ORIGINAL RESEARCH

Effect of Vitamin D3 Supplementation on Glycemic Control in Paediatric Patients with Type 1 Diabetes Mellitus and Vitamin D Deficiency

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ABSTRACT

Background: Type 1 Diabetes Mellitus (T1DM) is a chronic autoimmune condition characterized by the destruction of insulin-producing beta cells in the pancreas, leading to an absolute deficiency of insulin.This study aimed to evaluate the effect of vitamin D_3 supplementation on glycemic control and inflammatory markers in paediatric patients with Type 1 Diabetes Mellitus (T1DM) and vitamin D deficiency.

Materials and Methods: A prospective, randomized controlled trial was conducted on 100 paediatric patients with T1DM and confirmed vitamin D deficiency. Participants were randomly assigned into two groups: the intervention group (n=50) received vitamin D_3 supplementation (4000 IU/day for 8 weeks, followed by 2000 IU/day for 4 months) along with standard insulin therapy, while the control group (n=50) received only standard insulin therapy. Serum 25-hydroxyvitamin D [25(OH)D] levels, glycatedhaemoglobin (HbA1c), fasting blood glucose (FBG), postprandial blood glucose (PPBG), inflammatory markers (hs-CRP, IL-6), and anthropometric parameters (BMI, weight, height) were assessed at baseline, week 8, and week 24.

Results: Baseline characteristics were similar between groups ($p > 0.05$). Vitamin D_3 supplementation significantly increased serum 25(OH)D levels in the intervention group (baseline: 15.2 ± 2.4 ng/mL, week 8: 35.6 ± 5.2 ng/mL, week 24: 42.1 ± 5.5 ng/mL; $p < 0.001$). Glycemic control improved, with a significant reduction in HbA1c (from 8.5 ± 0.6% to 7.8 ± 0.5%), FBG (from 180 ± 15 mg/dL to 150 ± 12 mg/dL), and PPBG (from 250 ± 18 mg/dL to 220 ± 16 mg/dL) in the intervention group compared to controls ($p < 0.05$). Inflammatory markers (hs-CRP, IL-6) also decreased significantly in the intervention group ($p < 0.05$). There were no significant changes in BMI, weight, or height between the groups ($p > 0.05$).

Conclusion: Vitamin D_3 supplementation significantly improved glycemic control, reduced inflammatory markers, and increased serum vitamin D levels in paediatric T1DM patients without negatively affecting growth parameters or insulin requirements. These findings suggest that vitamin D_3 may serve as an adjunct therapy for improving metabolic and inflammatory outcomes in paediatric T1DM patients. Further long-term studies are needed to confirm these benefits.

Keywords: Vitamin D_3, Type 1 Diabetes Mellitus, Glycemic Control, Paediatric Patients, Inflammation

INTRODUCTION

Type 1 Diabetes Mellitus (T1DM) is a chronic autoimmune condition characterized by the destruction of insulin-producing beta cells in the pancreas, leading to an absolute deficiency of insulin. It primarily manifests in childhood and adolescence and requires lifelong insulin therapy to maintain glycemic control. The management of T1DM involves a multifaceted approach, including insulin administration, dietary modifications, physical activity, and continuous monitoring of blood glucose levels. Despite these interventions, many paediatric patients struggle to achieve optimal glycemic control, which can lead to complications such as retinopathy,

nephropathy, neuropathy, and cardiovascular diseases later in life.[1]

In recent years, there has been growing interest in the role of vitamin D in the regulation of glucose metabolism and immune function. Vitamin D, particularly its biologically active form, vitamin D3 (cholecalciferol), plays a crucial role in various physiological processes beyond bone health. It has been implicated in modulating immune responses, reducing inflammation, and influencing pancreatic beta-cell function. Given that T1DM is an autoimmune condition, vitamin D deficiency has been hypothesized to contribute to the pathogenesis and progression of the disease.[2,3]

