ORIGINAL RESEARCH ARTICLE

Effects of vitamin D supplementation on insulin resistance and lipid metabolic profiles in women with gestational diabetes mellitus: A randomized controlled trial

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Abstract

Gestational diabetes mellitus (GDM) commonly presents as insulin resistance and altered lipid metabolism, with evidence linking vitamin D deficiency to its pathogenesis. In this randomized, double-blind, placebo-controlled trial, 120 pregnant women diagnosed with GDM were assigned to receive either vitamin D3 supplements (2000 IU/day) or a placebo for 3 months. Serum vitamin D status, glycemic control parameters, and lipid profiles were evaluated pre- and post-intervention, adjusting for dietary and exercise confounders. After the 3-month intervention, serum 25-hydroxyvitamin D levels in the vitamin D group were significantly higher than those in the placebo group. Vitamin D supplementation effectively reduced fasting plasma glucose, fasting insulin, and HOMA-IR scores. Simultaneously, it significantly decreased triglycerides and LDL-C levels while increasing HDL-C. Total cholesterol levels showed no statistically significant difference between the two groups. Supplementation with 2000 IU vitamin D per day for 3 months yields significant improvements in glucose homeostasis and lipid metabolism in the GDM population. Vitamin D holds promise as an effective auxiliary treatment to enhance maternal metabolic health. *(Afr J Reprod Health 2026; 30 [14]: 86-93).*

Keywords: Gestational diabetes mellitus, Vitamin D, Insulin resistance, Lipid metabolism, Randomized controlled trial

Résumé

Le diabète gestationnel (DG) se manifeste fréquemment par une résistance à l'insuline et des anomalies du métabolisme lipidique, avec des données suggérant un lien entre la carence en vitamine D et sa physiopathologie. Dans cet essai randomisé, en double aveugle et contrôlé par placebo, 120 femmes enceintes diagnostiquées avec un diabète gestationnel ont été réparties pour recevoir soit une supplémentation en vitamine D3 (2000 UI/jour), soit un placebo pendant 3 mois. Le statut en vitamine D sérique, les paramètres de contrôle glycémique et les profils lipidiques ont été évalués avant et après l'intervention, en ajustant les facteurs de confusion liés à l'alimentation et à l'activité physique. Après 3 mois d'intervention, les concentrations sériques de 25-hydroxyvitamine D dans le groupe vitamine D étaient significativement plus élevées que dans le groupe placebo. La supplémentation en vitamine D a permis de réduire efficacement la glycémie à jeun, l'insuline à jeun et les scores HOMA-IR. Elle a également diminué significativement les triglycérides et le LDL-C, tout en augmentant le HDL-C. Aucun changement statistiquement significatif n'a été observé pour le cholestérol total entre les deux groupes. Une supplémentation de 2000 UI/jour de vitamine D pendant 3 mois améliore significativement l'homéostasie du glucose et le métabolisme lipidique chez les patientes atteintes de diabète gestationnel. La vitamine D constitue un traitement adjuvant prometteur pour améliorer la santé métabolique maternelle. *(Afr J Reprod Health 2026; 30 [14]: 86-93).*

Mots-clés: Diabète gestationnel ; Vitamine D ; Résistance à l'insuline ; Métabolisme lipidique ; Essai contrôlé randomisé

Introduction

Gestational diabetes mellitus (GDM) is abnormal glucose tolerance that occurs for the first time during pregnancy, and it is one of the most common metabolic diseases in pregnant women.[1] Based on the International Association of Diabetes and Pregnancy Study Groups (IADPSG) criteria for the diagnosis of diabetes and gestational diabetes, the weighted global prevalence of gestational diabetes was 14.0 percent. The prevalence in different regions varies considerably; 27.6 percent in the Middle East and North Africa and as low as 7.1 per cent in North America and the Caribbean.[2]

Although there are still differences in diagnostic thresholds, early recognition and glycemic control are essential to improve perinatal outcomes.[3] A meta-analysis of machine learning models confirmed that maternal age, BMI, fasting blood glucose, and family history of diabetes were all significant and measurable risk factors for GDM, and thus feasible for accurate screening.[4] GDM greatly increases the risk of pregnancy complications and has a prolonged negative impact on the metabolism of both the mother and the child.[1] Therefore, guidelines in many countries recommend the 75 g OGTT and long-term metabolic monitoring at 4-12 weeks after delivery.[3] However, in low-resource areas, there are still obstacles in the accessibility and insurance coverage of continuous glucose monitoring, and policy interventions are urgently needed to narrow the health gap.

