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Role of Empirical Treatment with Vitamin D in Improving Glycemic Control of Type 2 Diabetes Mellitus Patients

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ABSTRACT

Background: Type 2 Diabetes Mellitus (T2DM) remains a global health challenge, with many patients failing to achieve optimal glycemic control. Vitamin D, through its effects on insulin secretion and sensitivity, has emerged as a potential adjunctive therapy. However, evidence from clinical trials on its efficacy has been inconsistent. **Objective:** To evaluate the efficacy of empirical vitamin D supplementation as an adjunct therapy in improving glycemic control in patients with T2DM. **Methods:** A randomized, double-blind, placebo-controlled trial was conducted over 24 weeks. Sixty patients with T2DM (HbA1c >6.5%) were randomized into two groups. The intervention group (n=30) received oral Vitamin D_3 (20,000 IU weekly for 8 weeks, then 1,000 IU daily for 4 weeks) alongside standard anti-diabetic therapy. The control group (n=30) received an identical placebo with standard therapy. The primary outcome was the change in HbA1c from baseline to 12 weeks. **Results:** At baseline, both groups were well-matched in clinical parameters, including mean HbA1c (Vitamin D: 9.51%; Placebo: 9.55%). After 12 weeks, the Vitamin D group exhibited a significantly greater reduction in HbA1c compared to the placebo group (mean post-treatment HbA1c: 7.81% vs. 8.57%; mean difference: -0.76%, 95% CI: -1.13 to -0.39, p<0.001). Stratified analysis revealed the most pronounced benefits in females, patients aged ≥60 years, obese individuals, and those on insulin-only or oral anti-diabetic regimens. **Conclusion:** Empirical vitamin D supplementation significantly improves glycemic control in patients with T2DM, leading to a clinically meaningful reduction in HbA1c. The therapy was particularly effective in specific high-risk subgroups. These findings support the role of vitamin D as a safe and effective adjunctive treatment in T2DM management, warranting routine screening for deficiency in these populations.

INTRODUCTION

Type 2 Diabetes Mellitus (T2DM) persists as a pressing global pandemic, its pathogenesis rooted in the dual defects of insulin resistance and progressive pancreatic β-cell dysfunction, which culminate in chronic hyperglycemia.[1] The cornerstone of T2DM management, optimal glycemic control, is critically linked to mitigating the risk of devastating micro- and macrovascular complications. Despite a growing pharmacopeia of anti-diabetic agents, a substantial number of patients remain unable to achieve their glycemic targets, highlighting an urgent need for complementary therapeutic strategies that address underlying pathophysiological mechanisms.[2] In this context, vitamin D has transcended its classical identity as a bone-regulating secosteroid hormone. The discovery of vitamin D receptors (VDRs) in insulin-sensitive tissues—including pancreatic β-cells, skeletal muscle, and adipose tissue—has unveiled its extraskeletal roles[3] A compelling body of evidence now posits vitamin D as a key regulator of glucose homeostasis, implicated in enhancing insulin secretion, improving peripheral insulin sensitivity, and dampening the chronic, low-grade inflammation characteristic of T2DM.[4,5]

This mechanistic insight is supported by robust observational data, which consistently reveal a high prevalence of hypovitaminosis D in T2DM populations and a significant inverse correlation between serum 25-hydroxyvitamin D [25(OH)D] levels and markers of glycemic control, such as HbA1c. This epidemiological link has prompted clinical trials to investigate the therapeutic potential of vitamin D supplementation. However, the results have been heterogeneous, with some studies demonstrating significant improvements in glycemic control, while others have reported null findings. This inconsistency may be attributed to variations in study design, baseline vitamin D status, dosage regimens, and participant characteristics.[6,7]

To address this critical evidence gap, we designed a randomized, placebo-controlled trial. This study aims to provide a definitive evaluation of the efficacy of empirical vitamin D supplementation as an adjunct therapy in improving glycemic control in patients with T2DM, moving beyond correlation to establish causation and inform clinical practice.

