

# Non-obese gestational diabetes

## A unique risk profile for maternal and fetal outcomes

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### Abstract

This retrospective study aimed to compare maternal and neonatal outcomes in non-obese and obese gestational diabetes mellitus (GDM) patients to determine whether body mass index (BMI) exerts an independent effect on adverse pregnancy events. A total of 400 pregnant women diagnosed with GDM (200 non-obese and 200 obese) were evaluated. Relevant data, including baseline demographics, clinical parameters, and both maternal and neonatal outcomes, were extracted from the hospital records. Comparisons between groups were performed using standard statistical tests, and multivariable logistic regression models were constructed to identify independent predictors of complications, such as preeclampsia, macrosomia, and cesarean delivery. The obese GDM group demonstrated significantly elevated risks of preeclampsia, cesarean delivery, macrosomia, and neonatal intensive care unit admission. Logistic regression analyses identified elevated BMI and higher glycated hemoglobin levels as independent risk factors for adverse outcomes. While non-obese GDM patients generally had fewer complications, they were not exempt from critical risks, underscoring the importance of vigilant monitoring, regardless of BMI. These findings emphasize that both non-obese and obese women with GDM require comprehensive management. Early detection optimized glycemic control, and targeted perinatal follow-up may help mitigate complications and improve overall maternal and neonatal health across diverse BMI categories.

**Abbreviations:** BMI = body mass index, GDM = gestational diabetes mellitus, HbA1c = glycated hemoglobin, HT = hypertension, IUGR = intrauterine growth restriction, NICU = neonatal intensive care unit, OGTT = oral glucose tolerance test, OR = odds ratio.

**Keywords:** body mass index, gestational diabetes mellitus, maternal outcomes, neonatal outcomes, non-obese

### 1. Introduction

Gestational diabetes mellitus (GDM) is a metabolic condition characterized by the onset or initial recognition of glucose intolerance during pregnancy. It can lead to significant complications in both mothers and fetuses, potentially affecting short- and long-term health outcomes. While most discussions around GDM have traditionally focused on obesity as a primary risk factor, there is growing awareness that women with a normal body mass index (BMI) can also develop GDM.<sup>[1]</sup> Non-obese GDM patients may represent a unique subgroup with a distinct risk profile that differs from their obese counterparts, emphasizing the need for more targeted research and clinical strategies.<sup>[2]</sup>

The prevalence of GDM varies widely and is influenced by factors such as ethnicity, diagnostic criteria, and healthcare

practices. Although elevated BMI is strongly associated with insulin resistance and hyperglycemia, GDM can still occur in women without obesity.<sup>[3]</sup> The exact mechanisms underlying glucose intolerance in non-obese individuals remain poorly understood, and may involve diverse genetic, epigenetic, and lifestyle factors. Consequently, clinicians and researchers are confronted with the challenge of identifying high-risk, non-obese women who might otherwise be overlooked in standard screening processes.<sup>[4]</sup>

Maternal complications such as preeclampsia, gestational hypertension (GHT), cesarean delivery, and postpartum hemorrhage are often discussed in the context of obese pregnant women with GDM. However, non-obese women with GDM may still face significant challenges, even if their baseline risk profiles appear lower owing to a normal BMI. Emerging

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*The data that support the findings of this study are available from a third party, but restrictions apply to the availability of these data, which were used under license for the current study, and so are not publicly available. Data are available from the authors upon reasonable request and with permission of the third party.*

*This retrospective study was approved by the Ethics Committee of Haseki Training and Research Hospital (Approval No.: 06-2025, Date: January 30, 2025). Informed consent was not required because the study involved the use of anonymized data obtained from archived patient records, in accordance with national regulations and institutional guidelines.*

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evidence suggests that the incidence of certain maternal complications in non-obese GDM patients can be similar to or only modestly lower than those in obese women with GDM.<sup>[5]</sup> Such findings underscore the importance of close monitoring and early intervention in this subset of pregnant women, as failure to diagnose or adequately manage GDM can increase adverse outcomes regardless of BMI.<sup>[6]</sup>

Maternal hyperglycemia can lead to multiple fetal complications, most notably macrosomia. In obese pregnant women, the combination of excess adipose tissue, inflammatory pathways, and insulin resistance can exacerbate fetal overgrowth. Non-obese women with GDM, on the other hand, may present different metabolic dynamics, potentially resulting in varying rates of macrosomia or even an increased risk of intrauterine growth restriction (IUGR) in certain populations.<sup>[7]</sup> In addition to birth weight, neonates born to mothers with GDM are also at risk of complications, such as neonatal hypoglycemia, lower Apgar scores, and higher rates of admission to neonatal intensive care units (NICU). However, the degree to which these outcomes differ between obese and non-obese GDM groups remains an area of ongoing investigation.<sup>[8]</sup>

The clinical outcomes of GDM do not depend solely on BMI. Other variables such as maternal age, obstetric history, ethnicity, and comorbid conditions play equally critical roles. Non-obese mothers may develop GDM at different gestational stages, and their insulin resistance patterns may progress differently from those of obese pregnant women. This heterogeneity in clinical presentations and trajectories poses a challenge for standardizing care protocols but also highlights the importance of individualized patient management. In addition, how parameters such as glycated hemoglobin (HbA1c) or oral glucose tolerance test (OGTT) values differ between non-obese and obese GDM groups is still not fully elucidated, although it could have key implications for early diagnosis and targeted treatment strategies.<sup>[9]</sup>