Insulin resistance and atherogenic dyslipidaemia are the two typical characteristics of GDM. Cross-sectional studies have shown that in all three trimesters of pregnancy, GDM patients have increased fasting and postprandial blood glucose and glycated haemoglobin levels, as well as elevated triglycerides (TG), total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C) and very low-density lipoprotein (VLDL). Reduced high-density lipoprotein cholesterol (HDL-C) was also observed, and the study found a significant decrease in insulin levels and the homeostatic model assessment of insulin resistance (HOMA-IR).[5] A hyperglycaemic environment in the mother may cause fetal hyperinsulinemia, promote increased accumulation of adipose tissue, and alter the adipokine profile.[6] Evidence from long-term follow-up studies reveals that fetuses exposed to GDM in utero tend to develop metabolic disorders at an early age, such as insulin resistance, glucose intolerance, high blood pressure and abnormal lipid profiles.[7] Existing strategies can improve maternal glycemic status in pregnancy, but evidence for long-term metabolic protection in offspring is insufficient.[7] Unraveling GDM-associated fetal programming contributes to preventing chronic non-communicable diseases in offspring.[8]

Vitamin D and steroids are fat-soluble, so there have been studies on the link between them and the development of GDM. Pregnant women frequently have vitamin D deficiency (serum 25(OH)D < 20 ng/mL), and this is closely linked to the start of GDM9. Vitamin D receptors (VDRs) are widely distributed throughout the body, including in pancreatic beta cells, skeletal muscle, and adipose tissue, where they regulate glucose and lipid metabolism.[10, 11] Reduce fat synthesis and promote the breakdown of fat. Several studies have shown that pregnant women with vitamin D deficiency and those with gestational diabetes have an increased risk of insulin resistance and dyslipidemia.[13, 14] However, current interventional studies yield inadequate and heterogeneous results. Certain randomized controlled trials have verified that vitamin D intake ameliorates insulin sensitivity and lipid parameters in GDM populations[15,16], while others have found no significant effects. These discrepancies may be due to differences in study designs, sample sizes, vitamin D doses, and duration of interventions.

Therefore, this study aimed to investigate the effects of daily supplementation with 2000 IU of vitamin D for 3 months on insulin resistance and lipid metabolism in patients with GDM. In order to improve insulin sensitivity and reduce the proportion of body fat in pregnant women with diabetes, Vitamin D and a control group have been studied.

Methods

Study design and participants

This clinical investigation, which adopted a randomized, double-blind, placebo-controlled design, was conducted at Beijing Chaoyang Hospital of Capital Medical University from January 2020 to December 2021.

The following inclusion criteria were applied to the study population: (1) Age 18-40 years; (2) Diagnosed with gestational diabetes mellitus (GDM) at 24-28 weeks of gestation based on the International Association of Diabetes and Pregnancy Study Groups (IADPSG),**Error! Reference source not found.** which requires at least one plasma glucose value from a 75-g oral glucose tolerance test (OGTT) to meet or exceed the following thresholds: fasting ≥ 5.1 mmol/L, 1-hour ≥ 10.0 mmol/L, or 2-hour ≥ 8.5 mmol/L; (3) Singleton pregnancy; (4) No previous history of diabetes

mellitus; (5) No use of insulin or oral hypoglycemic drugs; and (6) No use of vitamin D supplements in the past 3 months. Exclusion criteria were as follows: (1) multiple gestation; (2) pre-existing diabetes mellitus; (3) hypertension; (4) thyroid disorders; (5) kidney or liver disease; (6) malabsorption syndrome; (7) medication affecting vitamin D metabolism; and (8) tobacco use, alcohol consumption, or illicit drug use.

