

# Correlation Between Vitamin D Deficiency and Gestational Diabetes Mellitus

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## Abstract

**Background:** Vitamin D (vitD) insufficiency is increasingly recognized as a potential risk factor for gestational diabetes mellitus (GDM), but data vary among different groups. The study examined the correlation between maternal vitamin D concentrations and the onset of gestational DM in pregnant women. **Material and Methods:** Case-control research was conducted with 280 pregnant women (140 diagnosed with gestational DM and 140 controls) between 24 and 28 weeks of gestation. Serum 25-hydroxyvitamin D [25(OH)D] concentrations were quantified via electrochemiluminescence immunoassay. VitD deficiency was characterised by a 25(OH)D level of below 20 ng/mL. The oral glucose tolerance test (OGTT) was conducted in compliance with the guidelines established by the International Association of Diabetes and Pregnancy Study Groups (IADPSG). Demographic, anthropometric, and biochemical data were gathered. **Results:** Women with gestational DM exhibited intense reduction of mean serum 25(OH)D levels in comparison to controls ( $16.4 \pm 6.8$  ng/mL vs.  $24.7 \pm 8.2$  ng/mL,  $p < 0.001$ ). VitD insufficiency was observed in 78.6% of gestational DM subjects than 42.1% of control subjects ( $p < 0.001$ ). Multivariate logistic regression indicated that vitD deficiency was independently correlated with gestational DM (adjusted odds ratio = 4.12, 95% confidence interval: 2.31-7.35,  $p < 0.001$ ) after adjusting for age, body mass index, familial diabetes history, and parity. Serum 25(OH)D levels revealed a substantial inverse connection with fasting plasma glucose ( $r = -0.412$ ,  $p < 0.001$ ) and 2-hour OGTT values ( $r = -0.389$ ,  $p < 0.001$ ). **Conclusion:** A deficit in vitamin D is markedly linked to an elevated risk of gestational DM. The study outcomes corroborate the prospective role of vitamin D in glucose metabolism during gestation and necessitate further intervention trials.

**Keywords:** Vitamin D deficiency; gestational diabetes mellitus; 25-hydroxyvitamin D; pregnancy; glucose metabolism; risk factors.

Received: 22 September 2025

Revised: 25 October 2025

Accepted: 03 November 2025

Published: 11 November 2025

## INTRODUCTION

Gestational diabetes mellitus (GDM) is characterized by glucose intolerance that develops or is first identified during pregnancy, affecting approximately 7-18% of pregnancies worldwide.<sup>[1]</sup> The worldwide frequency of gestational DM has markedly increased during the last two decades, coinciding with the obesity pandemic and the rise in maternal age.<sup>[2]</sup> The gestational DM is linked with an array of maternal and neonatal problems, notably preeclampsia, cesarean section, macrosomia, newborn hypoglycemia, and enduring metabolic repercussions for the mother and child.<sup>[3]</sup> Women with gestational DM have a sevenfold elevated chance of subsequently acquiring type 2 DM.<sup>[4]</sup>

Vitamin D (vitD), historically recognized for its role in calcium regulation and bone metabolism, has emerged as a key modulator of various physiological systems, including immune function, cell proliferation, and glucose metabolism.<sup>[5]</sup> The most active variant of vitamin D, 1,25-dihydroxyvitamin D, acts by interacting with vitamin D receptors (VDR) located in various organs, including pancreatic beta cells, skeletal muscle, and adipose tissue.<sup>[6]</sup> Experimental investigations have shown that vitamin D affects insulin secretion and sensitivity via multiple cascades, with tangible impacts on pancreatic beta-cell activity and the modulation of calcium flow.<sup>[7]</sup>

VitD deficiency, represented by serum 25-hydroxyvitamin D [25(OH)D] concentrations less than 20 ng/mL, is widespread among expectant women worldwide, with prevalence rates varying from 18% to 84% based on geographic location, ethnicity, and seasonal factors.<sup>[8]</sup> VitD requirements escalate during pregnancy to support fetal skeletal growth and maternal physiological changes.<sup>[9]</sup> Studies have indicated correlations between insufficient maternal vitamin D expression and negative pregnancy outcomes, including gestational DM.<sup>[10]</sup> Recent meta-analyses indicate a negative relationship between maternal vitamin D levels and the gestational DM risk,<sup>[11,12]</sup> with contradictory results. Hu et al. revealed that women with vitamin D insufficiency had 1.53 times greater risks of developing gestational DM than those with sufficient vitamin D levels.<sup>[11]</sup> Nevertheless, alternative investigations have yielded

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**DOI:**  
10.21276/amit.2025.v12.i3.170

**How to cite this article:** Raj S, Manoj MG. Correlation Between Vitamin D Deficiency and Gestational Diabetes Mellitus. *Acta Med Int.* 2025;12(3):753-757..

contradictory findings, with certain research indicating no significant correlation.<sup>[13]</sup> The inconsistencies may result from differences in study design, ethnicity, cutoff levels for vitamin D inadequacy, timing of measurement, and the presence of confounding variables.