Much of the existing research and clinical guidelines for GDM management have been shaped by studies that have focused on pregnant women with obesity. As a result, risk stratification tools and treatment algorithms may not adequately address the nuances of GDM in women with normal BMI. Identifying non-obese GDM patients who require aggressive intervention versus those who may respond well to diet and exercise is crucial for optimizing pregnancy outcomes.<sup>[10]</sup> Without sufficient data on this subgroup, healthcare providers may either overtreat or undertreat non-obese GDM patients, jeopardizing both maternal and fetal well-being. Moreover, clarifying the unique risk factors and comorbidities associated with non-obese GDM can inform early intervention strategies and support the development of personalized management plans.<sup>[11]</sup>

Countries with diverse BMI distributions, including those with a growing proportion of individuals at the lower or normal end of the BMI spectrum, face unique challenges in accurately identifying women at risk of GDM. Non-obese GDM may be underdiagnosed because of the widespread presumption that normal BMI confers inherent protection against insulin resistance and hyperglycemia. However, increased awareness that GDM is not exclusively an issue for overweight or obese pregnant women encourages more inclusive screening practices and targeted follow-ups. Studying the distinct characteristics of non-obese GDM women provides an opportunity to revise existing guidelines and improve care quality across different populations.

The present retrospective study aimed to compare maternal and fetal outcomes between non-obese and obese women diagnosed with GDM, focusing on the prevalence and severity of complications, such as preeclampsia, gestational HT, macrosomia, and neonatal hypoglycemia. By elucidating whether non-obese GDM individuals truly experience lower-risk profiles or are equally prone to adverse outcomes, this research could help shape future clinical guidelines and risk stratification

approaches. In turn, evidence-based management tailored to non-obese GDM patients may reduce unnecessary interventions for low-risk individuals and enable more precise therapeutic measures for high-risk cases.<sup>[12]</sup>

While the connection between obesity and GDM is well established, there is a growing need to recognize that gestational glucose intolerance also affects women with a normal BMI. Exploring the nuances of non-obese GDM can provide new insights into pathophysiology, risk assessment, and management of conditions that directly affect maternal and neonatal health. By examining population-based data in a hospital setting, this study hopes to provide valuable evidence that can guide personalized and effective care for pregnant women with GDM, regardless of their BMI status.<sup>[13]</sup>

## 2. Materials and methods

### 2.1. Study design and setting

This retrospective cohort study was conducted at the Perinatology Clinic of Haseki Training and Research Hospital affiliated with the University of Health Sciences (Istanbul). It analyzed patient records from 2018 to 2024. All medical data were retrieved from the hospital's electronic database. Participants included pregnant women diagnosed with GDM during the 24 to 28 weeks of gestation using a standard 75-g OGTT. All women completed their antenatal follow-up and delivered at the same institution, allowing for comprehensive retrieval of both prenatal and delivery outcomes.

Two groups were formed based on the pre-pregnancy BMI:

Non-obese GDM group: BMI < 25 kg/m<sup>2</sup>.

Obese GDM group: BMI ≥ 30 kg/m<sup>2</sup>.

Patients with BMIs between 25 and 29.9 kg/m<sup>2</sup> or lacking accurate BMI data were not included in this analysis.

Inclusion criteria included:

Singleton pregnancies.

Diagnosis of GDM confirmed by 75-g OGTT between 24 and 28 weeks of gestation.

Complete antenatal and delivery records accessible in the hospital database.

Age range of 18 to 45 years.

Exclusion criteria included:

Preexisting type 1 or type 2 diabetes mellitus.

Multiple gestations (e.g., twin or higher-order pregnancies).

Major fetal anomalies diagnosed antenatally.

Chronic HT or other major chronic diseases diagnosed prior to pregnancy.

Missing or incomplete medical records preventing adequate data collection.

### 2.2. Data collection

The electronic patient files were systematically reviewed by trained research staff. Relevant demographic, clinical, and laboratory parameters were extracted, including maternal age, gravidity, parity, pre-pregnancy weight, height, and BMI. Biochemical data, such as HbA1c levels and OGTT values, were also collected. Antenatal records were assessed for complications, including preeclampsia, gestational HT, mode of delivery (vaginal vs cesarean), and postpartum hemorrhage.

Fetal and neonatal outcomes were documented using delivery and neonatal notes, respectively. These outcomes included birth weight, incidence of macrosomia (>4000 g), IUGR, Apgar scores at 1 and 5 minutes, neonatal hypoglycemia (blood glucose < 40 mg/dL within the first few hours of life), and NICU admission.

### 2.3. Definitions and outcomes

GDM diagnosis was confirmed when 1 or more plasma glucose values exceeded the reference thresholds on the 75-g OGTT.

Non-obese versus obese: BMI < 25 kg/m<sup>2</sup> was considered non-obese, whereas BMI ≥ 30 kg/m<sup>2</sup> was considered obese.

Preeclampsia: new-onset HT (blood pressure ≥ 140/90 mm Hg) after 20 weeks of gestation accompanied by proteinuria or end-organ dysfunction.

Gestational hypertension: elevated blood pressure (≥140/90 mm Hg) after 20 weeks of gestation without proteinuria or other preeclamptic features.

Macrosomia: birth weight ≥ 4000 g.

IUGR: estimated fetal weight < 10th percentile for gestational age confirmed by ultrasound measurements and clinical assessments.

