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Correlation of Vitamin D Level with Body Physique and Glycemic Control in Type1 Diabetic Egyptian Children and Adolescents

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Vitamin D is increasingly recognized for its immunomodulatory and metabolic effects, with growing evidence linking deficiency to the development and progression of type 1 diabetes mellitus (T1DM).We aimed To evaluate vitamin D status among Egyptian T1D pediatric patients and explore its relationship with glycemic control and body physique. A cross-sectional study was conducted on pediatric patients with T1DM. Serum 25-hydroxyvitamin D [25(OH)D] levels were assessed and categorized as deficient, insufficient, or sufficient. Clinical, anthropometric and biochemical parameters, including glycated hemoglobin (HbA1c), were assessed to determine associations with vitamin D status. Body composition was also analyzed. Vitamin D deficiency was highly prevalent, with the majority of patients demonstrating either deficient or insufficient levels. Lower VD levels were significantly associated with poorer glycemic control and higher BMI and body fat. There was no direct correlation between VD and HbA1c. We concluded that Vitamin D deficiency is common in Egyptian diabetic pediatric patients and is associated with adverse glycemic outcomes and higher BMI and body fat. Routine screening and appropriate supplementation may represent a valuable adjunct in disease management, although further future researches are required to confirm causality and establish evidence-based supplementation strategies.

Keywords: Adiposity; Body composition; Glycemic control; Type 1 diabetes mellitus; Vitamin D; Vitamin D deficiency.

Type 1 diabetes mellitus (T1DM) is a common chronic autoimmune disease of childhood and adolescence, caused by damage of â-cells of the pancreas and leading to incomplete or complete deficiency of insulin, which could lead to severe complications if untreated.[1] It accounts for 5–10% of all diabetes cases and about 90% of diabetic pediatric patients.[2] Globally, more than 1.1 million children and adolescents

(0–19 years) live with T1DM, with about 128,900 new cases diagnosed annually and an incidence increase of nearly 3% per year.[3] Onset most frequently occurs in childhood (ages 4–6) and early puberty (10–14 years), with a peak at 13–14 years.[4]

Most individuals with T1DM are lean, though some may present with higher weight.[5] The condition is influenced by genetic susceptibility, environmental triggers, and adaptive immune dysfunction, with chronic inflammation and progressive â-cell loss causing impaired insulin secretion.[6] Optimizing glycemic control is particularly challenging in children; the Diabetes Control and Complications Trial (DCCT) showed that normalization of glucose levels in adolescents is more difficult than in adults.[7]

Although children with T1DM have traditionally tended to be underweight, sedentary lifestyles and increased consumption of energy-dense foods have led to higher rates of overweight and obesity. Excess adiposity, especially visceral fat, and reduced lean mass are associated with poorer glycemic control, adverse lipid profiles, systemic inflammation, and higher risks of vascular complications. [8,9] Intensive insulin therapy, while crucial, may contribute to weight gain if not balanced with proper diet and activity, with body mass increase largely reflecting fat accumulation.[10]

BMI alone cannot distinguish between fat and lean tissue and therefore cannot clarify whether overweight and obesity in children with T1DM reflect true increases in adiposity.[11] This is especially concerning during growth and development.[12] Few studies have comprehensively examined fat and lean mass in pediatric T1DM, or the role of HbA1c and disease-related factors in body composition. [13] Glycated hemoglobin remains the standard marker of glycemic control, strongly linked to long-term complication risk.[14] Moreover, regional fat distribution may influence insulin sensitivity, lipids, adipokines, and inflammation differently. [15]

Vitamin D, a fat-soluble steroid hormone, is essential for calcium and bone homeostasis but also exerts wider effects due to receptors expressed in pancreatic â-cells and immune cells.[16] Beyond skeletal health, vitamin D regulates immunity, reduces inflammation, and modulates glucose metabolism. Deficiency is a global health problem across all ages and sexes, [17] particularly in the Middle East. [18]

In recent years, vitamin D's extra-skeletal roles have attracted attention. Its receptors in â-cells and immune cells suggest potential involvement in T1DM pathogenesis.[19] Evidence indicates that vitamin D modulates autoimmunity, enhances insulin secretion and sensitivity, and reduces complications.[20] Supplementation in infancy has been linked to reduced risk of developing T1DM.[21] Vitamin D also supports glycemic control by inhibiting inflammation, promoting insulin synthesis, and improving sensitivity.[22]

