RESEARCH PAPER

Calcium–Magnesium Imbalance In Gestational Diabetes Mellitus: An Exploratory Assessment Using Albumin-Corrected Calcium And The Calcium–Magnesium Ratio

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Abstract

Background: Gestational diabetes mellitus (gdm) is a condition of metabolic dysregulation that extends well beyond hyperglycemia. Disruptions in divalent cation homeostasis—particularly involving calcium and magnesium—may represent upstream metabolic disturbances that precede and potentiate insulin resistance during pregnancy. Both minerals are integral to insulin secretion and insulin receptor signaling, yet their interdependent regulation as a ratio has received limited systematic evaluation in gdm. Furthermore, pregnancy-induced hypoalbuminemia confounds total serum calcium measurements, and most prior studies have not applied albumin correction when assessing calcium–magnesium balance.

Methods: This hospital-based cross-sectional study enrolled 50 pregnant women diagnosed with gdm at 24–28 weeks of gestation. Gdm was diagnosed using the dipsi single-step, non-fasting 75 g oral glucose challenge (2-hour venous plasma glucose ≥140 mg/dl). Serum magnesium, total calcium, and albumin were measured using validated automated colorimetric methods. Albumin-corrected calcium was calculated as: corrected ca = measured ca + 0.8 × [4.0 − serum albumin (g/dl)].

Results: Mean serum magnesium was 1.35 ± 0.18 mg/dl (range: 1.04–1.78). Mean albumin-corrected calcium was 8.96 ± 0.41 mg/dl. The ca/mg ratio demonstrated a mean of 6.75 ± 0.95 (range: 4.58–8.69). No statistically significant correlation was observed between the ratio and dipsi 2-hour plasma glucose ($r = -0.060$, $p = 0.680$).

Conclusion: Women with gdm demonstrate considerable inter-individual variability in calcium–magnesium balance, with serum magnesium trending toward the lower boundary of normal reference values. Mineral dysregulation may reflect chronic upstream metabolic perturbations rather than acute hyperglycemia.

Keywords: Gestational Diabetes Mellitus, Albumin-Corrected Calcium, Magnesium, Calcium–Magnesium Ratio, Insulin Resistance, Mineral Homeostasis, Dipsi Criteria, Pregnancy.

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1. INTRODUCTION

Gestational diabetes mellitus (GDM) is defined as glucose intolerance with onset or first recognition during pregnancy and affects an estimated 3–25% of pregnancies globally.[1] Although hyperglycemia constitutes the defining diagnostic criterion, GDM represents a multifaceted metabolic disturbance encompassing impaired insulin secretion, peripheral insulin resistance, altered adipokine signaling, and chronic low-grade inflammation.[2] Recognizing upstream biochemical events that precede or amplify insulin resistance is central to a comprehensive understanding of GDM pathophysiology.

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Among the metabolic perturbations implicated in GDM, disruption of divalent cation homeostasis—particularly involving calcium and magnesium—has emerged as an important, yet underexplored, upstream pathway. These minerals are mechanistically linked to insulin secretion, insulin receptor function, and intracellular glucose metabolism through distinct but interacting cascades.[3] Magnesium is an obligate cofactor for more than 300 enzymatic reactions, including those governing glucose transporter translocation, tyrosine kinase activity at insulin receptors, adenosine triphosphate-dependent phosphorylation, and rate-limiting enzymes of glycolysis.[3] Reduced intracellular magnesium availability impairs insulin receptor substrate-1 phosphorylation and diminishes GLUT-4 membrane insertion, thereby contributing to peripheral insulin resistance.[4] Hypomagnesemia has been reported in women with GDM across multiple populations, and urinary magnesium wasting induced by osmotic diuresis from hyperglycemia may perpetuate a bidirectional cycle of mineral depletion and metabolic dysregulation.[5]

Calcium plays a pivotal role in pancreatic β-cell insulin secretion. Glucose-stimulated membrane depolarization triggers calcium influx through voltage-dependent L-type calcium channels in β-cells, initiating insulin granule exocytosis.[6] Beyond the β-cell, intracellular calcium serves as a second messenger in muscle and adipose tissue, modulating glycogen synthesis, mitochondrial oxidative metabolism, and insulin-responsive signaling networks.[7]

Calcium and magnesium are physiological antagonists that reciprocally regulate each other at multiple cellular sites. Magnesium competitively inhibits calcium entry through voltage-gated channels, modulates plasma membrane Ca^{2+}-ATPase activity, and regulates intracellular calcium gradients.[8] Conversely, elevated intracellular calcium can displace magnesium from ATP-binding sites and attenuate magnesium-dependent kinase activity.[9] This functional antagonism implies that a ratio-based approach may more accurately reflect the physiological balance between these competing ions.