Neonatal hypoglycemia: capillary or venous blood glucose level < 40 mg/dL in the neonatal period.

NICU admission: admission to the NICU for any medical reason within the first 24 hour postpartum.

### 2.4. Statistical analysis

All data were compiled into a secure database and analyzed using a standard statistical software package (IBM SPSS Statistics, version 29; IBM Corp., Armonk). Continuous variables were examined for normality using descriptive statistics, histograms, and the Shapiro–Wilk test. Depending on the distribution, continuous data are presented as mean ± standard deviation or median (interquartile range). Comparisons between groups were performed using the independent samples *t* test or Mann–Whitney *U* test. Categorical variables are expressed as frequencies and percentages, and group comparisons were performed using the chi-square test or Fisher exact test, as appropriate.

Multivariable logistic regression analyses were conducted to identify the factors independently associated with adverse maternal and neonatal outcomes. The independent variables included maternal age, BMI category, HbA1c level, and relevant comorbidities. Results were reported as odds ratios (ORs) with 95% confidence intervals. Statistical significance was defined as a 2-sided *P*-value of < .05.

### 2.5. Ethical considerations

The study adhered to the institutional guidelines for patient privacy and confidentiality, ensuring that all information was anonymized. This research was conducted in accordance with the principles outlined in the Helsinki Declaration. The study protocol was approved by the Ethics Committee of our institution with registry number: 06-2025 on January 30, 2025. The requirement for informed consent was waived because of the retrospective nature of the investigation. Through this standardized process of data collection and analysis, this study aimed to provide a comprehensive comparison of maternal and neonatal outcomes between non-obese and obese women with GDM.

## 3. Results

In total, 400 pregnant women diagnosed with GDM met the inclusion criteria. Of these, 200 were categorized as non-obese (BMI < 25 kg/m<sup>2</sup>) and 200 as obese GDM (BMI ≥ 30 kg/m<sup>2</sup>). All participants completed antenatal care and were delivered at our center, allowing for the comprehensive retrieval of maternal and neonatal outcomes.

Table 1 compares the initial demographic and clinical attributes of the non-obese and obese GDM groups. By design, the obese group displays a significantly higher BMI (32.8 ± 2.5 kg/m<sup>2</sup>) compared to the non-obese group (23.2 ± 1.5 kg/m<sup>2</sup>; *P* < .001). Both groups were otherwise similar regarding maternal age, parity, and family history of diabetes, indicating that apart from obesity, they share comparable clinical backgrounds. The mean gestational age at GDM diagnosis was also very similar between the 2 groups (approximately 26 weeks), suggesting that the timing of GDM detection was not significantly affected by BMI status. These findings imply that the cohort is sufficiently balanced in most baseline variables, allowing for a focused assessment of how body mass index might impact subsequent maternal and neonatal outcomes.

Laboratory parameters, especially fasting plasma glucose and 1-hour and 2-hour OGTT values, were uniformly higher in the obese GDM group, signifying more pronounced glucose intolerance than in non-obese patients. The fasting glucose difference (*P* = .002) and the 1-hour/2-hour OGTT values (*P* < .05) highlighted a trend of poorer glycemic control associated with higher

**Table 1**

**Baseline demographic and clinical characteristics.**

| Characteristic                      | Non-obese GDM (n = 200) | Obese GDM (n = 200) | <i>P</i> -value |
|-------------------------------------|-------------------------|---------------------|-----------------|
| Age (years), mean ± SD              | 29.4 ± 4.2              | 30.1 ± 4.6          | .213            |
| BMI (kg/m <sup>2</sup> ), mean ± SD | 23.2 ± 1.5              | 32.8 ± 2.5          | <.001           |
| Gravidity ≥ 2, n (%)                | 80 (40.0)               | 86 (43.0)           | .528            |
| Family history of DM, n (%)         | 72 (36.0)               | 81 (40.5)           | .379            |
| GA at GDM diagnosis (wk)            | 25.9 ± 1.4              | 26.1 ± 1.3          | .678            |

Values are presented as mean ± standard deviation (SD) or number (percentage), unless otherwise specified.

BMI = body mass index, DM = diabetes mellitus, GA = gestational age, GDM = gestational diabetes mellitus.

**Table 2**

**Laboratory finding.**

| Parameter               | Non-obese GDM (n = 200) | Obese GDM (n = 200) | <i>P</i> -value |
|-------------------------|-------------------------|---------------------|-----------------|
| Fasting glucose (mg/dL) | 95.3 ± 8.2              | 98.6 ± 10.1         | .002            |
| 1-hour OGTT (mg/dL)     | 175.2 ± 20.5            | 181.3 ± 22.6        | .012            |
| 2-hour OGTT (mg/dL)     | 153.9 ± 15.7            | 159.8 ± 18.1        | .009            |
| HbA1c (%)               | 5.8 ± 0.6               | 6.1 ± 0.7           | <.001           |

Values are presented as mean ± standard deviation (SD).

GDM = gestational diabetes mellitus, HbA1c = glycated hemoglobin, OGTT = oral glucose tolerance test.

BMI. Notably, the mean HbA1c level was also significantly greater in obese GDM patients ( $6.1 \pm 0.7\%$  vs  $5.8 \pm 0.6\%$ ;  $P < .001$ ). These data collectively indicate that excess adiposity may exacerbate insulin resistance and deteriorate metabolic homeostasis, underscoring the importance of more aggressive monitoring and possible early interventions in obese GDM patients (Table 2).