Vitamin D deficiency is highly prevalent in Egyptian pediatric population. In the general adolescent population, 94.8% were deficient and 4.2% insufficient.[23] Among children with T1DM, deficiency rates were 84.9% versus 15.1% insufficiency, [24] while a more recent study reported 91.7% deficiency. [25]

Overall, vitamin D deficiency is closely linked to T1DM occurrence, progression, and complications.[26] It has been associated with ketoacidosis at onset, [27] poor glycemic control, [28] increased insulin needs, reduced sensitivity, higher fasting glucose, and elevated HbA1c. [29] Vitamin D also protects â-cells from immune attack, [30] and modulates T-cell differentiation and cytokine production, promoting anti-inflammatory pathways.[31] Despite its importance, deficiency remains underestimated, highlighting the need for routine screening and supplementation in pediatric T1DM. [19]

Finally, vitamin D deficiency has been inversely associated with obesity measures such as BMI, fat mass, and waist-to-hip ratio.[32,33] Potential explanations include volumetric dilution, diminished hepatic hydroxylation, impaired cutaneous synthesis, decreased exposure to the sun, and altered receptor expression in adipose tissue. [34]

Despite conflicting evidence, vitamin D deficiency remains a major concern in T1DM progression and prognosis. Data in Egyptian children with T1DM are scarce; therefore, we thought to investigate vitamin D state and its relation to body composition and glycemic control among the pediatric population. Therefore, the aim of this work was to: (i) assess vitamin D deficiency and insufficiency prevalence among T1DM pediatric patients (ii) identify factors associated with its deficiency, and (iii) examine associations between VD, body adiposity, and glycemic control among these patients.

MATERIALS AND METHODS

Ethical approval

The study was approved by the National Research Centre Ethical Committee (Approval 16390). Written informed consents were obtained after explaining the study to the children's parents or care givers.

Study design and settings

This present study was a cross-sectional study comprised 62 children with T1DM of both sexes aged 7-18 y. Patients were enrolled from the Diabetic Endocrine Metabolic Pediatric Unit outpatient clinic of Abu El-Rish Hospital Cairo University during the time from January 2018 – December 2018.

Participants

62 cases aged 7-18y already diagnosed with Type1 Diabetes and receiving insulin treatment > 1 year were enrolled. The diagnosis of T1DM was performed according to the International Society for Pediatric and Adolescent Diabetes (ISPAD) criteria.[35] Only those not on vitamin D supplementation in the past 6 months were included.

Children with chronic diseases, e.g. hepatic, hyperparathyroidism, Celiac disease or receiving any drugs that can affect VD metabolism such as; phenytoin, phenobarbital, and thyroid hormone were excluded from the study. Children with micro- or macro-vascular complications, patients having serious pathological risk factors accompanying deficiency of vitamin D, like; osteogenesis imperfecta, renal affection or cystic fibrosis were not included.

The demographic and clinical data including onset of diabetes, insulin requirement (IU/Kg/day) were collected from medical records.

Each child was subjected to:

Complete clinical examination

Anthropometric examination

Confirming that the child wore minimal clothes and took off shoes, measures including weight assessed via a Seca scale (Seca Balance Beam Scale Model 700, seca deutschland Medical Scales and Measuring Systems; seca GmBH&Co. KG, Hamburg, Germany) to the nearest 0.01 kg, height assessed via Holtain stadiometer (The Harpenden Portable Stadiometer; Holtain Ltd, Cross well Pembrokeshire Wales, UK) to the nearest 0.1 cm. Mid-upper arm, waist, and hip circumferences assessed via non-elastic measuring tape to the nearest 0.1cm. Skinfold thicknesses were measured including; Biceps, triceps, suprailiac and subscapular skinfolds using Holtain skinfold caliper to the nearest 0.1 mm. The mean of three successive measurements was recorded. The instructions of the International Biological Program were followed.[36] Some indices were calculated as BMI according to the following equation: BMI = weight (kg)/height (m^2) and waist/hip ratio.