Notwithstanding increasing evidence, numerous information gaps persist about the association between vitamin D and gestational DM. Most prior research has been conducted on Western populations, with insufficient data from diverse ethnic groups. The independent effect of vitamin D insufficiency, even controlling for known gestational DM risk factors, necessitates more clarification. Understanding this connection has significant clinical implications, as vitamin D supplementation constitutes a potentially straightforward and cost-effective intervention.

The objective of this case-control study was to examine the relationship between maternal vitamin D concentrations and gestational DM in pregnant women, and to ascertain if vitamin D deficiency independently forecasts gestational DM risk after adjusting for conventional risk elements.

## MATERIALS AND METHODS

This case-control study was conducted in the Departments of Obstetrics & Gynecology and General Medicine of Dr Moopen's Medical College, Wayanad, Kerala. A total of 280 expectant women between 24 and 28 weeks of gestation were recruited, consisting of 140 gestational DM cases and 140 pregnant women with normal oral glucose tolerance test (OGTT) as a control group. The sample size was determined using a two-proportion approach, achieving 80% power and a 5% significance level, with a presumed gestational DM prevalence of 15% and anticipated vitamin D deficiency rates of 75% in cases and 50% in controls, resulting in a minimum requisite sample of 126 per group.

### Criteria for Inclusion and Exclusion

Inclusion criteria for cases: (1) singleton pregnancy, (2) gestational age between 24 and 28 weeks, (3) newly diagnosed gestational DM based on IADPSG criteria, and (4) age range of 18 to 40 years. Controls were matched for gestational age and exhibited normal OGTT results.

The exclusion criteria comprised: (1) pre-existing DM, (2) current vitamin D supplementation surpassing 400 IU/day, (3) chronic kidney disease, liver disease, or parathyroid disorders, (4) malabsorption syndromes, (5) administration of medications influencing vitamin D metabolism (anticonvulsants, glucocorticoids), (6) multiple pregnancy, and (7) known fetal anomalies.

**Data collection:** Comprehensive demographic and clinical data were collected using standardized questionnaires, encompassing maternal age, parity, family history of diabetes, pre-pregnancy body mass index (BMI), gestational weight gain (GWG), physical activity levels, and dietary

practices. Anthropometric measurements (height, weight, and BMI) were obtained using standardized techniques.

**Biochemical Evaluations:** Following an overnight fast of 8-10 hours, venous blood samples were obtained to assess fasting plasma glucose, lipid profile, and serum 25(OH)D levels. All subjects completed a 75-gram OGTT. Plasma glucose levels were evaluated at fasting, 1-hour, and 2-hour intervals with the glucose oxidase method on an automated analyzer (Cobas 6000, Roche Diagnostics). Gestational DM was diagnosed based on IADPSG guidelines if any of the following thresholds were attained or surpassed: fasting  $\geq 92$  mg/dL, 1-hour  $\geq 180$  mg/dL, or 2-hour  $\geq 153$  mg/dL.

The concentration of serum 25(OH)D was determined using an electrochemiluminescence immunoassay (ECLIA) with a Cobas e601 analyzer (Roche Diagnostics). The test exhibits a measurement range of 3-70 ng/mL, with inter-assay and intra-assay coefficients of deviation less than 8%. VitD level was classified as: deficient ( $<20$  ng/mL), insufficient (20-29.9 ng/mL), or sufficient ( $\geq 30$  ng/mL).

Glycosylated hemoglobin (HbA1c), serum calcium, albumin, creatinine, and lipid profile (total cholesterol (TC), triglycerides (TG), HDL-cholesterol, LDL-cholesterol) were assessed with standard laboratory techniques.