In the Figure 1, the incidence of key maternal complications, preeclampsia, gestational hy HT pertension, cesarean delivery, and postpartum hemorrhage, is illustrated side by side for the non-obese and obese GDM groups. The chart shows noticeably higher percentages in the obese GDM cohort, reflecting how elevated BMI and greater insulin resistance correlate with an increased risk of these complications. In particular, preeclampsia and cesarean delivery percentages stand out, suggesting that obesity can exacerbate the underlying pathophysiological processes, leading to adverse maternal outcomes.

Several critical maternal outcomes have revealed how obesity intensifies complications (Table 3). The obese GDM group exhibited a higher prevalence of preeclampsia (16.0% vs 7.0%,  $P = .005$ ) and a significantly higher rate of cesarean deliveries (57.0% vs 39.0%,  $P < .001$ ). Although the difference in gestational HT between the 2 cohorts did not reach statistical significance, there was a suggestive trend for more hypertensive disorders in the obese group. Postpartum hemorrhage was also

more frequent among the obese GDM participants (8.0% vs 4.0%;  $P = .049$ ). Moreover, the need for insulin therapy was markedly higher in the obese group, indicating that obesity likely compounds insulin resistance to the point where dietary management and exercise alone are insufficient to control glycemia in a substantial subset of patients.

Figure 2 compares the macrosomia and IUGR rates between the 2 groups. Obese GDM patients exhibit a higher prevalence of macrosomia, demonstrating the strong influence of maternal obesity and suboptimal glycemic control on fetal overgrowth. IUGR rates, however, show negligible differences, implying that while obesity prominently raises the likelihood of excessive fetal growth, it does not necessarily rule out other growth-related complications that may arise in any GDM setting.

In terms of neonatal findings, obese GDM mothers had a markedly higher mean birth weight ( $3490 \pm 500$  g vs  $3350 \pm 420$  g;  $P = .001$ ) and an increased incidence of macrosomia (16.0% vs 7.0%;  $P = .003$ ) (Table 4). Interestingly, the rate of IUGR did not differ significantly between the 2 groups ( $P = .655$ ), suggesting that while obesity elevates the risk of fetal overgrowth, it does not necessarily protect against growth restriction in all cases. Notably, neonatal hypoglycemia and the need for NICU admission were considerably more common among newborns of obese GDM mothers ( $P < .05$ ), indicating that higher maternal BMI and suboptimal glycemic control may predispose infants to postnatal complications that warrant intensive medical care. In the Figure 3, the birth weights are displayed for the non-obese and obese GDM groups. The median birth weight and overall distribution were shifted upward in the obese GDM cohort, consistent with increased macrosomia rates. The wider interquartile range in the obese group also indicated more variability, possibly reflecting heterogeneous glucose control and other metabolic factors within this population. Overall, this figure underscores how maternal obesity can significantly skew birth weight toward higher values.

Table 5 presents the adjusted odds ratios for variables predicting preeclampsia. Being in the obese GDM group emerged as an independent risk factor, nearly doubling the odds of preeclampsia (OR, 2.11;  $P = .039$ ). Elevated HbA1c levels also significantly increased preeclampsia risk (OR: 1.72 per 1% increase;  $P = .008$ ), underscoring the importance of stringent glycemic management to reduce hypertensive complications. Interestingly, maternal age and a family history of diabetes did not show a statistically significant effect in this model. These findings highlight how obesity and chronic hyperglycemia may converge to drive adverse vascular outcomes that are central to the pathophysiology of preeclampsia.

Focusing on macrosomia, the analysis identified 2 key factors independently boosting its likelihood: belonging to the obese GDM group (OR, 2.58;  $P = .007$ ) and having a higher HbA1c level (OR: 1.59 per 1% increase;  $P = .013$ ) (Table 6). This aligns well with the notion that excessive adiposity and chronic hyperglycemia foster an environment conducive to fetal overgrowth. Other variables, such as maternal age and family history of diabetes, demonstrated weak or non-significant associations. Consequently, these findings underscore that women with both

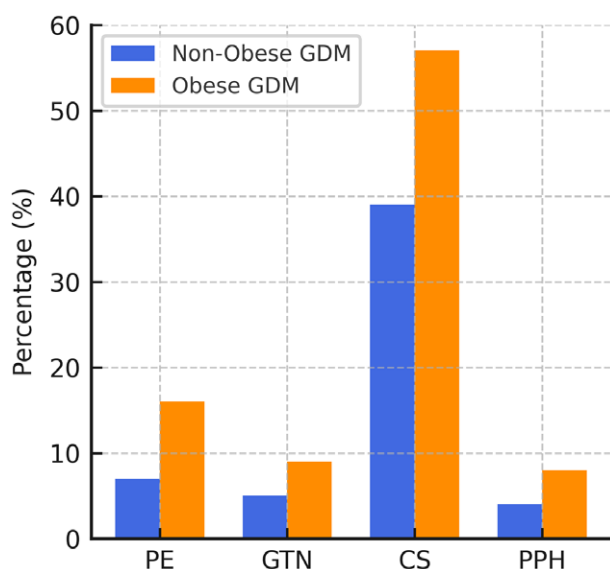


**Figure 1.** Maternal complications in women with gestational diabetes mellitus (GDM) stratified by obesity status. Blue bars represent the non-obese GDM group and orange bars represent the obese GDM group. The prevalence of preeclampsia (PE), gestational hypertension (GTN), cesarean section (CS), and postpartum hemorrhage (PPH) is depicted as percentages. Obese women with GDM exhibited consistently higher rates of adverse maternal outcomes compared with their non-obese counterparts, most notably in cesarean delivery.