- Fat % was estimated via body composition analyzer (Computerized Holtain Body Composition Analyzer, Holtain's BCA, Crosswell No.646512; Holtain Ltd, Wales, UK).
- The children's nutritional status was assessed, conferring to the BMI-for-age Z-scores proposed by the WHO. [37] According to the WHO Growth Reference data for 5-19; BMI was classified as :
- Normal: BMI for age z- score of < +1SD to > - 2SD
- Overweight: BMI for age z- score of > +1SD
- Obesity: BMI for age z- score of > +2 SD
- Thinness: BMI for age z- score of <"2SD.
- Severe thinness: BMI for age z- score of <"3SD.

Laboratory investigations

5mL of blood were withdrawn from our cases and transferred within 1–2 h of collection time to the Clinical and Chemical pathology laboratory at the Medical Research center of Excellence at the National Research Centre for processing: part of the blood samples was used immediately for:

HbA1c

Glycated Hb (HbA1c) was done using nephelometric assay on mispa I2 from AGAPPE.

Glycemic control was determined as follows: HbA1c <7% ('good' control). The HbA1c concentration ranged from 7 to 8% was considered as 'inadequate' control and 'poor' at concentrations more than 8%.[38]

The remaining blood samples were centrifuged and sera were separated and stored at "80C freezer for further evaluation of VD.

25(OH) vitamin D

Serum 25-hydroxyvitamin D [25(OH) D] is the standard indicator of VD status.[39] Concentrations of 25(OH) D were assayed by chemiluminescence immunoassay on Cobas e 411 (Roche).

Vitamin D deficiency threshold was 12 ng/mL and insufficiency level was 12-20 ng/mL. 25-OHD serum concentrations > 20 ng/mL were defined as vitamin D sufficiency.[40]

Statistical analysis

Data were analyzed using the statistical package for social sciences, version 23.0 (SPSS Inc., Chicago, Illinois, USA). The normality of Data was examined using Kolmogorov-Smirnov and Shapiro-Wilk Test. The quantitative data were expressed as mean± SD when they were normally distributed and parametric tests were used, whereas data with non-normal distribution (non-parametric data) were expressed as median with inter-quartile range (IQR). Qualitative variables were expressed as number and percentages. The subsequent tests were used: *Independent-samples t-test* to compare between two means & *Mann Whitney U test* to compare two-groups with non-parametric data. *Chi-square test* and *Fisher's exact test* were used to compare groups with qualitative data. Correlation was tested by Spearman's correlation tests. $P < 0.05$ was considered significant.

Table 1. Z BMI distribution among the total studied patients.

Z BMI	Total (n=62)
Normal (< +1SD to > - 2SD)	53 (85.5%)
Overweight (> +1SD)	7 (11.3%)
Obese (> +2 SD)	2 (3.2%)
Total	62 (100%)

RESULTS

Demographic data

This study included 62 children with type 1 diabetes (T1D), comprising 34 (54.8%) females and 28 (45.2%) males, with a mean age of 10.88 ± 2.66 y (range: 6.9–17.4 y). The duration of diabetes ranged from 0 to 10 y, with a median of 2 years (IQR: 0.75–5).

Anthropometric Characteristics

The median BMI Z-score was –0.13 (IQR: –0.58 to 0.44; range: –1.56 to 2.46 kg/m^2), while the mean body fat percentage was 22.00 ± 6.14%. Most participants (85.5%) had a normal BMI Z-score (< +1SD to > –2SD), whereas 11.3% were overweight and 3.2% were obese (Table 1).

Vitamin D status

Serum vitamin D levels ranged between 1.4 and 15.2 ng/mL, with a median

Table 2. Vitamin D level distribution and glycemic control among the total studied patients

Vitamin D level (ng/ml)	No.	%
Deficiency	56	90.3%
Insufficiency	6	9.7%
Total	62	100.0%
HbA1C Level	No.	%
Poor	42	67.7%
Inadequate	12	19.4%
Good Control	8	12.9%
Total	62	100%

of 5.8 ng/mL (IQR: 3.78–9.6). The majority (90.3%) had vitamin D deficiency (mean = 6.15 ± 2.85 ng/mL), while 9.7% had vitamin D insufficiency (mean = 14.05 ± 1.00 ng/mL). None had sufficient vitamin D levels (> 20 ng/mL), table (2). Vitamin D concentrations were normally distributed (Kolmogorov–Smirnov p > 0.05), figure (1). No significant sex difference was detected in vitamin D levels (males: 6.23 ± 3.35 ng/mL; females: 7.47 ± 3.75 ng/mL; p = 0.177).