An important methodological consideration is the confounding effect of physiological hypoalbuminemia on total serum calcium measurements. Serum albumin concentrations decline by approximately 10–15 g/L during normal gestation, resulting in a reduction of approximately 0.5–1.0 mg/dL in total serum calcium without any change in ionized calcium.[10] Despite this, many published studies have reported uncorrected total calcium values, potentially introducing significant measurement error.[5,11,12]

The downstream consequences of calcium–magnesium imbalance and the resulting insulin resistance in GDM may include perturbations in inflammatory cytokines such as IL-6 and TNF-α, and dysregulation of microRNAs such as miR-29a and miR-132—all of which have been reported in the literature as molecular associates of insulin resistance and metabolic inflammation in pregnancy, although these biomarkers were not measured in the present study.[13,14]

The present investigation evaluates the calcium–magnesium ratio—calculated using albumin-corrected calcium values—as a marker of integrated mineral homeostasis in women with GDM. The primary objective of this study was to characterize the albumin-corrected calcium–magnesium ratio in women diagnosed with GDM at 24–28 weeks of gestation, and to examine its relationship with acute glycemic status as assessed by DIPSI 2-hour venous plasma glucose concentration.

2. MATERIALS AND METHODS

3.1 Study Design and Setting

A hospital-based cross-sectional study was conducted at the Departments of Biochemistry and Obstetrics and Gynecology, Mahatma Gandhi Medical College and Research Institute, Sri Balaji Vidyapeeth (Deemed-to-be University), Puducherry, India. Institutional Human Ethics Committee approval was obtained (Project No: MGMCRI/2024/02/IHEC/76; 09 November 2024). The study complied with the Declaration of Helsinki.

3.2 Study Population and Sampling

Fifty consecutive pregnant women diagnosed with GDM at 24–28 weeks of gestation were enrolled. This sample was appropriate for an exploratory descriptive investigation; formal power calculation was not performed.

3.3 Eligibility Criteria

Inclusion: Pregnant women at 24–28 completed weeks of gestation with confirmed GDM (DIPSI criteria). Exclusion: Pre-existing diabetes mellitus, chronic kidney disease, hypo/hyperparathyroidism, malabsorption syndromes, or current calcium or magnesium supplementation beyond standard prenatal multivitamins.

3.4 Diagnosis of GDM (DIPSI Criteria)

GDM was diagnosed using the Diabetes in Pregnancy Study Group India (DIPSI) single-step non-fasting oral glucose challenge test, as recommended by the Ministry of Health and Family Welfare, Government of India.[15] All participants received an oral glucose load

of 75 g anhydrous glucose dissolved in 300 mL water. Venous blood was collected exactly 2 hours post-ingestion and plasma glucose was estimated by the glucose oxidase–peroxidase method. A 2-hour venous plasma glucose ≥140 mg/dL was diagnostic of GDM.[15]

3.5 Biochemical Analysis

Serum magnesium was estimated by the calmagite colorimetric method. The pregnancy reference range for serum magnesium is 1.7–2.2 mg/dL.[16] Total serum calcium was estimated by o-cresolphthalein complexone (OCPC). Serum albumin was quantified by bromocresol green (BCG). Plasma glucose was estimated by GOD-POD. Internal quality control was maintained using two-level control materials; coefficient of variation was <5% within-run and <7% between-run.

3.6 Calculation of Albumin-Corrected Calcium and Ca/Mg Ratio

Total serum calcium was corrected for albumin using the established Payne correction formula:[10,17]

Corrected Calcium (mg/dL) = Measured Total Calcium + 0.8 × [4.0 − Serum Albumin (g/dL)]

The calcium–magnesium ratio was calculated as:

Ca/Mg Ratio = Albumin-Corrected Calcium (mg/dL) ÷ Serum Magnesium (mg/dL)

3.7 Statistical Analysis

IBM SPSS Statistics version 25.0 was used. Normality was assessed by the Shapiro–Wilk test. Continuous variables are expressed as mean ± SD with range. Pearson's correlation coefficient (r) assessed the relationship between the Ca/Mg ratio and DIPSI 2-hour plasma glucose. Statistical significance was set at $p < 0.05$ (two-tailed).