**Table 3**  
Maternal pregnancy outcomes.

| Outcome                      | Non-obese GDM (n = 200) | Obese GDM (n = 200) | P-value |
|------------------------------|-------------------------|---------------------|---------|
| Preeclampsia, n (%)          | 14 (7.0)                | 32 (16.0)           | .005    |
| Gestational HTN, n (%)       | 10 (5.0)                | 18 (9.0)            | .107    |
| Cesarean delivery, n (%)     | 78 (39.0)               | 114 (57.0)          | <.001   |
| Postpartum hemorrhage, n (%) | 8 (4.0)                 | 16 (8.0)            | .049    |
| Insulin therapy, n (%)       | 38 (19.0)               | 60 (30.0)           | .014    |

Values are presented as number (percentage).

GDM = gestational diabetes mellitus, HTN = hypertension.

obesity and elevated glycemic indices during pregnancy should be monitored closely for potential fetal overnutrition. The receiver operating characteristic curve assesses the predictive accuracy of a multivariable model (or score) for macrosomia (Fig. 4). The curve plots the true positive rate against the false positive rate for different thresholds, with the area under the curve quantifying discriminative power. A higher area under the curve suggests a better predictive ability. In scenarios where BMI and other metabolic indicators, such as HbA1c, are incorporated, the model typically demonstrates moderate to good performance, emphasizing the importance of these factors in identifying pregnancies at high risk for fetal overgrowth. Table 7 details how each group managed GDM. While dietary intervention alone is often sufficient for more than half of non-obese GDM patients (53.0%), a substantial fraction of obese GDM participants require either insulin (20.0%) or oral anti-hyperglycemic agents (10.0%). The disparity in treatment approaches points to the greater severity or complexity of glycemic dysregulation in obese women with GDM. Interestingly, while diet plus exercise was relatively popular in both cohorts, the obese GDM group still had a higher likelihood of escalating treatment beyond lifestyle modifications. This pattern

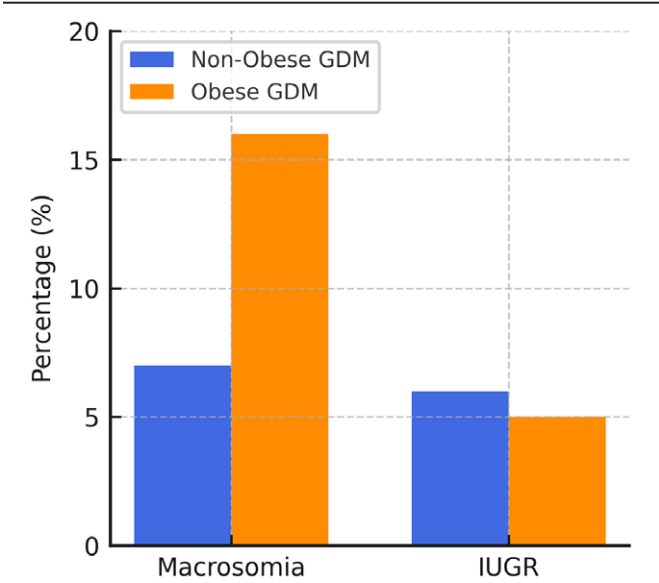


**Figure 2.** Comparison of neonatal outcomes. The figure compares the macrosomia and intrauterine growth restriction (IUGR) rates between the 2 groups. Obese gestational diabetes mellitus (GDM) patients exhibit a higher prevalence of macrosomia, demonstrating the strong influence of maternal obesity and suboptimal glycemic control on fetal overgrowth. IUGR rates, however, show negligible differences, implying that while obesity prominently raises the likelihood of excessive fetal growth, it does not necessarily rule out other growth-related complications that may arise in any GDM setting.

underscores how BMI can profoundly influence the clinical course and management choices for GDM. Table 8 connects treatment modalities with maternal and neonatal outcomes. Patients who received pharmacological interventions, such as insulin or oral agents, generally displayed higher rates of preeclampsia and macrosomia, along with increased NICU admissions, compared to the diet-only subgroup. Specifically, the insulin-treated group exhibited a significantly elevated risk of adverse outcomes (e.g., preeclampsia at 13.0% vs only 3.6% in diet-only cases), suggesting that pharmacotherapy can be a proxy for more severe disease. Importantly, the higher mean birth weights in these groups align with the idea that intensified treatment correlates with worse baseline metabolic indicators, further reinforcing the need for vigilant follow-up and precise therapeutic adjustments in high-risk pregnancies.