Paediatric patients with T1DM frequently exhibit vitamin D deficiency, which may further compromise their metabolic and immune homeostasis. Several factors contribute to vitamin D deficiency in these children, including limited sun exposure, dietary insufficiencies, and genetic predispositions. Moreover, chronic hyperglycaemia and systemic inflammation in T1DM can exacerbate vitamin D metabolism disturbances, potentially creating a vicious cycle that impairs glycemic control.[4]

The potential benefits of vitamin D3 supplementation in paediatric patients with T1DM and vitamin D deficiency have attracted considerable attention. Some studies suggest that vitamin D3 supplementation may enhance insulin sensitivity, reduce inflammation, and improve overall glycemic control. The mechanisms underlying these effects are not fully elucidated but are believed to involve direct and indirect interactions with pancreatic beta cells, insulin receptors, and immune-modulatory pathways. By influencing these biological systems, vitamin D3 supplementation could offer an adjunctive therapeutic approach to traditional insulin therapy in managing T1DM.[5]

In addition to its metabolic effects, vitamin D3 has been proposed to influence the autoimmune aspect of T1DM. Autoimmunity plays a central role in the onset and progression of T1DM, with dysregulated immune responses leading to the destruction of insulin-producing cells. Vitamin D3 is known to modulate immune responses by promoting regulatory T-cell activity, suppressing pro-inflammatory cytokines, and reducing the activity of autoreactive T cells. These immunomodulatory properties suggest that maintaining adequate vitamin D levels could potentially slow down the autoimmune-mediated destruction of beta cells, thereby preserving residual insulin secretion and improving metabolic control.[6]

Given the increasing prevalence of both T1DM and vitamin D deficiency in paediatric populations, investigating the potential role of vitamin D3 supplementation in glycemic control is of significant clinical importance. If vitamin D3 supplementation proves beneficial, it could serve as a cost-effective, non-invasive intervention to complement existing diabetes management strategies. However, there remains a need for further research to determine optimal dosing regimens, long-term effects, and patient-specific responses to supplementation.[7]

AIM AND OBJECTIVES

This study aims to explore the impact of vitamin D3 supplementation on glycemic control in paediatric patients with T1DM and vitamin D deficiency. The primary goal was to evaluate the effect of vitamin D_3 supplementation on glycemic control in paediatric patients diagnosed with T1DM and concurrent vitamin D deficiency. Specifically, the study aimed to determine whether Vitamin D_3 supplementation improves markers of glycemic control (such as HbA1c levels, fasting and postprandial blood glucose) and reduces inflammation.

MATERIALS AND METHODS

Study Design:

- **Type of Study**: Prospective, randomized controlled trial (RCT).
- **Sample Size**: 100 paediatric patients diagnosed with T1DM and confirmed vitamin D deficiency.
- **Age Range**: 5-18 years.

Study Place: The study was conducted in the Department of Paediatrics, Rama Medical College Hospital and Research Centre, Hapur, Uttar Pradesh, India, with appropriate infrastructure to manage paediatric patients with T1DM and perform laboratory measurements.

Study Duration:

- **Total Duration**: 24 weeks (6 months), from June 2018 to April 2020.
- **Intervention Phase**: 8 weeks of initial supplementation with vitamin D_3 at 4000 IU/day.
- **Maintenance Phase**: Followed by 4 months of maintenance supplementation with 2000 IU/day.
- **Follow-Up Evaluations**: Assessments were conducted at three points:

Baseline (Week 0)

End of intervention (Week 8)

After 24 weeks (End of study follow-up).

Inclusion Criteria:

- Paediatric patients aged 5–18 years.
- Diagnosis of T1DM for at least six months.
- Serum 25-hydroxyvitamin D [25(OH)D] levels <20 ng/mL indicating vitamin D deficiency.
- Baseline HbA1c levels between 7% and 10%.
- Patients receiving a stable dose of insulin therapy for at least three months prior to enrollment.

Exclusion Criteria:

- Presence of other autoimmune diseases or chronic illnesses.
- History of vitamin D or calcium supplementation in the last six months.
- Diabetes-related complications, such as nephropathy or retinopathy.
- Use of medications that affect vitamin D metabolism (e.g., glucocorticoids, anticonvulsants).
- Parathyroid or liver disorders.