3. RESULTS

4.1 Participant Characteristics

Fifty pregnant women with GDM were enrolled. Mean age was 27.8 ± 3.4 years (range: 22–35 years). Mean gestational age at enrollment was 25.6 ± 1.2 weeks (range: 24–28 weeks). Mean DIPSI 2-hour venous plasma glucose was 168.4 ± 22.6 mg/dL (range: 142–218 mg/dL).

4.2 Biochemical Parameters

Results are summarized in Table 1. Mean serum magnesium was 1.35 ± 0.18 mg/dL (range: 1.04–1.78 mg/dL), below the lower boundary of the pregnancy reference range (1.7–2.2 mg/dL). Mean albumin-corrected calcium was 8.96 ± 0.41 mg/dL (range: 7.98–9.96 mg/dL). The Ca/Mg ratio demonstrated a mean of 6.75 ± 0.95 (range: 4.58–8.69, CV: 14%), indicating substantial inter-individual variability.

Table 1. Descriptive Statistics of Biochemical Parameters in Women with GDM (n = 50)

Parameter	Mean ± SD	Min	Max
Serum Magnesium (mg/dL)	1.35 ± 0.18	1.04	1.78
Albumin-Corrected Calcium (mg/dL)	8.96 ± 0.41	7.98	9.96
Calcium–Magnesium Ratio	6.75 ± 0.95	4.58	8.69
DIPSI 2-Hour Plasma Glucose (mg/dL)	168.4 ± 22.6	142	218

SD = standard deviation; DIPSI = Diabetes in Pregnancy Study Group India.

4.3 Correlation Analysis

Pearson correlation analysis revealed no statistically significant association between the Ca/Mg ratio and DIPSI 2-hour venous plasma glucose ($r = −0.060$, $p = 0.680$).

4. DISCUSSION

This cross-sectional investigation provides descriptive biochemical data on an integrated mineral homeostasis index—the albumin-corrected calcium–magnesium ratio—in women with GDM. The principal findings are: (1) serum magnesium trended toward the lower limit of the pregnancy reference range; (2) albumin-corrected calcium was largely preserved; (3) the Ca/Mg ratio exhibited substantial inter-individual variability (CV: 14%); and (4) no significant correlation was identified between the Ca/Mg ratio and acute glycemic status.

5.1 Magnesium Deficiency as an Upstream Driver of Insulin Resistance

The mean serum magnesium of 1.35 ± 0.18 mg/dL is below the lower boundary of the pregnancy reference range of 1.7–2.2 mg/dL, consistent with published literature documenting hypomagnesemia in women

with GDM.[5,18] Magnesium is required for the activation of the insulin receptor's tyrosine kinase domain; at reduced intracellular concentrations, autophosphorylation at critical tyrosine residues is impaired, attenuating downstream IRS-1/PI3K signaling and reducing GLUT-4 vesicle insertion into the plasma membrane.[4] Magnesium depletion also affects hexokinase and phosphofructokinase, impairing intracellular glucose utilization independently of insulin receptor function.[3]

During pregnancy, three concurrent mechanisms compound the risk of magnesium depletion: (1) enhanced renal magnesium excretion from increased glomerular filtration rate; (2) dilutional effect from 40–50% plasma volume expansion; and (3) active placental transfer to support fetal requirements.[16,19] In GDM, osmotic diuresis further augments urinary magnesium losses through glucose-mediated inhibition of proximal tubular magnesium reabsorption.[20]

Although not measured in the present study, chronic magnesium depletion has been reported in the literature to be associated with upregulation of inflammatory cytokines such as IL-6 and TNF-α, which are known mediators of insulin resistance in GDM.[13,21] Similarly, microRNA-29a and microRNA-132 have been reported in the literature as potential epigenetic modulators of insulin signaling and glucose transporter expression in the context of GDM and related metabolic conditions.[14,22]

5.2 Calcium's Role in β-Cell Function and Insulin Secretion

Calcium plays an indispensable role in pancreatic β-cell physiology. Following glucose-stimulated closure of KATP channels, membrane depolarization triggers L-type calcium channel opening, producing a cytosolic calcium surge that initiates insulin granule exocytosis.[6,23] Magnesium modulates this process by regulating KATP channel conductance, influencing membrane potential stability, and affecting calcium channel kinetics.[8] The largely normal albumin-corrected calcium values (mean: 8.96 ± 0.41 mg/dL) are consistent with tight ionized calcium regulation during pregnancy through enhanced intestinal calcium absorption and TRPV6 upregulation.[10]

