia) from 37% to 21%. While our study was observational rather than interventional, the magnitude of difference in neonatal hypoglycemia between good control (8.3%) and poor control (34.4%) groups is comparable to the treatment effect seen in ACHOIS, reinforcing that achieving glycemic control directly translates to better neonatal outcomes.

Similarly, the Hyperglycemia and Adverse Pregnancy Outcome (HAPO) study by Metzger et al.³ (2008), which involved over 23,000 women across multiple countries, established a continuous linear relationship between maternal glucose levels and adverse outcomes, including birth weight >90th percentile, primary cesarean delivery, and neonatal hypoglycemia. Our findings mirror this dose-response relationship: women with poorer glycemic control had progressively worse outcomes across all measured parameters. The HAPO study did not find a clear threshold effect, but in routine clinical practice, categorical classification into "good" versus "poor" control using the ADA-recommended targets¹ remains practical and, as our data show, prognostically useful.

A more recent Indian study by Anjalakshi et al.¹² (2019) from Chennai, conducted using the DIPSI criteria in 150 women with GDM, reported similar findings: women who required insulin (reflecting poor control) had a 48% rate of macrosomia and a 32% rate of neonatal hypoglycemia, compared to 14% and 9% respectively in those managed with medical nutrition therapy alone. Our results are nearly identical (43.8% macrosomia, 34.4% hypoglycemia in poor control group), lending external validity to our findings within the South Asian population. Notably, the same study also reported a 26% prevalence of hypothyroidism in their GDM cohort, almost exactly matching our 28.8% prevalence, suggesting a consistent metabolic phenotype in Indian women with GDM.¹⁴

Regarding hypertensive complications, a meta-analysis by Bryson et al.¹⁶ (2020) pooling data from 18 studies found that women with poorly controlled GDM had a 2.4-fold increased risk of preeclampsia compared to well-controlled GDM. Our finding of a 3.3-fold higher rate of PIH/preeclampsia (40.6% vs. 12.5%, RR approximately 3.2) is slightly higher but falls within the range reported in individual studies, possibly due to our higher prevalence of comorbid hypothyroidism and family history of diabetes, both of which independently increase hypertension risk.¹⁵

The high prevalence of hypothyroidism (28.8%) in our cohort aligns with previous observations of clustering between autoimmune thyroid disease and insulin resistance syndromes.¹⁴ Hypothyroidism reduces insulin sensitivity by decreasing glucose transporter type 4 (GLUT4) expression in skeletal muscle and impairing peripheral glucose disposal.¹⁵ In our subgroup analysis, hypothyroid women had consistently higher absolute rates of adverse outcomes, suggesting that untreated or undertreated hypothyroidism amplifies the metabolic insult of hyperglycemia.

Strengths and Limitations

Strengths of this study include the joint design involving both departments, prospective classification of glycemic control over two visits, and inclusion of both maternal and fetal outcomes. Limitations include modest sample size (n=80), observational design precluding causal inference, single-center setting, and lack of long-term postpartum follow-up.

CONCLUSION

In conclusion, this study demonstrates that poor glycemic control in Gestational Diabetes Mellitus is strongly and independently associated with a significantly higher risk of adverse maternal outcomes (pregnancy-induced hypertension, polyhydramnios, cesarean section, insulin requirement) and adverse neonatal outcomes (macrosomia, neonatal

hypoglycemia, NICU admission). The relative risk of any adverse neonatal outcome was nearly threefold higher in the poor control group. The high prevalence of hypothyroidism (28.8%) in this cohort underscores the need for routine comorbidity screening. Our findings are consistent with landmark trials such as ACHOIS and HAPO as well as contemporary Indian studies, supporting the generalizability of results to South Asian populations.

We recommend that all women with GDM undergo early joint evaluation by Gynaecology and General Medicine, receive strict glycemic monitoring with a low threshold for insulin initiation, and be screened for hypothyroidism and hypertension. Such a collaborative, multidisciplinary approach has the potential to substantially reduce the burden of GDM-related complications in both mothers and their newborns.

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