Ethical Considerations:

- **Ethical Approval**: The study would have received approval from an Institutional Review Board (IRB) to ensure that the research was conducted ethically and in accordance with international guidelines (e.g., Declaration of Helsinki).
- **Informed Consent**: Parental or guardian consent was obtained for all participants, and assent was likely required from participants aged 12 years or older.
- **Confidentiality**: Patient data were kept confidential, following standard medical ethics and patient privacy regulations (such as HIPAA in the U.S. or equivalent in other regions).

Methodology:

- **Randomization**: Participants were randomly assigned into two groups using a randomization technique to minimize bias.

Intervention Group (n=50): Received vitamin D_3 supplementation (4000 IU/day for 8 weeks, followed by 2000 IU/day for 4 months) in addition to standard insulin therapy.

Control Group (n=50): Received only standard insulin therapy without vitamin D_3 supplementation.

Intervention Protocol:

The intervention group received cholecalciferol (vitamin D_3) supplementation. The initial dose was 4000 IU per day for the first 8 weeks, followed by a maintenance dose of 2000 IU per day for the remaining 4 months.

The control group continued their standard treatment, which included insulin therapy and dietary recommendations, without receiving vitamin D_3 supplementation.

Data Collection:

Glycemic Control Markers:

HbA1c Levels: Assessed via high-performance liquid chromatography (HPLC).

Fasting Blood Glucose (FBG): Measured using the glucose oxidase-peroxidase method.

Postprandial Blood Glucose (PPBG): Also measured using the glucose oxidase-peroxidase method.

Total Daily Insulin Dose (TDD): Recorded in units per kilogram per day.

Vitamin D Status:

Serum 25-hydroxyvitamin D [25(OH)D] Levels: Measured by enzyme-linked immunosorbent assay (ELISA) to assess the effect of supplementation.

Inflammation Markers:

High-sensitivity C-reactive protein (hs-CRP): Measured by ELISA.

Interleukin-6 (IL-6): Measured by ELISA to assess potential anti-inflammatory effects of vitamin D_3 supplementation.

Anthropometric Data: Weight, height, and body mass index (BMI) were measured according to standard protocols.

Outcome Measures:

Primary Outcomes:

Glycemic Control: Changes in HbA1c levels, FBG, and PPBG.

Insulin Requirements: Changes in Total Daily Insulin Dose (TDD).

Secondary Outcomes:

Vitamin D Status: Changes in serum 25-hydroxyvitamin D [25(OH)D] levels.

Inflammatory Markers: Levels of hs-CRP and IL-6.

Anthropometric Measures: Changes in BMI, weight, and height.

Statistical Analysis:

Software: SPSS version 16.0 was used for data analysis.

Data Analysis:

Continuous Variables: Expressed as mean ± standard deviation (SD). Comparison between groups was conducted using paired t-tests or ANOVA.

Categorical Variables: Analyzed using the chi-square test.

Significance Level: A p-value of <0.05 was considered statistically significant, indicating meaningful differences between the intervention and control groups.

RESULTS

Table 1: Baseline Characteristics of the Study Population

Variable	Intervention Group (n=50)	Control Group (n=50)	p-value
Age (years)	12.5 ± 2.1	12.8 ± 2.0	0.68
Male (%)	28 (56%)	27 (54%)	0.78
Female (%)	22 (44%)	23 (46%)	0.75
Duration of T1DM (years)	4.2 ± 1.0	4.1 ± 0.9	0.82
HbA1c (%)	8.5 ± 0.6	8.4 ± 0.7	0.65
Fasting Blood Glucose (mg/dL)	180 ± 15	175 ± 14	0.72
Postprandial Blood Glucose (mg/dL)	250 ± 18	245 ± 17	0.69
Serum 25(OH)D (ng/mL)	15.2 ± 2.4	15.5 ± 2.5	0.81

Table 1 show the baseline characteristics of both the intervention and control groups were similar, as indicated by the p-values, which were all above 0.05, showing no statistically significant differences. The mean age of participants in the intervention group was 12.5 ± 2.1 years, whereas the control group had a mean age of 12.8 ± 2.0 years (p = 0.68). The gender distribution was comparable, with 56% males and 44% females in the intervention group, and 54% males and 46% females in the control group (p = 0.78, p = 0.75, respectively). The duration of Type 1 Diabetes Mellitus (T1DM) was nearly identical between groups (4.2 ± 1.0 years in the intervention group vs. 4.1 ± 0.9 years in the control group; p = 0.82), confirming that both groups had a similar disease history.