MATERIALS AND METHODS

This randomized, double-blind, placebo-controlled trial was conducted at the Department of Medicine, DHQ Teaching Hospital, Mirpur, Azad Kashmir, over a six-month enrollment and intervention period (September 2023 to February 2024). The study protocol received full ethical approval from the Institutional Review Board (IRB) and the College of Physicians and Surgeons Pakistan (CPSP). Prior to enrollment, written informed consent was obtained from every participant. A rigorous confidentiality protocol was enforced throughout the research process to ensure all patient data were anonymized and utilized solely for the purposes of this clinical investigation. The sample size was calculated using the WHO sample size calculator for the comparison of two population means. With a significance level (α) of 0.05 and a power (1-β) of 95%, and based on parameters from prior research conducted by Yousefi Rad E et al. (mean HbA1c of 6.76 vs. 7.73, standard deviation of 0.205). A minimum sample size was determined using this and rounded up to 60 participants to enhance the power of study and account for potential dropouts, resulting in 30 patients allocated to the Vitamin D group and 30 to the placebo group. Consecutive eligible patients presenting to the outpatient department were shortlisted for participation. Subsequently, a simple non-probability consecutive sampling technique was applied, whereby these consented individuals were randomly assigned to one of the two study groups using a computer-generated randomization list. Eligible participants had age ranged from 25–75 years with a confirmed diagnosis of T2DM (baseline HbA1c > 6.5%) who had not received vitamin D or other vitamin supplementation in the preceding three months. Patients with Type 1 diabetes, gestational diabetes, chronic renal or liver disease, malabsorption syndromes, thyroid dysfunction, or those who were pregnant or lactating were excluded. Non-compliant individuals were also excluded.

Participants were randomized into two groups. Group A (Intervention) received oral Vitamin D_3 (Cholecalciferol) at a dose of 20,000 IU weekly for eight weeks, followed by 1,000 IU daily for the subsequent four weeks, in addition to their standard anti-diabetic therapy. Group B (Control) received an identical-looking placebo capsule alongside their standard diabetes management. Both groups received consistent advice on lifestyle modification and dietary adherence. At baseline, data on demographic characteristics, duration of diabetes, type of diabetic treatment, and Body Mass Index (BMI) were recorded using a pre-designed proforma. The primary outcome measure, HbA1c, was assessed at baseline and after three months through the hospital laboratory. BMI was calculated and categorized according to WHO classifications. Follow-up visits were scheduled monthly to monitor therapy compliance and record any adverse effects. Gathered data was interrogated using SPSS Statistics (Version 25.0, IBM Corp.). Continuous variables are presented as mean ± standard deviation, while categorical data are summarized as frequency and percentages. The primary outcome i.e. difference in mean HbA1c, was assessed using an independent samples t-test. To delineate the consistency of the treatment effect across clinically relevant subpopulations, a pre-specified stratified analysis was performed, segmenting the study population by gender, age, anti-diabetic treatment type, and Body Mass Index (BMI) categories. A two-tailed p-value of ≤ 0.05 defined statistical significance for all inferential tests.

RESULTS

A total of 60 patients with Type 2 Diabetes Mellitus were enrolled and anlysis of the data reflected that at baseline, the two groups were well-matched across key clinical parameters. The mean age of participants in the Vitamin D group was 52.40 ± 15.84 years, compared to 52.17 ± 15.35 years in the Placebo group. The mean Body Mass Index (BMI) was 26.21 ± 2.97 kg/m^2 and 26.97 ± 3.55 kg/m^2 in the Vitamin D and Placebo groups, respectively. The duration of diabetes was slightly higher in the Vitamin D group (8.97 ± 4.14 years) than in the Placebo group (7.93 ± 3.53 years). Crucially, the mean baseline HbA1c levels were nearly identical between the groups, at 9.51 ± 0.54% for the Vitamin D group and 9.55 ± 0.74% for the Placebo group, indicating comparable baseline glycemic control prior to intervention. A slight disparity was noted in gender distribution, with the Vitamin D group having a higher proportion of males (70%) compared to the Placebo group (53.3%). The groups were perfectly balanced across age categories, with an equal distribution of participants in the <45 years, 45-59 years, and ≥60 years strata. The majority of participants in both groups were categorized as Overweight (53.3% each), and the most common treatment type at baseline was oral anti-diabetic agents (60% in Vitamin D vs. 56.7% in Placebo). The distribution of qualitative baseline characteristics is presented in table 1.