**Statistical Analysis:** Data were analyzed utilizing SPSS version 26.0 (IBM Corp., Armonk, NY). The Kolmogorov-Smirnov test was employed to evaluate the normality of continuous variables, which were presented as mean  $\pm$  standard deviation (SD) and compared between groups utilizing either the independent t-test or the Mann-Whitney U test, as deemed suitable. Categorical variables were expressed as frequencies and percentages, and the chi-square test or Fisher's exact test was applied.

Pearson or Spearman correlation analysis was conducted to evaluate the associations between serum 25(OH)D levels and glycemic indicators. A multivariate logistic regression analysis was employed to identify independent predictors of gestational DM, adjusting for relevant confounders such as maternal age, pre-pregnancy BMI, family history of DM, parity, and gestational weight gain. Odds ratios (OR) accompanied by 95% confidence intervals (CI) were computed. A two-tailed p-value of less than 0.05 was deemed statistically significant.

## RESULTS

**Baseline Characteristics:** The fundamental demographical and clinical attributes are delineated in Table 1. Gestational DM women were considerably older ( $30.8 \pm 4.6$  vs.  $28.2 \pm 4.1$  years,  $p < 0.001$ ), had a higher pre-pregnancy BMI ( $27.4 \pm 3.8$  vs.  $24.1 \pm 3.2$  kg/m<sup>2</sup>,  $p < 0.001$ ), and had elevated rates of familial diabetes history (52.9% vs. 28.6%,  $p < 0.001$ ) than the control group. No notable differences were observed in gestational age at assessment, parity distribution, or gestational weight gain between the groups.

**Table 1: Baseline Characteristics of Study Groups**

Characteristic	Gestational DM Cases (n=140)	Controls (n=140)	p-value
Age (years)	30.8 $\pm$ 4.6	28.2 $\pm$ 4.1	<0.001
Gestational age (weeks)	25.8 $\pm$ 1.2	25.6 $\pm$ 1.1	0.162
Pre-pregnancy BMI (kg/m <sup>2</sup> )	27.4 $\pm$ 3.8	24.1 $\pm$ 3.2	<0.001
Gestational weight gain (kg)	8.6 $\pm$ 2.9	8.2 $\pm$ 2.7	0.246

Nulliparous, n (%)	56 (40.0)	62 (44.3)	0.477
Multiparous, n (%)	84 (60.0)	78 (55.7)	
Family history of diabetes, n (%)	74 (52.9)	40 (28.6)	<0.001
Previous gestational DM, n (%)	28 (20.0)	0 (0.0)	<0.001
Regular physical activity, n (%)	38 (27.1)	67 (47.9)	<0.001

**Vitamin D Levels and Biochemical Indicators**

[Table 2] illustrates the analysis of vitamin D levels and biochemical markers among groups. The average serum 25(OH)D levels were markedly reduced in the gestational DM group than the control group (16.4 ± 6.8 ng/mL vs. 24.7 ± 8.2 ng/mL, p<0.001). The incidence of vitD deficiency (<20 ng/mL) was markedly greater in the gestational DM women (78.6% vs. 42.1%, p<0.001). Only 7.1% of gestational diabetes mellitus cases exhibited adequate

vitamin D levels (≥30 ng/mL), in contrast to 30.7% of the control group.

Fasting plasma glucose, 1-hour and 2-hour OGTT readings, and HbA1c were markedly elevated in the gestational DM cohort (all p<0.001). The gestational DM group exhibited markedly elevated TG levels and reduced HDL-cholesterol levels. Insignificant differences were noted in serum calcium, albumin, or creatinine scores across the groups.

**Table 2: Comparative analysis of Vitamin D Status and Biochemical factors**

Parameter	Gestational DM Cases (n=140)	Controls (n=140)	p-value
Serum 25(OH)D (ng/mL)	16.4 ± 6.8	24.7 ± 8.2	<0.001
Vitamin D, n (%)			<0.001
Deficient (<20 ng/mL)	110 (78.6)	59 (42.1)	
Insufficient (20-29.9 ng/mL)	20 (14.3)	38 (27.2)	
Sufficient (≥30 ng/mL)	10 (7.1)	43 (30.7)	
Fasting glucose (mg/dL)	96.8 ± 8.4	82.4 ± 6.2	<0.001
1-hour OGTT (mg/dL)	188.6 ± 24.3	132.5 ± 18.7	<0.001
2-hour OGTT (mg/dL)	162.4 ± 22.1	118.3 ± 16.4	<0.001
HbA1c (%)	5.6 ± 0.4	5.1 ± 0.3	<0.001
Total cholesterol (mg/dL)	218.6 ± 38.4	206.3 ± 34.2	0.006
Triglycerides (mg/dL)	186.4 ± 52.6	152.8 ± 41.3	<0.001
HDL-cholesterol (mg/dL)	52.3 ± 10.8	58.6 ± 12.4	<0.001
LDL-cholesterol (mg/dL)	128.7 ± 32.4	117.2 ± 28.6	0.002
Serum calcium (mg/dL)	9.2 ± 0.6	9.3 ± 0.5	0.168