4. Discussion

This retrospective cohort analysis highlights how GDM manifests distinct clinical trajectories in non-obese versus obese pregnant women, underscoring the influence of BMI and metabolic control on maternal and neonatal outcomes. Overall, our data confirm that while obesity consistently amplifies adverse events, non-obese women diagnosed with GDM cannot be overlooked as they face significant risks, albeit at a lower frequency.<sup>[14]</sup> One of the central findings pertains to maternal outcomes such as preeclampsia, gestational HT, and cesarean deliveries. Our results showed significantly higher rates of preeclampsia and cesarean deliveries in the obese GDM group, reflecting the accentuated pathophysiological burden associated with obesity. These complications may stem from a convergence of pro-inflammatory pathways, endothelial dysfunction, and heightened insulin resistance in obese individuals. Although non-obese GDM patients generally experience milder courses, they remain susceptible to complications if their glycemic control deteriorates, highlighting the multifactorial nature of GDM.<sup>[15]</sup> The neonatal outcomes mirrored these patterns. Notably, the birth weight and macrosomia rates substantially increased in the obese GDM group. This finding is consistent with the concept that elevated maternal adiposity coupled with inadequate glycemic regulation fuels excessive fetal glucose transfer, leading to increased fetal insulin secretion and adipose deposition. However, it is important to acknowledge that non-obese GDM mothers also deliver macrosomic infants, although at a lower rate, signifying that BMI alone does not safeguard against fetal overgrowth when maternal hyperglycemia is present. Additionally, we noted that neonatal hypoglycemia and NICU admissions were higher in the obese GDM group, possibly reflecting both metabolic instability at delivery and altered fetal insulin responses to prolonged intrauterine hyperglycemia. Our multivariate analyses further clarified the impact of obesity and glycemic levels. The obese GDM category proved to be an independent risk factor for both preeclampsia and

**Table 4**  
Neonatal outcomes.

| Outcome                      | Non-obese GDM (n = 200) | Obese GDM (n = 200) | P-value |
|------------------------------|-------------------------|---------------------|---------|
| Birth weight (g), mean ± SD  | 3350 ± 420              | 3490 ± 500          | .001    |
| Macrosomia (>4000 g), n (%)  | 14 (7.0)                | 32 (16.0)           | .003    |
| IUGR, n (%)                  | 12 (6.0)                | 10 (5.0)            | .655    |
| Apgar < 7 at 5 min, n (%)    | 6 (3.0)                 | 12 (6.0)            | .141    |
| NICU admission, n (%)        | 10 (5.0)                | 26 (13.0)           | .004    |
| Neonatal hypoglycemia, n (%) | 18 (9.0)                | 30 (15.0)           | .045    |

Values are presented as mean ± standard deviation (SD) or number (percentage), as appropriate.  
GDM = gestational diabetes mellitus, IUGR = intrauterine growth restriction, NICU = neonatal intensive care unit.

macrosomia, paralleling other studies suggesting that greater adiposity intensifies pregnancy-related risks. Likewise, higher HbA1c levels were significantly correlated with these outcomes, indicating suboptimal glycemic control as a pivotal determinant. These findings reinforce the idea that maternal glucose levels must be diligently managed across the entire BMI spectrum to mitigate adverse outcomes.<sup>[16]</sup>

In terms of management strategies, obese GDM mothers frequently require pharmacological interventions, such as insulin or oral agents, rather than diet and exercise alone. This observation underscores the greater severity of insulin resistance and impaired glucose tolerance associated with a high BMI. Interestingly, those receiving insulin or oral agents also

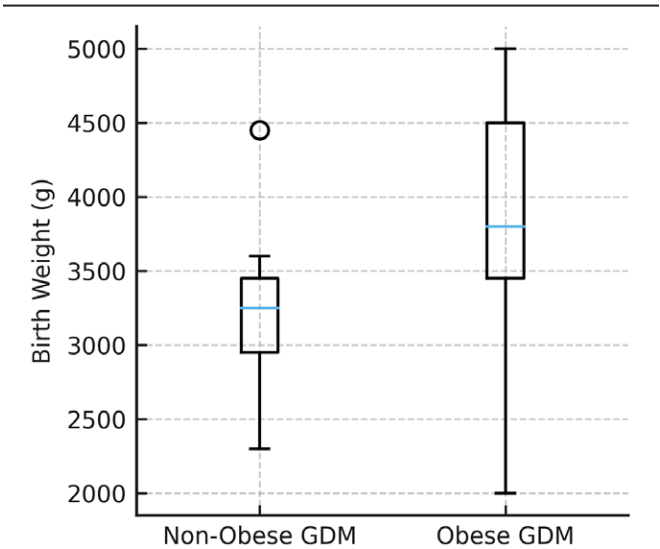


**Figure 3.** Birth weight distribution by group. The birth weights are displayed for the non-obese and obese gestational diabetes mellitus (GDM) groups. The median birth weight and overall distribution were shifted upward in the obese GDM cohort, consistent with increased macrosomia rates. The wider interquartile range in the obese group also indicated more variability, possibly reflecting heterogeneous glucose control and other metabolic factors within this population.

experienced increased complications, suggesting that the need for pharmacotherapy can be an indicator of a higher-risk clinical profile. However, further prospective evaluations are needed to determine whether pharmacological therapy itself influences outcomes or simply reflects the underlying severity of GDM that predisposes to complications.<sup>[17]</sup>

From a broader perspective, our results have several clinical implications. First, healthcare providers must recognize that GDM in non-obese women, although generally less severe, is not benign; timely screening, individualized monitoring, and interventions remain critical. Second, in obese GDM patients, stricter glycemic targets, closer surveillance for hypertensive disorders, and consideration of early pharmacotherapy may be warranted to prevent adverse events. Third, our findings call for integrated care strategies that address lifestyle and nutritional counseling, not just for overweight or obese women but for all pregnant women at risk of GDM. Although obesity increases the burden of GDM, non-obese individuals require equal vigilance regarding glucose monitoring and perinatal follow-up.