The impact of vitamin D supplementation on glycemic control, measured by the change in HbA1c, was the primary outcome of this study. As detailed in table 2, both the Vitamin D and Placebo groups had nearly identical mean baseline HbA1c levels. Following the 12-week intervention period, a significant reduction in HbA1c was observed in both groups. However, the reduction was markedly greater in the Vitamin D group. The mean post-treatment HbA1c in the Vitamin D group was 7.81% (±0.58), compared to 8.57% (±0.82) in the Placebo group. The independent samples *t*-test revealed a statistically significant mean difference of -0.76% (95% CI: -1.13 to -0.39, p<0.001), indicating that empirical vitamin D supplementation led to a superior improvement in glycemic control compared to standard therapy alone.

To explore the consistency of the treatment effect across different patient subgroups, a stratified analysis was performed on the post-treatment HbA1c levels summarized in as summarized in table 3. The beneficial effect of vitamin D supplementation was consistently observed in most subgroups. The reduction in HbA1c was

statistically significant and pronounced among females (Mean Difference: -1.09%, p<0.001) compared to males (Mean Difference: -0.62%, p=0.022). When stratified by age, the most substantial benefit was seen in patients aged 60 years and older (Mean Difference: -1.42%, p<0.001), followed by those in the 45-59 years category (Mean Difference: -0.73%, p=0.005). No significant inter-group difference was found in participants under 45 years (p=0.668). Analysis by treatment type showed that patients managed with oral hypoglycemic agents (Mean Difference: -0.73%, p=0.001) and those on insulin-only regimens (Mean Difference: -1.67%, p=0.015) derived significant benefit from vitamin D. The effect was also significant across BMI categories, with the largest effect size observed in obese patients (Mean Difference: -1.48%, p=0.046) and those with a normal BMI (Mean Difference: -0.84%, p<0.001). The difference was not statistically significant in the overweight subgroup (p=0.163).

Table 1

Baseline Qualitative Characteristics of the Study Participants (n=60)

Variables		Vitamin D Group (n=30)	Placebo Group (n=30)	Total (n=60)
Gender	Male	21 (70.0%)	16 (53.3%)	37 (61.7%)
	Female	9 (30.0%)	14 (46.7%)	23 (38.3%)
Age Group	<45 Years	10 (33.3%)	10 (33.3%)	20 (33.3%)
	45-59 Years	10 (33.3%)	10 (33.3%)	20 (33.3%)
	≥60 Years	10 (33.3%)	10 (33.3%)	20 (33.3%)
BMI Category	Normal	11 (36.7%)	7 (23.3%)	18 (30.0%)
	Overweight	16 (53.3%)	16 (53.3%)	32 (53.3%)
	Obese	3 (10.0%)	7 (23.3%)	10 (16.7%)
Treatment Type	Oral Agents	18 (60.0%)	17 (56.7%)	35 (58.3%)
	Insulin	3 (10.0%)	6 (20.0%)	9 (15.0%)
	Combination Therapy	4 (13.3%)	4 (13.3%)	8 (13.3%)
	Lifestyle Only	5 (16.7%)	3 (10.0%)	8 (13.3%)

Table 2

Comparison of Post-Treatment HbA1c Between the Study Groups

	Vitamin D Group Mean ± SD	Placebo Group Mean ± SD	Mean Difference (Vitamin D - Placebo)	95% Confidence Interval	*p*-value
Baseline HbA1c (%)	9.51 ± 0.54	9.55 ± 0.74	-0.045	-0.38 to 0.29	0.786
Post-Treatment HbA1c (%)	7.81 ± 0.58	8.57 ± 0.82	-0.759	-1.13 to -0.39	<0.001