**Correlation Analysis and Logistic Regression**

A correlation study indicated substantial inverse relationships between serum 25(OH)D levels and glucose metrics. Serum 25(OH)D exhibited an inverse correlation with fasting plasma glucose (r = -0.412, p<0.001), 1-hour OGTT (r = -0.398, p<0.001), 2-hour OGTT (r = -0.389, p<0.001), and HbA1c (r = -0.356, p<0.001).

Multivariate logistic regression analysis (Table 3) indicated that vitD deficiency was independently correlated with gestational DM after controlling for maternal age, pre-

pregnancy BMI, familial diabetes history, parity, prenatal weight gain, and physical activity. Women with vitamin D insufficiency exhibited 4.12 times greater risks of developing gestational DM (adjusted OR = 4.12, 95% CI: 2.31-7.35, p<0.001) in comparison to those with adequate vitamin D levels. Additional independent predictors comprised pre-pregnancy BMI (adjusted OR = 1.18, 95% CI: 1.09-1.28, p<0.001), family history of DM (adjusted OR = 2.64, 95% CI: 1.52-4.59, p<0.001), and maternal age (adjusted OR = 1.09, 95% CI: 1.02-1.17, p = 0.012).

**Table 3: Multivariate Logistic Regression for Predictors of gestational DM**

Variable	Crude OR (95% CI)	p-value	Adjusted OR (95% CI)	p-value
Vitamin D deficiency (<20 ng/mL)	5.02 (3.08-8.19)	<0.001	4.12 (2.31-7.35)	<0.001
Maternal age (year)	1.14 (1.08-1.21)	<0.001	1.09 (1.02-1.17)	0.012
Pre-pregnancy BMI (kg/m²)	1.26 (1.17-1.35)	<0.001	1.18 (1.09-1.28)	<0.001
Family history of DM	2.79 (1.74-4.48)	<0.001	2.64 (1.52-4.59)	<0.001
Multiparity	1.19 (0.75-1.89)	0.457	1.32 (0.76-2.29)	0.322
Previous gestational DM	-	-	3.87 (1.68-8.91)	0.001
Low physical activity	2.48 (1.52-4.05)	<0.001	1.86 (1.04-3.32)	0.036

**DISCUSSION**

This case-control study reveals a significant independent correlation between vitamin D insufficiency and gestational DM. Our data indicate that the gestational DM group exhibited markedly reduced serum 25(OH)D levels and an increased frequency of vitamin D insufficiency compared to the normoglycemic control group. Furthermore, vitD insufficiency was deemed an independent risk element for

gestational DM, with a four-fold rise in odds after controlling for existing risk variables.

The average serum 25(OH)D level recorded in the gestational DM group (16.4 ± 6.8 ng/mL) in our investigation aligns with other findings from comparable populations.<sup>[14,15]</sup> The elevated frequency of vitamin D deficiency (78.6%) in gestational DM women highlights the severity of this issue and its possible therapeutic implications. These results correspond with the findings of Zhang et al., which indicated that pregnant women in

the lowest quartile of serum 25(OH)D exhibited a 2.5-fold elevated chance of gestational DM.<sup>[16]</sup>

Various molecular mechanisms substantiate the biological plausibility of the link between vitamin D and gestational DM. VDR and 1 $\alpha$ -hydroxylase, the enzyme responsible for converting 25(OH)D to its active form, are present in pancreatic beta cells, indicating a direct influence on insulin secretion.<sup>[17]</sup> In vitro research has demonstrated that vitamin D enhances glucose-induced insulin production and protects beta cells from cytokine-mediated cell death.<sup>[18]</sup> Moreover, vitamin D influences insulin sensitivity by regulating intracellular calcium flux in insulin-responsive tissues and inhibiting inflammatory cytokines that disrupt insulin signaling.<sup>[19]</sup>

The current study disclosed strong inverse connections between serum 25(OH)D and glucose parameters, aligning with the findings of Zuhur et al., who observed analogous negative correlations between vitD levels and fasting glucose and HbA1c in individuals with gestational DM.<sup>[20]</sup> This dose-response association reinforces the causal connection between vitamin D level and glucose metabolism during gestation.