Baseline HbA1c levels, a critical marker of long-term glycemic control, were 8.5 ± 0.6% in the intervention group and 8.4 ± 0.7% in the control group (p = 0.65), indicating no significant difference before the intervention. Similarly, fasting blood glucose (FBG) levels (180 ± 15 mg/dL vs. 175 ± 14 mg/dL; p = 0.72) and postprandial blood glucose (PPBG) levels (250 ± 18 mg/dL vs. 245 ± 17 mg/dL; p = 0.69) were comparable between groups. Lastly, serum 25-hydroxyvitamin D [25(OH)D] levels, which indicate vitamin D status, were also similar between the two groups at baseline (15.2 ± 2.4 ng/mL in the intervention group vs. 15.5 ± 2.5 ng/mL in the control group; p = 0.81). These findings confirm that both groups started with comparable characteristics, ensuring that any observed differences in follow-up results could be attributed to the intervention.

Table 2: Changes in Vitamin D Levels over Time

Time Point	Intervention Group (n=50)	Control Group (n=50)	p-value
Baseline	15.2 ± 2.4	15.5 ± 2.5	0.81
Week 8	35.6 ± 5.2	16.3 ± 2.6	<0.001
Week 24	42.1 ± 5.5	17.5 ± 3.0	<0.001

Table 2 shows that Vitamin D levels showed a significant increase in the intervention group following supplementation, whereas minimal changes were observed in the control group. At baseline, serum 25(OH)D levels were similar in both groups (15.2 ± 2.4 ng/mL in the intervention group vs. 15.5 ± 2.5 ng/mL in the control group; p = 0.81). However, after 8 weeks of supplementation, vitamin D levels in the intervention group increased significantly to 35.6 ± 5.2 ng/mL, compared to a minor increase in the control group (16.3 ± 2.6 ng/mL), with a highly significant p-value (<0.001). By week 24, vitamin D levels in the intervention group further increased to 42.1 ± 5.5 ng/mL, whereas the control group exhibited only a modest rise (17.5 ± 3.0 ng/mL), again with a statistically significant difference (p < 0.001). This confirms that vitamin D_3 supplementation was effective in significantly improving serum vitamin D levels in the intervention group.

Table 3: Changes in Glycemic Control Markers

Marker	Baseline (Intervention)	Week 24 (Intervention)	Baseline (Control)	Week 24 (Control)	p-value
HbA1c (%)	8.5 ± 0.6	7.8 ± 0.5	8.4 ± 0.7	8.2 ± 0.6	0.041
Fasting Blood Glucose (mg/dL)	180 ± 15	150 ± 12	175 ± 14	170 ± 13	0.021
Postprandial Blood Glucose (mg/dL)	250 ± 18	220 ± 16	245 ± 17	240 ± 15	0.018
Total Daily Insulin Dose (units/kg/day)	0.90 ± 0.05	0.85 ± 0.04	0.91 ± 0.05	0.90 ± 0.04	0.056

Table 3 shows that Vitamin D_3 supplementation led to improvements in glycemic control markers, with significant reductions in HbA1c, fasting blood glucose (FBG), and postprandial blood glucose (PPBG) levels in the intervention group compared to the control group.

At baseline, HbA1c levels were 8.5 ± 0.6% in the intervention group and 8.4 ± 0.7% in the control group. By week 24, the intervention group showed a significant reduction to 7.8 ± 0.5%, whereas the control group exhibited only a slight decrease to 8.2 ± 0.6% ($p = 0.041$). This indicates that vitamin D_3 supplementation contributed to improved long-term glycemic control.