The normality of Data was examined via Kolmogorov-Smirnov test of normality. The results of Kolmogorov-Smirnov test demonstrated that VD level had normal distribution among the study group (parametric data).

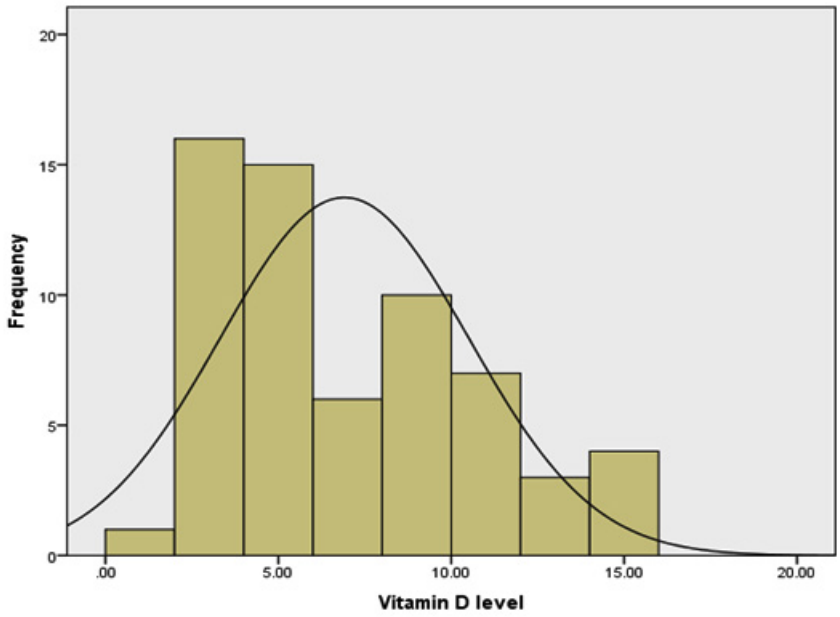

Fig. 1. Vit D levels distribution among study group

Table 3. Comparison of demographic parameters and Z BMI according to vitamin D distribution

Characteristics	Level of Vit. D		p-value
	Deficiency N= 56	Insufficiency N=6	
Sex			
Male	27 (48.2%)	1 (16.7%)	0.140
Female	29 (51.8%)	5 (83.3%)	
Duration	2.5 (0.77 - 5.50)	1.25 (0.15 - 2.75)	0.169
Age (years)	11.05 ± 2.63	9.33 ± 2.67	0.135
BMI			
Normal	49 (87.5%)	4(66.7%)	<0.001*
Overweight	5 (8.9%)	2 (33.3%)	
Obese	2 (3.6%)	0 (0.0%)	

IQR: Interquartile range, Using: U=Mann-Whitney test for Non-parametric data "Median (IQR)"; t-Independent Sample t-test for Mean±SD; x2: Chi-square test for Number (%) or Fisher's exact test, when appropriate. *p-value <0.05 is significant.

Comparison by Vitamin D Status

Children with vitamin D deficiency had significantly higher BMI ($p < 0.001$), higher body fat % ($p = 0.024$), and lower height Z-scores ($p = 0.016$) than those with insufficiency (Tables 3, 4). No significant differences were found in age, diabetes duration, or waist/hip indices ($p > 0.05$).

Glycemic Control

The mean HbA1c among all patients was 8.62 ± 1.58 % (range = 5.5–12). Most participants (67.7%) had poor glycemic control (HbA1c > 8%), while 12.9% achieved good control (HbA1c < 7%) (Table 2).

Vitamin D deficiency was present in 85.7% of patients with poor glycemic control and in all patients with good control. Although mean HbA1c was higher among vitamin D–deficient patients (9.70 ± 1.28%) than among those with insufficiency (8.50 ± 1.58%), the difference was not statistically significant ($p = 0.077$), table (5).

The duration of diabetes was significantly longer in those with poor glycemic control (median = 3.00 years; IQR: 1.15–5.71) than in those with inadequate (0.88 years; $p < 0.05$) or good control (1.04 years; $p < 0.05$), table (6).