The multivariate analysis in our study, controlling for several covariates, revealed that vitamin D insufficiency independently predicted gestational DM with an adjusted odds ratio of 4.12. This exceeds the pooled estimate reported by Wei et al. (OR = 1.53),<sup>[21]</sup> potentially indicating a greater likelihood of vitamin D deficiency in the present study group or variations in cutoff definitions. Our results align closely with the prior systematic review findings of Hu et al., which indicated that severe vitamin D insufficiency (<12 ng/mL) was linked to a 2.7-fold heightened gestational DM risk.<sup>[11]</sup>

Numerous intervention trials have examined the effectiveness of vitamin D supplementation in decreasing the prevalence of gestational DM. Shahgheibi et al. documented that vitamin D administration (50,000 IU twice a week) during pregnancy substantially reduced the gestational DM risk compared to placebo.<sup>[22]</sup> A Cochrane review determined that the evidence is inadequate to endorse routine vitamin D administration for the alleviation of gestational DM, highlighting the heterogeneity and methodological shortcomings of current trials.<sup>[23]</sup> Comprehensive, well-structured trials with appropriate dose protocols, commenced early in gestation, are essential to establish the preventive efficacy of vitamin D supplementation conclusively.

Our research also recognized conventional risk variables for gestational DM, such as pre-pregnancy BMI, familial diabetes history, and maternal age, aligning with existing literature.<sup>[24]</sup> The interplay of modifiable factors (vitamin D levels, BMI, and physical activity) and non-modifiable risk components provides a comprehensive understanding of gestational DM pathophysiology and potential intervention targets.

The considerable frequency of vitamin D deficiency reported in both groups, particularly among the gestational DM group, indicates a public health issue that necessitates intervention. Geographic location, restricted sunlight exposure due to cultural customs, nutritional inadequacy, and the absence of food fortification initiatives may contribute to the prevalence

of vitamin D deficiency among expectant mothers.<sup>[25]</sup> These findings underscore the need for regular vitamin D assessment during pregnancy and the establishment of targeted supplementation protocols.

This investigation possesses some noteworthy strengths that warrant attention. The thorough evaluation of many confounding variables and stringent statistical adjustments improve the validity of our results. Employing defined IADPSG criteria for gestational DM diagnosis and verified laboratory techniques for 25(OH)D testing enhances dependability. Nonetheless, limitations exist in the case-control design, which hinders the establishment of timing and causation. Residual confounding from unmeasured variables, like dietary vitamin D consumption and sun exposure, cannot be eliminated. The cross-sectional assessment of vitamin D at 24-28 weeks may not accurately represent first-trimester levels when pancreatic beta-cell adaptation commences. Future prospective studies assessing vitamin D concentrations in early pregnancy would yield more robust information concerning temporal correlations.

## CONCLUSION

This research reveals a considerable correlation between vitamin D deficiency and gestational DM. The gestational DM group exhibited profoundly reduced blood 25-hydroxyvitamin D levels and an elevated spike of vitamin D insufficiency compared to the normoglycemic pregnant women. VitD insufficiency has been determined as an independent risk element for gestational DM, resulting in a fourfold rise in risk after adjusting for conventional risk variables, notably maternal age, BMI, and familial DM history. The inverse relationships between vitamin D levels and glucose markers further substantiate vitamin D's function in glucose homeostasis during pregnancy.

These findings possess significant clinical and public health ramifications. Due to the substantial incidence of vitamin D deficiency in pregnant women and the likelihood of modifying this risk factor, systematic screening for vitamin D levels during pregnancy warrants consideration. Before the implementation of extensive supplementation programs, comprehensive randomized controlled studies are essential to determine effective dose regimens, timing of intervention, and efficacy in preventing gestational DMs. VitD supplementation is a potentially safe, cost-effective, and accessible solution that alleviates the burden of gestational DM and its related consequences. Subsequent research ought to concentrate on intervention studies to ascertain the effectiveness of vitamin D supplementation in mitigating gestational DM and enhancing maternal and newborn outcomes.

## Financial support and sponsorship

Nil.

## Conflicts of interest

There are no conflicts of interest.

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