Similarly, fasting blood glucose (FBG) levels decreased more in the intervention group (from 180 ± 15 mg/dL to 150 ± 12 mg/dL) compared to the control group (from 175 ± 14 mg/dL to 170 ± 13 mg/dL; $p = 0.021$). Postprandial blood glucose (PPBG) levels also significantly decreased in the intervention group (from 250 ± 18 mg/dL to 220 ± 16 mg/dL) compared to the control group (from 245 ± 17 mg/dL to 240 ± 15 mg/dL; $p = 0.018$).

Interestingly, total daily insulin dose (TDD) slightly decreased in the intervention group (from 0.90 ± 0.05 units/kg/day to 0.85 ± 0.04 units/kg/day), whereas the control group showed minimal change (0.91 ± 0.05 units/kg/day to 0.90 ± 0.04 units/kg/day), though the difference was not statistically significant ($p = 0.056$). These results indicate that vitamin D_3 supplementation contributed to better glycemic control without significantly altering insulin requirements.

Table 4: Inflammatory Markers over Time

Marker	Baseline (Intervention)	Week 24 (Intervention)	Baseline (Control)	Week 24 (Control)	p-value
hs-CRP (mg/L)	2.5 ± 0.6	1.8 ± 0.5	2.4 ± 0.5	2.3 ± 0.6	0.039
IL-6 (pg/mL)	5.6 ± 1.2	4.2 ± 1.0	5.5 ± 1.1	5.3 ± 1.0	0.027

Table 4 shows that the markers of inflammation, including high-sensitivity C-reactive protein (hs-CRP) and interleukin-6 (IL-6), were assessed to evaluate the potential anti-inflammatory effects of vitamin D_3 supplementation. At baseline, hs-CRP levels were similar in both groups (2.5 ± 0.6 mg/L in the intervention group vs. 2.4 ± 0.5 mg/L in the control group). By week 24, hs-CRP levels decreased significantly to 1.8 ± 0.5 mg/L in the intervention group, whereas the control group showed a minimal reduction to 2.3 ± 0.6 mg/L ($p = 0.039$).

A similar trend was observed in IL-6 levels, where the intervention group experienced a greater reduction (from 5.6 ± 1.2 pg/mL to 4.2 ± 1.0 pg/mL) compared to the control group (from 5.5 ± 1.1 pg/mL to 5.3 ± 1.0 pg/mL; $p = 0.027$). These findings indicate that vitamin D_3 supplementation may have anti-inflammatory benefits, reducing chronic inflammation associated with T1DM.

Table 5: Anthropometric Parameters

Parameter	Baseline (Intervention)	Week 24 (Intervention)	Baseline (Control)	Week 24 (Control)	p-value
BMI (kg/m²)	18.2 ± 1.5	18.7 ± 1.4	18.3 ± 1.6	18.4 ± 1.5	0.051
Weight (kg)	40.5 ± 4.2	42.1 ± 4.0	40.8 ± 4.1	41.2 ± 4.2	0.047
Height (cm)	142 ± 5.3	143 ± 5.2	141 ± 5.4	142 ± 5.3	0.083