Sample size calculation

Based on the first goal of HOMA-IR, a sample size was selected. Based on the data from the previous study (14), a mean difference of 0.8 and a standard deviation of 1.2 for HOMA-IR in the vitamin D and placebo groups were observed; therefore, a sample size of 48 participants per group was required to achieve a power of 80% and a two-sided alpha of 0.05 for the detection of a significant difference. A dropout rate of 20% was set, and a total of 120 participants (60 per group) were obtained.

Randomization and blinding

A 1:1 randomization of participants to receive either vitamin D supplementation or placebo was achieved via a computerized random number generation system. The distribution schedule was in an invisible sealed envelope, and a research assistant uninvolved in either the recruitment or evaluation of subjects had to open it. Participants, investigators, and outcome assessors were blinded to group allocation throughout the study.

Intervention

Study subjects in the vitamin D group received 2000 IU vitamin D3 orally each day for 3 months, and the placebo group was supplied with indistinguishable placebo capsules. Vitamin D3 supplements and matching placebo capsules were identical in appearance, colour, size, taste, and odour to ensure blinding throughout the study period. Participants took one capsule daily with food and were asked to return any unused capsules. Compliance was measured by counting the remaining capsules, and consuming at least 80% of the prescribed supplements was considered compliant.

Outcome measures

The first end point was the change in HOMA-IR at 3 months from baseline. Other endpoints included changes in FPG, fasting insulin, TG, TC, LDL-C, HDL-C, and serum 25(OH)D levels.

Blood samples were taken before the start of the study and again three months later. DiaSorin, Stillwater, MN, USA was employed to perform a chemiluminescent immunoassay (CLIA) of serum 25(OH)D levels. HOMA-IR was calculated as: [fasting insulin (μU/mL) times FPG (mmol/L)] / 22.5. A Hitachi 7600 automatic biochemistry analyzer (Tokyo, Japan) is employed to measure TG, TC, LDL-C and HDL-C in the lipid area.

A semi-quantitative Food Frequency Questionnaire (FFQ) (20) was employed to record the diet of the subjects, and at the beginning and end of the intervention, the International Physical Activity Questionnaire (IPAQ) (21) was used to assess physical activity levels.

Statistical analysis

Statistical analysis of all collected data was implemented with Statistic Package for Social Science (SPSS) 24.0 (IBM Corporation, Armonk, NY, USA). Data normality was examined using the Kolmogorov-Smirnov test. Continuous variables were presented as mean ± SD or median (interquartile range), and categorical variables were summarized by case numbers and proportional percentages. Differences in baseline clinical and demographic parameters between the two cohorts were determined by independent t-test, Mann-Whitney U test or chi-square test depending on data normality. This study adopted the intention-to-treat (ITT) analysis framework, and the last observation carried forward (LOCF) technique was employed to compensate for missing data. Statistical comparisons of outcome changes after 3 months of intervention were conducted between groups. Analysis of covariance (ANCOVA) was performed to adjust for potential confounders, including maternal age, pre-pregnancy body mass index, gestational age, dietary status and physical activity level. Significance was set at a two-tailed P value below 0.05 for all analyses.

Ethical considerations

This study was conducted in accordance with the ethical principles of the Declaration of Helsinki. Ethical approval was obtained from the Institutional Review Board of Beijing Chaoyang Hospital, Capital Medical University. Written informed consent was obtained from all participants prior to enrollment. Participation was voluntary, and all personal information was kept confidential and used solely for research purposes. Participants were free to withdraw from the study at any stage without any consequences to their medical care.