Despite the strengths of our dataset, this study had inherent limitations. Being retrospective, unmeasured confounders, such as dietary habits, exact timing of glycemic control measures, and genetic predispositions, could influence outcomes. Additionally, the data stems from a single institution, potentially limiting external generalizability. Future prospective and multicenter studies examining non-obese GDM in diverse settings will help validate and deepen our understanding of these findings. Moreover, mechanistic research into insulin sensitivity pathways among non-obese women with GDM may reveal novel approaches for early detection and targeted treatment.<sup>[18]</sup>

In conclusion, this study demonstrates how maternal obesity significantly exacerbates both maternal and fetal risks in GDM and confirms that non-obese women with GDM remain susceptible to complications. Tailored clinical strategies, including precise glycemic surveillance, judicious use of pharmacotherapy, and comprehensive management of comorbidities, are crucial for mitigating adverse outcomes. Ultimately, these insights can guide the design of more nuanced guidelines, ensuring that all women with GDM, regardless of their BMI, receive appropriate care to optimize pregnancy and neonatal outcomes.<sup>[19]</sup>

**Table 5**  
**Multivariable logistic regression for preeclampsia.**

| Variable                           | Adjusted OR (95% CI) | P-value |
|------------------------------------|----------------------|---------|
| BMI category (obese vs non-obese)  | 2.11 (1.05–4.22)     | .039    |
| Maternal age (per 1-year increase) | 1.04 (0.97–1.10)     | .236    |
| Family history of DM               | 1.32 (0.68–2.01)     | .144    |
| Gravidity ≥ 2                      | 1.13 (0.62–1.84)     | .312    |
| HbA1c (per 1% increase)            | 1.72 (1.14–2.28)     | .008    |

Values are presented as adjusted odds ratios (OR) with 95% confidence intervals (CI) derived from multivariable logistic regression analysis.  
BMI = body mass index, DM = diabetes mellitus, HbA1c = glycated hemoglobin.

**Table 6**  
**Multivariable logistic regression for macrosomia.**

| Variable                           | Adjusted OR (95% CI) | P-value |
|------------------------------------|----------------------|---------|
| BMI category (obese vs non-obese)  | 2.58 (1.19–3.80)     | .007    |
| Maternal age (per 1-year increase) | 1.03 (0.97–1.09)     | .261    |
| Family history of DM               | 1.45 (0.78–2.10)     | .089    |
| Gravidity ≥ 2                      | 1.05 (0.65–1.69)     | .512    |
| HbA1c (per 1% increase)            | 1.59 (1.22–2.10)     | .013    |

Values are presented as adjusted odds ratios (OR) with 95% confidence intervals (CI) obtained from multivariable logistic regression analysis.  
BMI = body mass index, DM = diabetes mellitus, HbA1c = glycated hemoglobin.

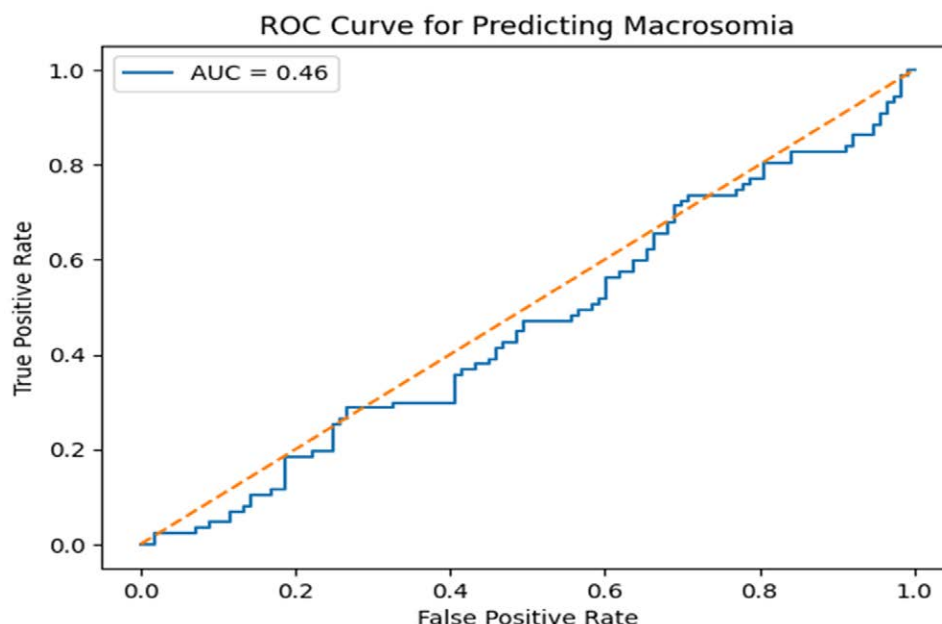


**Figure 4.** ROC curve for predicting macrosomia. The ROC curve assesses the predictive accuracy of a multivariable model (or score) for macrosomia. The curve plots the true positive rate against the false positive rate for different thresholds, with the area under the curve (AUC) quantifying discriminative power. A higher AUC suggests a better predictive ability. In scenarios where BMI and other metabolic indicators, such as HbA1c, are incorporated, the model typically demonstrates moderate to good performance, emphasizing the importance of these factors in identifying pregnancies at high risk for fetal overgrowth. BMI = body mass index, HbA1c = glycated hemoglobin, ROC = receiver operating characteristic.