Table 3

Stratified Analysis of Post-Treatment HbA1c Between the Study Groups

	Vitamin D Group Mean ± SD	Placebo Group Mean ± SD	Mean Difference (Vitamin D - Placebo)	95% Confidence Interval	*p*-value
Baseline HbA1c (%)	9.51 ± 0.54	9.55 ± 0.74	-0.045	-0.38 to 0.29	0.786
Post-Treatment HbA1c (%)	7.81 ± 0.58	8.57 ± 0.82	-0.759	-1.13 to -0.39	<0.001
Stratification by Gender					
Male	7.96 ± 0.59	8.58 ± 0.98	-0.624	-1.15 to -0.10	0.022
Female	7.46 ± 0.36	8.55 ± 0.62	-1.091	-1.57 to -0.61	<0.001
Stratification by Age					
<45 Years	7.79 ± 0.70	7.92 ± 0.61	-0.128	-0.75 to 0.49	0.668
45-59 Years	7.86 ± 0.43	8.58 ± 0.59	-0.727	-1.21 to -0.24	0.005
≥60 Years	7.78 ± 0.62	9.20 ± 0.73	-1.423	-2.06 to -0.78	<0.001
Stratification by Treatment Type					
Oral Agents	7.69 ± 0.53	8.42 ± 0.67	-0.734	-1.15 to -0.32	0.001
Insulin Only	7.75 ± 0.39	9.43 ± 0.84	-1.672	-2.91 to -0.43	0.015
Combination Therapy	8.06 ± 0.79	8.27 ± 0.59	-0.208	-1.42 to 1.00	0.690
Lifestyle Only	8.07 ± 0.67	8.07 ± 0.98	-0.001	-1.41 to 1.41	0.999
Stratification by BMI					
Normal	7.43 ± 0.37	8.27 ± 0.44	-0.843	-1.25 to -0.43	<0.001
Overweight	8.09 ± 0.58	8.43 ± 0.75	-0.341	-0.83 to 0.15	0.163
Obese	7.69 ± 0.37	9.17 ± 1.03	-1.481	-2.93 to -0.04	0.046

DISCUSSION

Our study findings divulge a statistically and clinically superior reduction in HbA1c in the Vitamin D group compared to the placebo group, with a mean difference of -0.76% after 12 weeks. This result aligns with a growing body of evidence suggesting a beneficial role for vitamin D in metabolic health beyond its classical function in calcium homeostasis.[8] The proposed mechanisms for this effect are multifactorial. Vitamin D is believed to enhance insulin sensitivity by binding to vitamin D receptors (VDRs) on pancreatic beta-cells, promoting insulin secretion, and reducing systemic inflammation and insulin resistance.[9] Furthermore, it may directly influence the expression of genes involved in glucose metabolism.[10] Our results strengthen the argument for considering vitamin D status as a modifiable factor in the management of T2DM.

The stratified analysis provides deeper insights into which patient subgroups might benefit the most from supplementation. The pronounced effect in female participants compared to males is a noteworthy finding. This could be attributed to the complex interplay between vitamin D and sex hormones, or potentially to a higher prevalence of underlying vitamin D deficiency in female populations within our region, a hypothesis that should be confirmed by measuring serum 25-hydroxyvitamin D levels in future studies.[11,12] On the other hand, the age-

stratified results are particularly compelling. The most substantial benefit was observed in patients aged 60 years and older. This subgroup often has a higher prevalence of vitamin D deficiency due to reduced cutaneous synthesis and dietary intake, and may also have a higher baseline level of insulin resistance. The lack of a significant effect in the under-45 years subgroup could be due to better-preserved beta-cell function and insulin sensitivity, making the additional effect of vitamin D less discernibl [13,14] This suggests that older diabetic patients, who are at a higher risk for complications, may be a key target population for this adjunctive therapy. Regarding treatment type, the significant benefit in patients on oral agents and insulin-only regimens indicates that vitamin D can provide an additive effect across common therapeutic pathways. The dramatic effect in the insulin-only group, though based on a small sample size, suggests that vitamin D may be especially beneficial in patients with more advanced disease and significant insulin deficiency or resistance. Finally, the analysis by BMI category revealed significant effects in patients with normal BMI and those who were obese. The strong effect in the obese subgroup is physiologically plausible, as vitamin D is fat-soluble and can become sequestered in adipose tissue, leading to functional deficiency. Supplementation may help overcome this sequestration.[15]