Results

Participant characteristics

A total of 120 women with gestational diabetes mellitus (GDM) were randomized into the vitamin D group (n=60) and the placebo group (n=60). Ultimately, 56 patients in the vitamin D group and 55 in the placebo group completed the trial, with dropout rates of 6.7% and 8.3%, respectively, showing no significant intergroup difference (P=0.75). Baseline characteristics, including age, pre-pregnancy BMI, gestational week, parity, educational background, occupation, family history of diabetes, physical activity, diet, and serum 25(OH)D concentration, were comparable between the two groups (all P>0.05), as summarized in Table 1.

Effects on insulin resistance

After the 3-month intervention period, participants receiving vitamin D showed a significant increase in serum 25(OH)D compared to the placebo group (mean difference: 15.2 ng/mL, 95% CI: 12.4-18.0, P<0.001) (Table 2).

Vitamin D supplementation also significantly reduced fasting plasma glucose (FPG), fasting insulin, and HOMA-IR (P=0.02, P=0.01, and P=0.003, respectively). All these significant findings remained robust after adjusting for potential confounding factors (all P<0.05).

Effects on lipid metabolism

Relative to placebo, vitamin D supplementation yielded notable reductions in triglycerides (TG, mean difference: -0.4 mmol/L, 95% CI: -0.7 to -0.1, P=0.04) and low-density lipoprotein cholesterol (LDL-C, mean difference: -0.3 mmol/L, 95% CI: -0.5 to -0.1, P=0.03). Meanwhile, high-density lipoprotein cholesterol (HDL-C) was significantly elevated (mean difference: 0.2 mmol/L, 95% CI: 0.1 to 0.3, P=0.02) (Table 2). No significant intergroup difference was observed in total cholesterol (TC) levels (mean difference: -0.2 mmol/L, 95% CI: -0.5 to 0.1, P=0.08). All significant differences remained valid after adjusting for the aforementioned confounders (all P<0.05).

Adverse events

No serious adverse reactions occurred during the entire intervention period. The incidences of mild discomforts including nausea, vomiting and constipation were similar in the two study groups, with no statistically significant differences (all P > 0.05), as shown in Table 3.

Table 1: Baseline characteristics of the participants

Characteristic	Vitamin D group (n=60)	Placebo group (n=60)	P value
Age (years)	28.5 ± 4.2	29.1 ± 3.9	0.42
Pre-pregnancy BMI (kg/m²)	22.3 ± 3.1	22.8 ± 2.9	0.36
Gestational age (weeks)	26.2 ± 1.4	26.5 ± 1.3	0.23
Parity (nulliparous)	33 (55.0)	36 (60.0)	0.58
Education level (college)	42 (70.0)	39 (65.0)	0.55
Employment status (employed)	45 (75.0)	48 (80.0)	0.51
Family history of diabetes	18 (30.0)	15 (25.0)	0.53
Dietary intake	-	-	-
Energy (kcal/day)	1985 ± 354	1942 ± 329	0.49
Carbohydrate (g/day)	275 ± 52	268 ± 48	0.44
Protein (g/day)	85 ± 18	82 ± 16	0.34
Fat (g/day)	68 ± 15	65 ± 14	0.26
Vitamin D (IU/day)	135 ± 42	128 ± 39	0.35
Physical activity (MET-min/week)	1125 ± 486	1058 ± 452	0.43
Serum 25(OH)D (ng/mL)	16.8 ± 5.2	17.3 ± 4.9	0.58

Table 2: Modulatory effects of vitamin D supplementation on insulin sensitivity and blood lipid metabolism