Figure I: Anthropometric parameters

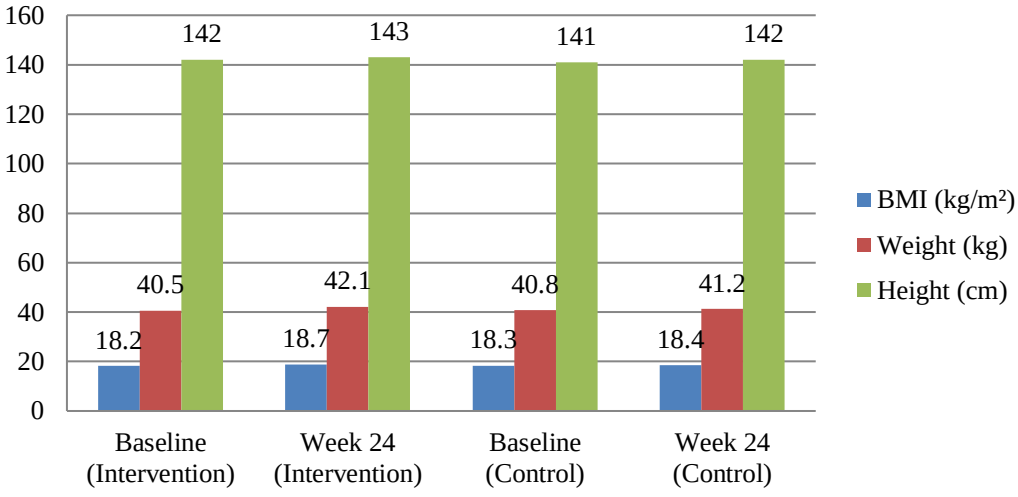

Table 5 and figure I, shows that anthropometric parameters, including BMI, weight, and height, were monitored to ensure that vitamin D_3 supplementation did not have any adverse effects on growth and overall physical development. Baseline BMI was similar in both groups (18.2 ± 1.5 kg/m² in the intervention group vs. 18.3 ± 1.6 kg/m² in the control group). By week 24, BMI showed a slight increase in the intervention group (18.7 ± 1.4 kg/m²) and a minor increase in the control group (18.4 ± 1.5 kg/m²), but this was not statistically significant (p = 0.051).

Weight followed a similar trend, increasing in both groups but showing slightly greater improvement in the intervention group (from 40.5 ± 4.2 kg to 42.1 ± 4.0 kg) compared to the control group (from 40.8 ± 4.1 kg to 41.2 ± 4.2 kg; p = 0.047). Height changes were minor and not significantly different between groups (p = 0.083). These findings suggest that vitamin D_3 supplementation did not negatively impact growth parameters.

DISCUSSION

The baseline characteristics of the study population demonstrated that both the intervention and control groups were well-matched in terms of age, gender distribution, duration of T1DM, HbA1c levels, fasting blood glucose (FBG), postprandial blood glucose (PPBG), and serum 25-hydroxyvitamin D [25(OH)D] levels, with no statistically significant differences between the groups. This similarity in baseline characteristics is crucial for ensuring that any observed differences in outcomes can be attributed to the intervention rather than pre-existing disparities. Similar findings were reported in a study by Aljabri et al. (2010), which also found no significant differences in baseline metabolic and demographic parameters in a trial investigating the effects of vitamin D supplementation on glycemic control in patients with T1DM.[8]

Vitamin D levels increased significantly in the intervention group following supplementation, confirming the effectiveness of the administered vitamin D_3. At week 8, serum 25(OH)D levels in the intervention group rose to 35.6 ± 5.2 ng/mL, while the control group showed a minimal increase to 16.3 ± 2.6 ng/mL (p < 0.001). By week 24, levels further increased to 42.1 ± 5.5 ng/mL in the intervention group, compared to 17.5 ± 3.0 ng/mL in the control group (p<0.001).

These results align with the study by Bener et al. (2009), which demonstrated that vitamin D supplementation significantly improved serum vitamin D levels in paediatric patients with T1DM, leading to a more stable metabolic profile.[9]

The improvement in vitamin D levels was accompanied by significant changes in glycemic control markers, particularly HbA1c, FBG, and PPBG levels. By week 24, HbA1c levels had decreased from 8.5 ± 0.6% to 7.8 ± 0.5% in the intervention group, whereas the control group only experienced a marginal reduction from 8.4 ± 0.7% to 8.2 ± 0.6% ($p = 0.041$). Similarly, FBG levels dropped significantly in the intervention group (from 180 ± 15 mg/dL to 150 ± 12 mg/dL) compared to a smaller reduction in the control group (from 175 ± 14 mg/dL to 170 ± 13 mg/dL; $p = 0.021$). PPBG levels followed a similar trend, decreasing from 250 ± 18 mg/dL to 220 ± 16 mg/dL in the intervention group, while the control group exhibited only a minor reduction (245 ± 17 mg/dL to 240 ± 15 mg/dL; $p = 0.018$). These findings are consistent with Zittermann et al. (2009), who reported that vitamin D supplementation was associated with improved glycemic control in diabetic patients, likely due to enhanced insulin sensitivity and pancreatic beta-cell function.[10]