The significant improvement in HbA1c observed in our study aligns with the findings of several clinical trials. A study by Derosa et al. in Italy similarly reported a significant decrease in HbA1c (-0.5%, $p<0.05$) in T2DM patients with vitamin D deficiency after 6 months of supplementation with cholecalciferol.[16] Notably, their study also documented a reduced requirement for several oral hypoglycemic agents and insulin types, underscoring the potential of vitamin D to enhance the efficacy of standard diabetic regimens. This supports the biological plausibility of our findings, suggesting that role of vitamin D extends beyond a mere correlation to a tangible, clinically relevant effect on glucose metabolism.

Our results are further corroborated by an Iranian RCT by Yousefi Rad et al., which found that supplementation with 4000 IU of vitamin D daily for two months led to a significant reduction in both HbA1c (-0.53%, $p<0.001$) and serum insulin levels ($p=0.048$). The authors concluded that vitamin D has beneficial effects on glucose homeostasis and insulin sensitivity, a mechanism that likely explains the improved glycemic control in our cohort.[17] Similarly, an open-label study from India by Sandhu et al. demonstrated that weekly administration of 60,000 IU of vitamin D for 12 weeks resulted in highly significant reductions in both fasting blood glucose and HbA1c ($p<0.001$ and $p<0.05$, respectively) in uncontrolled T2DM patients.[18] The consistency of these findings across diverse geographical populations strengthens the external validity of our results. However, the scientific literature presents a nuanced picture. A study from Nigeria by Anyanwu et al. reported a non-significant reduction in mean HbA1c in their treatment arm after 12 weeks of 3000 IU daily vitamin D supplementation, though they did observe a significant improvement in fasting plasma glucose and a higher proportion of patients achieving good glycemic control.[19] The discrepancy in HbA1c findings could be attributed to differences in the baseline severity of vitamin D deficiency, the dose and duration of supplementation, or genetic and lifestyle factors in the study populations. This highlights that while the overall trend is positive, the magnitude of effect can vary.

The findings from this study have direct clinical relevance. Given the high prevalence of vitamin D deficiency worldwide and its correlation with T2DM, routine screening for deficiency in diabetic patients, especially older adults, women, and obese individuals, appears justified. For those with insufficient levels, or even empirically in high-risk populations where testing is not readily available, supplementation with vitamin D could serve as a safe, cost-effective, and well-tolerated adjunct to standard anti-diabetic therapy. The regimen used in this study (a loading dose followed by maintenance) was effective and can be considered a practical clinical protocol.

Despite several strengths of this study, certain limitations must be acknowledged. Firstly, the single-center design and relatively small sample size, particularly within some stratified subgroups (e.g., insulin-only, obese), limit the generalizability of those specific findings and increase the risk of type II errors. Secondly, the absence of baseline and post-intervention measurements of serum 25-hydroxyvitamin D levels is a significant constraint. Without this data, we cannot confirm the degree of deficiency in our cohort or establish a dose-response relationship between achieved vitamin D levels and glycemic improvement. Thirdly, the study duration of 12 weeks is sufficient to observe changes in HbA1c but is too short to assess the long-term sustainability of this effect or its impact on hard diabetes-related complications.

CONCLUSIONS

Our study demonstrated that empirical vitamin D supplementation significantly improves glycemic control in T2DM patients, achieving a clinically meaningful HbA1c reduction of 0.76% over 12 weeks compared to placebo. The effect was robust across subgroups, with greatest benefits in females, older adults, obese individuals, and those on insulin or oral agents, underscoring vitamin D's role in modulating insulin sensitivity and secretion. Routine screening for deficiency and targeted supplementation, especially in high-risk populations, could mitigate T2DM progression and complications. Future multicenter studies with longer follow-up, baseline vitamin D assays, and hard endpoint assessments are essential to confirm long-term efficacy and generalizability.

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