	Vitamin D group (n=60)	Placebo group (n=60)	Mean difference (95% CI)	P value
Serum 25(OH)D (ng/mL)	Baseline: 16.8 ± 5.2 3 months: 35.2 ± 6.4	Baseline: 17.3 ± 4.9 3 months: 18.1 ± 5.1	15.2 (12.4 to 18.0)	<0.001
FPG (mmol/L)	Baseline: 5.2 ± 0.6 3 months: 4.8 ± 0.5	Baseline: 5.1 ± 0.5 3 months: 5.0 ± 0.6	-0.3 (-0.5 to -0.1)	0.02
Fasting insulin (μU/mL)	Baseline: 14.5 ± 4.2 3 months: 11.2 ± 3.5	Baseline: 13.9 ± 3.8 3 months: 13.4 ± 4.1	-2.5 (-4.4 to -0.6)	0.01
HOMA-IR	Baseline: 3.4 ± 1.2 3 months: 2.4 ± 0.9	Baseline: 3.2 ± 1.1 3 months: 3.0 ± 1.0	-0.8 (-1.3 to -0.3)	0.003
TG (mmol/L)	Baseline: 2.8 ± 0.9 3 months: 2.3 ± 0.7	Baseline: 2.7 ± 0.8 3 months: 2.6 ± 0.8	-0.4 (-0.7 to -0.1)	0.04
TC (mmol/L)	Baseline: 6.2 ± 1.1 3 months: 5.8 ± 0.9	Baseline: 6.1 ± 1.0 3 months: 6.0 ± 1.0	-0.2 (-0.5 to 0.1)	0.08
LDL-C (mmol/L)	Baseline: 3.5 ± 0.8 3 months: 3.1 ± 0.6	Baseline: 3.4 ± 0.7 3 months: 3.3 ± 0.7	-0.3 (-0.5 to -0.1)	0.03
HDL-C (mmol/L)	Baseline: 1.2 ± 0.3 3 months: 1.5 ± 0.4	Baseline: 1.3 ± 0.3 3 months: 1.3 ± 0.3	0.2 (0.1 to 0.3)	0.02

Results are presented as mean ± SD. The abbreviations are defined as follows: FPG, fasting plasma glucose; HOMA-IR, homeostasis model assessment of insulin resistance; TG, triglycerides; TC, total cholesterol; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol.

Table 3: Adverse events during the study

Adverse event	Vitamin D group (n=60)	Placebo group (n=60)	P value
Nausea	5 (8.3)	4 (6.7)	0.73
Vomiting	3 (5.0)	2 (3.3)	0.65
Constipation	6 (10.0)	5 (8.3)	0.75
Headache	4 (6.7)	3 (5.0)	0.70
Dizziness	2 (3.3)	1 (1.7)	0.56
Rash	1 (1.7)	2 (3.3)	0.56

Data are presented as number (percentage).

Discussion

This three-month randomized controlled trial demonstrated that daily supplementation with 2000 IU of vitamin D significantly ameliorated insulin resistance and improved the lipid profile in women with GDM. To date, few RCTs have concurrently evaluated the effects of vitamin D on both glucose homeostasis and lipid metabolism in this specific population.

Consistent with previous meta-analyses, our findings support that vitamin D supplementation enhances insulin sensitivity in GDM patients. A recent systematic review reported a mean reduction in HOMA-IR of -1.1 (95% CI: -1.5 to -0.7) following vitamin D administration during pregnancy, and suggested that doses ≥2000 IU/day may be particularly effective in preventing insulin resistance.[0] Despite the biological plausibility, existing clinical evidence on vitamin D's effect on insulin sensitivity in GDM remains heterogeneous. Some well-designed RCTs failed to demonstrate significant improvements in glycemic control.[0, 0] These discrepancies likely stem from differences in population characteristics (e.g., race, skin color, geographic latitude), intervention timing (gestational week at initiation), dosage regimens (daily vs. weekly, total cumulative dose), baseline vitamin D status, and even gestational age at assessment.[0] Consequently, while vitamin D is mechanistically capable of alleviating insulin resistance, further large-scale, high-quality RCTs are warranted to define its actual clinical effectiveness in GDM.[0]

The mechanisms by which vitamin D improves insulin sensitivity are not fully elucidated but likely involve multiple pathways. First, vitamin D upregulates insulin receptor expression and enhances receptor sensitivity in insulin-target tissues (e.g., skeletal muscle and adipose tissue), thereby amplifying insulin signaling.[0] Second, it maintains calcium homeostasis, which is essential for normal insulin secretion and peripheral insulin action.[0] Third, vitamin D exerts immunomodulatory and anti-inflammatory effects that may mitigate the chronic low-grade inflammation characteristic of insulin-resistant states.[0, 0]