5.3 Calcium–Magnesium Antagonism and the Ratio Concept

The Ca/Mg ratio demonstrated a coefficient of variation of 14%, indicating inter-individual heterogeneity that is not apparent from examination of either mineral in isolation. Calcium and magnesium are physiological antagonists across multiple regulatory axes: magnesium competitively inhibits calcium influx through voltage-gated channels; plasma membrane Ca^{2+}-ATPase activity is modulated by cytosolic magnesium; and the two ions compete for binding sites on metabolic enzymes and signal transduction proteins.[8,9]

5.4 Absence of Correlation with Acute Glycemic Status

The absence of a significant correlation between the Ca/Mg ratio and DIPSI 2-hour plasma glucose ($r = -0.060$, $p = 0.680$) suggests that the ratio does not serve as a real-time surrogate for hyperglycemic severity. This is consistent with positioning mineral imbalance as a chronic upstream disturbance. Studies examining adverse outcomes associated with mild gestational hyperglycemia have similarly demonstrated that metabolic consequences extend beyond what a single glucose measurement may indicate.[24] These findings are congruent with prior reports of inconsistent associations between individual mineral levels and glycemic parameters in GDM.[5,11] A conceptual overview of the proposed calcium–magnesium imbalance is illustrated in Figure 1.

5.5 Clinical and Therapeutic Implications

Serum magnesium concentrations trending below the pregnancy reference range highlight a potentially modifiable metabolic risk factor not routinely assessed in GDM management. Magnesium supplementation has been reported in some trials to improve insulin sensitivity, reduce fasting glucose, and attenuate inflammatory markers in diabetic pregnancies.[16,19] Understanding the role of magnesium deficiency in insulin resistance may inform adjunctive supplementation strategies alongside conventional GDM management, as emphasized in recent clinical updates on GDM care.[25] Adequate dietary intake of calcium and magnesium throughout pregnancy may represent a modifiable component of preventive perinatal care.[26]

5.6 Strengths and Limitations

Strengths: use of albumin-corrected calcium for ratio calculation, standardized biochemical methods, and unmodified DIPSI protocol application. Limitations: cross-sectional design, absence of a normoglycemic control group, sample size of 50, no dietary assessment, no genetic analysis of mineral transporter polymorphisms, and measurement of serum rather than ionized or intracellular mineral fractions. These findings should be interpreted as exploratory in view of the cross-sectional design.

5. CONCLUSION

This cross-sectional study characterizes albumin-corrected calcium–magnesium balance in women with GDM. Serum magnesium trended toward the lower limit of the pregnancy reference range, while albumin-corrected calcium was predominantly preserved within the physiological range. The Ca/Mg ratio demonstrated substantial inter-individual variability, and no significant correlation was identified with acute glycemic status. These findings suggest that calcium–magnesium imbalance may represent a chronic upstream metabolic perturbation in GDM, independent of acute hyperglycemia. Future longitudinal studies with normoglycemic controls, comprehensive dietary and genetic analysis, and measurement of downstream biomarkers are needed to elucidate the causal role of mineral homeostasis in GDM pathophysiology.

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ETHICS APPROVAL AND CONSENT TO PARTICIPATE

This study received approval from the Institutional Human Ethics Committee, Sri Balaji Vidyapeeth (Deemed to be University), Puducherry, India (Project No: MGMCRI/2024/02/IHEC/76; Date: 09 November 2024). The study was conducted in full compliance with the Declaration of Helsinki. Written informed consent was obtained from all participants prior to enrolment. Participants were informed of study objectives, procedures, potential risks and benefits, and their right to withdraw at any time without consequence to their medical care.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

AUTHOR CONTRIBUTIONS

R. Nalliarasi: Conceptualization, Methodology, Investigation, Data Curation, Formal Analysis, Writing – Original Draft, Writing – Review & Editing.

Reeta R: Supervision, Validation, Methodology, Writing – Review & Editing.

P. Pallavee: Investigation, Clinical Support, Writing – Review & Editing.

S. Sumathi: Conceptualization, Supervision, Writing – Review & Editing.

All authors have read and approved the final manuscript for submission.

DECLARATION OF GENERATIVE AI USE

During manuscript preparation, AI-assisted tools were utilized for language editing, grammar refinement, and manuscript structuring guidance. All content was critically reviewed, edited, and verified by the authors for scientific accuracy, methodological rigor, and intellectual integrity. The authors take full responsibility for the final published content.

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Figure 1. Conceptual representation of calcium–magnesium imbalance as a potential upstream metabolic disturbance in gestational diabetes mellitus.