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Gestational diabetes mellitus and neonatal complications: Macrosomia, neonatal hypoglycemia and shoulder dystocia

Nayane Alves Ferreira *, Lindomar Gomes Lacerda Junior, Igor Diniz Sato, Jeniffer Aparecida de Morais Rodrigues and Marinaldo Soares Leite

Centro Universitário Alfredo Nasser.

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Abstract

Gestational diabetes mellitus (GDM) is a common metabolic disorder of pregnancy associated with important neonatal complications. The primary adverse outcomes include macrosomia, neonatal hypoglycemia and shoulder dystocia, all of which are closely related to maternal hyperglycemia and fetal hyperinsulinism. This narrative review evaluates recent evidence published between 2020 and 2025. Studies consistently demonstrate that GDM increases the risk of macrosomia by two- to five-fold (AMERICAN DIABETES ASSOCIATION, 2024), predisposes neonates to early hypoglycemia due to persistent hyperinsulinism (RODRIGUEZ; CHEN; BRACKEN, 2024), and contributes to shoulder dystocia secondary to excessive fetal growth (ACOG, 2018). Early diagnosis, strict glycemic control and individualized delivery planning remain essential for reducing neonatal morbidity.

Keywords: Gestational Diabetes; Macrosomia; Neonatal Hypoglycemia; Shoulder Dystocia; Neonatal Outcomes

1. Introduction

Gestational diabetes mellitus (GDM) is defined as glucose intolerance first identified during pregnancy. Its global prevalence ranges from 7% to 18%, depending on diagnostic methods and population characteristics (AMERICAN DIABETES ASSOCIATION, 2024). The rising incidence reflects increased maternal obesity and delayed childbearing.

The most relevant neonatal complications—macrosomia, neonatal hypoglycemia and shoulder dystocia—share a common pathophysiological pathway driven by maternal hyperglycemia. According to the Pedersen hypothesis, maternal glucose crosses the placenta freely, leading to fetal hyperglycemia and compensatory hyperinsulinism (RODRIGUEZ; CHEN; BRACKEN, 2024). Insulin functions as a fetal anabolic hormone, promoting accelerated somatic growth and fat deposition (NAKSHINE et al., 2023).

Current guidelines from the American Diabetes Association (2024) and the American College of Obstetricians and Gynecologists (2018) highlight the need for early screening, tight glycemic management and individualized obstetric decision-making to mitigate neonatal risks.

2. Methodology

This study is a narrative review. Searches were performed in PubMed, Scopus and Google Scholar using the descriptors gestational diabetes mellitus, macrosomia, neonatal hypoglycemia, shoulder dystocia, and neonatal outcomes. Articles published between 2020 and 2025 were included. Inclusion criteria: Full-text articles in English (2020–2025);

* Corresponding author: Nayane Alves Ferreira

Systematic reviews, meta-analyses, cohort studies and clinical guidelines; Studies evaluating neonatal outcomes in GDM. Exclusion criteria: Studies on pregestational diabetes only; Case reports; Studies lacking neonatal outcome information.

Priority was given to high-quality evidence, including ADA and ACOG guidelines, cohort studies and systematic reviews.

3. Discussion

3.1. Pathophysiology

Maternal hyperglycemia results in increased transplacental glucose transport to the fetus, stimulating pancreatic β-cell hyperplasia and hyperinsulinism (RODRIGUEZ; CHEN; BRACKEN, 2024). Insulin drives increased adipose tissue deposition and somatic overgrowth, explaining the relationship between GDM, macrosomia and delivery complications (NAKSHINE et al., 2023). After delivery, persistent fetal hyperinsulinism coupled with abrupt interruption of maternal glucose leads to neonatal hypoglycemia (AL BEKAI et al., 2025).

3.2. Macrosomia

Macrosomia is defined as birth weight ≥4,000 g or ≥4,500 g. According to the ADA (2024), GDM increases the likelihood of macrosomia two- to five-fold. A recent review also reported increased rates of large-for-gestational-age infants, especially in women with poor postprandial glycemic control (SOUSA et al., 2024).

Clinical implications include higher rates of cesarean delivery, birth trauma and postpartum complications (PUNNOSE et al., 2025).

3.3. Neonatal Hypoglycemia

Neonatal hypoglycemia is the most common metabolic complication in infants of mothers with GDM. Systematic reviews report a three- to twelve-fold increased risk (NASHIF et al., 2023). Persistent fetal hyperinsulinism accounts for the rapid drop in postnatal glucose (ABURISHEH et al., 2024).

Symptoms may include tremors, respiratory distress, lethargy or seizures. Early breastfeeding and glucose monitoring are essential (AMERICAN DIABETES ASSOCIATION, 2024).

3.4. Shoulder Dystocia

Shoulder dystocia results from impaction of the fetal shoulders after the head has been delivered. Its risk increases significantly in pregnancies complicated by GDM due to higher incidence of macrosomia (ACOG, 2018). The likelihood of this complication is particularly elevated when estimated fetal weight exceeds 4,500 g (ATHANASIADOU et al., 2025).

Associated neonatal injuries include brachial plexus palsy and clavicular fracture.

3.5. Impact of Glycemic Control

Tight glycemic control is the main modifiable factor for reducing macrosomia and neonatal hypoglycemia (AMERICAN DIABETES ASSOCIATION, 2024). Continuous glucose monitoring (CGM) improves glycemic stability and reduces neonatal complications (ABURISHEH et al., 2024).

Insulin remains the gold standard for pharmacologic treatment. Metformin, although increasingly used, requires further long-term safety evaluation (PUNNOSE et al., 2025).

4. Conclusion

Gestational diabetes mellitus significantly increases the risk of macrosomia, neonatal hypoglycemia and shoulder dystocia. These outcomes arise from a shared mechanism involving maternal hyperglycemia and fetal hyperinsulinism. Evidence highlights the need for early diagnosis, glycemic optimization and appropriate delivery planning to reduce neonatal morbidity. Preventing excessive fetal growth and ensuring rapid detection of neonatal hypoglycemia may reduce long-term metabolic complications in childhood.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

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