**Table 7**

#### Glycemic management strategies.

| Treatment                 | Non-obese GDM (n = 200) | Obese GDM (n = 200) | P-value |
|---------------------------|-------------------------|---------------------|---------|
| Diet only, n (%)          | 106 (53.0)              | 60 (30.0)           | <.001   |
| Diet + exercise, n (%)    | 72 (36.0)               | 80 (40.0)           | .129    |
| Diet + insulin, n (%)     | 14 (7.0)                | 40 (20.0)           | <.001   |
| Diet + oral agents, n (%) | 8 (4.0)                 | 20 (10.0)           | .015    |

Values are presented as number (percentage).

GDM = gestational diabetes mellitus.

**Table 8**

#### Maternal and neonatal outcomes by treatment type.

| Outcome               | Diet only (n = 166) | Diet + insulin (n = 54) | Diet + oral agents (n = 28) | P-value |
|-----------------------|---------------------|-------------------------|-----------------------------|---------|
| Preeclampsia, n (%)   | 6 (3.6)             | 7 (13.0)                | 3 (10.7)                    | .019    |
| Macrosomia, n (%)     | 10 (6.0)            | 9 (16.7)                | 4 (14.3)                    | .035    |
| NICU admission, n (%) | 6 (3.6)             | 8 (14.8)                | 2 (7.1)                     | .027    |
| Mean birth weight (g) | 3350 ± 300          | 3510 ± 320              | 3450 ± 380                  | .008    |

Values are presented as mean ± standard deviation (SD) or number (percentage), as appropriate.

NICU = neonatal intensive care unit.

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#### References

- [1] Bashir M, Fagier Y, Ahmed B, Konje JC. An overview of diabetes mellitus in pregnant women with obesity. *Best Pract Res Clin Obstet Gynaecol.* 2024;93:102469.
- [2] Weir TL, Majumder M, Glastras SJ. A systematic review of the effects of maternal obesity on neonatal outcomes in women with gestational diabetes. *Obes Rev.* 2024;25:e13747.
- [3] Zehravi M, Maqbool M, Ara I. Correlation between obesity, gestational diabetes mellitus, and pregnancy outcomes: an overview. *Int J Adolesc Med Health.* 2021;33:339–45.

- [4] Simmons D. Diabetes and obesity in pregnancy. *Best Pract Res Clin Obstet Gynaecol.* 2011;25:25–36.
- [5] Moore TR. Fetal exposure to gestational diabetes contributes to subsequent adult metabolic syndrome. *Am J Obstet Gynecol.* 2010;202:643–9.
- [6] Buchanan TA, Xiang AH, Page KA. Gestational diabetes mellitus: risks and management during and after pregnancy. *Nat Rev Endocrinol.* 2012;8:639–49.
- [7] Vohr BR, Boney CM. Gestational diabetes: the forerunner for the development of maternal and childhood obesity and metabolic syndrome?. *J Matern Fetal Neonatal Med.* 2008;21:149–57.
- [8] Ijäs H, Koivunen S, Raudaskoski T, Kajantie E, Gissler M, Väärasmäki M. Independent and concomitant associations of gestational diabetes and maternal obesity to perinatal outcome: a register-based study. *PLoS One.* 2019;14:e0221549.
- [9] Miao M, Dai M, Zhang Y, Sun F, Guo X, Sun G. Influence of maternal overweight, obesity and gestational weight gain on the perinatal outcomes in women with gestational diabetes mellitus. *Sci Rep.* 2017;7:305.
- [10] Alfadhli EM. Maternal obesity influences birth weight more than gestational diabetes. *BMC Pregnancy Childbirth.* 2021;21:111.
- [11] Sathyapalan T, Mellor D, Atkin SL. Obesity and gestational diabetes. *İçinde Seminars in Fetal and Neonatal Medicine.* Elsevier. 2010:89–3
- [12] Bo S, Menato G, Signorile A, et al. Obesity or diabetes: what is worse for the mother and for the baby?. *Diabetes Metab.* 2003;29:175–8.
- [13] Yogev Y, Visser GH. Obesity, gestational diabetes and pregnancy outcome *İçinde Seminars in Fetal and Neonatal Medicine.* Elsevier. 2009:77–4.
- [14] Yogev Y, Langer O. Pregnancy outcome in obese and morbidly obese gestational diabetic women. *Eur J Obstet Gynecol Reprod Biol.* 2008;137:21–6.
- [15] Roman AS, Rebarber A, Fox NS, et al. The effect of maternal obesity on pregnancy outcomes in women with gestational diabetes. *J Matern Fetal Neonatal Med.* 2011;24:723–7.
- [16] Reece EA. The fetal and maternal consequences of gestational diabetes mellitus. *J Matern Fetal Neonatal Med.* 2010;23:199–203.
- [17] Catalano PM. The impact of gestational diabetes and maternal obesity on the mother and her offspring. *J Dev Orig Health Dis.* 2010;1:208–15.
- [18] Wahabi HA, Fayed AA, Alzeidan RA, Mandil AA. The independent effects of maternal obesity and gestational diabetes on the pregnancy outcomes. *BMC Endocr Disord.* 2014;14:47.
- [19] Dennedy MC, Dunne F. The maternal and fetal impacts of obesity and gestational diabetes on pregnancy outcome. *Best Pract Res Clin Endocrinol Metab.* 2010;24:573–89.