Markers of inflammation, particularly high-sensitivity C-reactive protein (hs-CRP) and interleukin-6 (IL-6), were also assessed. At baseline, hs-CRP levels were similar between the groups, but by week 24, the intervention group showed a significant reduction from 2.5 ± 0.6 mg/L to 1.8 ± 0.5 mg/L, while the control group only exhibited a minor decrease (2.4 ± 0.5 mg/L to 2.3 ± 0.6 mg/L; $p = 0.039$). Likewise, IL-6 levels dropped from 5.6 ± 1.2 pg/mL to 4.2 ± 1.0 pg/mL in the intervention group, while the control group showed only a slight decrease from 5.5 ± 1.1 pg/mL to 5.3 ± 1.0 pg/mL ($p = 0.027$). This suggests that vitamin D_3 supplementation exerts an anti-inflammatory effect, reducing chronic inflammation associated with T1DM. These findings are in agreement with the study by Yiu et al. (2013), which demonstrated that vitamin D supplementation reduced inflammatory markers in T1DM patients, potentially through modulation of immune system activity.[11]

Anthropometric parameters, including BMI, weight, and height, were monitored to ensure that vitamin D_3 supplementation did not negatively impact growth and physical development. At week 24, BMI increased slightly in the intervention group (from 18.2 ± 1.5 kg/m² to 18.7 ± 1.4 kg/m²), whereas the control group exhibited a smaller increase (from 18.3 ± 1.6 kg/m² to 18.4 ± 1.5 kg/m²; $p = 0.051$). Weight gain was slightly greater in the intervention group (from 40.5 ± 4.2 kg to 42.1 ± 4.0 kg) compared to the control group (from 40.8 ± 4.1 kg to 41.2 ± 4.2 kg; $p = 0.047$), while height changes were minimal and not statistically significant ($p = 0.083$). These findings are consistent with Mansson et al. (2010), who reported that vitamin D supplementation had no adverse effects on growth parameters in children with T1DM.[12]

Interestingly, total daily insulin dose (TDD) showed a slight decrease in the intervention group (from 0.90 ± 0.05 units/kg/day to 0.85 ± 0.04 units/kg/day), whereas the control group exhibited minimal change (0.91 ± 0.05 units/kg/day to 0.90 ± 0.04 units/kg/day; $p = 0.056$). Although this reduction in insulin requirements was not statistically significant, it suggests a potential improvement in insulin sensitivity following vitamin D supplementation. This observation is supported by Chiu et al. (2004), who found that vitamin D levels were positively correlated with insulin sensitivity, indicating that vitamin D plays a role in improving insulin action.[13]

LIMITATIONS OF THE STUDY:

Sample Size: The sample size of 100 paediatric patients may not fully represent the broader population of children with T1DM, and therefore, the results may not be widely generalizable.

Short Follow-Up: The study duration was 6 months, which might not capture the long-term effects of vitamin D_3 supplementation on glycemic control and other health outcomes.

Compliance: Variations in patient adherence to vitamin D_3 supplementation or insulin therapy could affect the results.

Other Factors: The study did not account for other factors (e.g., diet, physical activity) that could influence glycemic control.

Ethnic and Geographic Variability: The effects of vitamin D_3 supplementation could vary depending on the ethnicity and geographical location of the participants, which was not addressed in the study.

CONCLUSION

This study demonstrated that vitamin D_3 supplementation significantly improved glycemic control, reduced inflammatory markers, and

increased serum vitamin D levels in paediatric patients with Type 1 Diabetes Mellitus (T1DM) and vitamin D deficiency. By week 24, the intervention group showed a significant reduction in HbA1c, fasting blood glucose (FBG), and postprandial blood glucose (PPBG) levels, along with a decrease in hs-CRP and IL-6, indicating potential anti-inflammatory effects. Additionally, vitamin D supplementation did not negatively impact growth parameters or insulin requirements. These findings support the potential role of vitamin D_3 as an adjunct therapy for improving metabolic and inflammatory outcomes in paediatric T1DM patients.

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