Our results also demonstrate that vitamin D supplementation significantly reduces TG and LDL-C while raising HDL-C in GDM patients, consistent with previous findings in other insulin-resistant populations (e.g., metabolically unhealthy obese individuals and multiple sclerosis patients).[0, 0, 0] However, evidence specifically in GDM has been limited. A recent RCT enrolling GDM women with baseline serum 25(OH)D <75 nmol/L reported that 1600 IU/day of vitamin D_3 for 8 weeks (starting at 24-28 weeks of gestation) significantly increased HDL-C and decreased the triglyceride-glucose index, supporting the notion that vitamin D may improve cardiovascular risk markers during pregnancy.[0]At the molecular level, vitamin D exerts lipid-modulating effects by suppressing the transcription of lipogenic genes (e.g., fatty acid synthase [FAS] and sterol regulatory element-binding protein-1c [SREBP-1c])while upregulating lipolytic genes(e.g., hormone-sensitive lipase [HSL] and adipose triglyceride lipase [ATGL]).[0] Additionally, vitamin D regulates key enzymes in cholesterol metabolism, including HMG-CoA reductase and CYP7A1.[0]

Our findings suggest that routine vitamin D screening and supplementation at a dose of 2000 IU/day could be considered as an affordable, low-risk adjunctive strategy to improve metabolic health in GDM patients, particularly in regions with a high prevalence of vitamin D deficiency. Healthcare policymakers should consider integrating vitamin D assessment into standard GDM care protocols. Clinicians may advise GDM patients with low baseline 25(OH)D levels to initiate supplementation early after diagnosis to optimize glycemic and lipid outcomes. Nevertheless, further cost-effectiveness analyses are needed before large-scale implementation.

A major strength of this study lies in its rigorous randomized, double-blind, placebo-controlled design. Furthermore, the protocol featured several distinct methodological advantages, including the administration of a robust therapeutic dose of vitamin D3 (2000 IU/day), an extended intervention period (3 months) that exceeds that of many previous trials, and a comprehensive, simultaneous assessment of both glucose homeostasis and lipid metabolism.

Crucially, the validity of our findings is bolstered by the strict statistical control of potential confounding variables, specifically dietary intake and physical activity levels.Nevertheless, several limitations warrant cautious interpretation. First, our protocol did not track long-term clinical endpoints for the maternal-fetal dyad, such as gestational hypertension, preeclampsia, cesarean delivery rates, fetal macrosomia, or neonatal hypoglycemia.

Second, we did not evaluate intermediary mechanistic biomarkers, such as adiponectin, pro-inflammatory cytokines, apolipoproteins, or oxidized low-density lipoprotein (ox-LDL), which might provide deeper insights into the underlying pathways.

Third, being a single-center trial with a relatively modest sample size, the generalizability of our conclusions to broader ethnic or regional cohorts remains to be established. Lastly, the 3-month follow-up window was confined to the gestational period, thereby precluding an assessment of whether these metabolic benefits persist into the postpartum phase.

Conclusion

2000 IU/day of vitamin D_3 supplementation for 3 months effectively reduces insulin resistance and dyslipidaemia in patients with GDM, and it is safe. Based on the above results, Vitamin D supplementation shows promising clinical value; however, long-term follow-up studies are still essential to confirm its protective effects on neonates.

Conflict of interests

The authors declared no conflict of interest.

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Authors' contributions

Yunhui Duan: Conceptualization, Methodology, Formal analysis, Writing-original draft. Shixuan Zhang: Investigation, Data curation, Formal analysis. Xiaojun Ma: Investigation, Resources, Project administration. Li Li: Validation, Methodology, Visualization. Kai Jia: Conceptualization, Supervision, Funding acquisition, Writing-review & editing.

Data availability

The datasets generated and/or analysed during the current study are available from the corresponding author on reasonable request.

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