Table 4. Comparing the anthropometric measurements according to vitamin D distribution

Anthropometric measurements	Level of Vit. D		p-value
	Deficiency	Insufficiency	
ZWt	-0.44 (-0.84 - 0.28)	0.03 (- 0.48 - 0.64)	0.213
ZHt	- 0.62 (-1.39 - 0.10)	- 0.56 (-5.32 - 0.43)	0.016*
Z BMI	0.52 (- 0.61 - 2.22)	- 0.14 (- 0.58- 0.30)	0.024*
ZMUAC	-0.36(-0.91- 0.17)	0.37(-0.80 - 0.98)	0.185
Waist C	66.61 ± 7.92	66.33 ± 11.54	0.939
Hip C	77.54 ± 14.55	77.17 ± 12.92	0.952
Waist/Hip	0.85 (0.81 - 0.91)	0.88 (0.81 - 0.89)	0.760
MUAC	21.50 ± 3.82	21.49 ± 3.66	0.994
Biceps	7.75 (5.13 - 13.50)	7.00 (6.00 - 9.88)	0.724
Triceps	15.00 (9.75 - 25.63)	15.00(11.13-19.38)	0.478
SubscapSF	11.00 (6.75 - 23.38)	11.00 (7.13 - 14.75)	0.478
SupraIlSF	10.25 (7.00 - 14.00)	7.50 (6.00 - 21.63)	0.679
Fat %	27.33 ± 7.04	21.43 ± 5.82	0.024*

IQR: Interquartile range, Using: U=Mann-Whitney test for Non-parametric data "Median (IQR)"; t-Independent Sample t-test for Mean±SD; *p-value <0.05 is significan

Table 5. Comparison of laboratory findings according to vitamin D distribution

Laboratory data	Level of Vit. D		p-value
	Deficiency N=56	Insufficiency N=6	
Vitamin D level(ng/ml)	6.15±2.85	14.05±1.00	<0.001**
HbA1C (gm%)	9.70±1.28	8.50±1.58	0.077
HbA1C Level			
Poor	36 (64.3%)	6 (100 %)	0.206
Inadequate	12 (21.4%)	0(0.0%)	
Good Control	8 (14.3%)	0 (0.0%)	

Using: t-Independent Sample t-test for Mean±SD; x2: Chi-square test for Number (%) or Fisher's exact test, when appropriate. *p-value <0.05 is significant.

Anthropometry and Glycemic Control

Hip circumference was significantly lower among patients with good glycemic control compared to those with inadequate or poor control ($p = 0.008$), whereas waist-to-hip ratio was significantly higher ($p = 0.016$). No significant differences were observed in BMI Z-scores or body fat % ($p > 0.05$), Table (7).

Furthermore, there was no significant difference in vitamin D levels between the three groups of glycemic control ($p > 0.05$). While, there was a significant difference in HbA1c level between the three groups ($p = 0.001$), table (8).

Correlation Analysis

Vitamin D levels showed no significant correlation with anthropometric

Table 6. Comparison of demographic parameters and Z BMI according to glycemic control

Characteristics	HbA1C Level			P value
	Poor N=42	Inadequate N=12	Good Control N=8	
Sex				
Male	18 (42.9%)	7 (58.3%)	3 (37.5%)	0.571
Female	24 (57.1%)	5 (41.7%)	5 (62.5%)	
Duration of diabetes (years)	3.00 (1.15- 5.71) A	0.88 (0.35 - 1.75) B	1.04 (0.56 - 4.92) B	0.049*
Age (years)	11.17±2.85	10.49±2.32	9.94±2.04	0.421
ZBMI classification				
Normal	37 (88.1%)	10 (83.3%)	6 (75.0%)	0.611
Overweight & Obese	5 (11.9%)	2 (16.7%)	2 (25.0%)	

IQR: Interquartile range, Using: One way Analysis of Variance test was performed for Mean±SD & Multiple comparison between groups through Post Hoc test: Tukey's test, Kruskal–Wallis was performed for Median (IQR) & Multiple comparison between groups through Mann-Whitney test, x2: Chi-square test for Number (%) or Fisher's exact test, when appropriate. Different capital letters indicate significant difference at ($p<0.05$) among means in the same row. *p-value <0.05 is significant.

Table 7. Comparison of anthropometric measurements according to the glycemic control

Anthropometric measurements	HbA1C Level			P value
	Poor N=42	Inadequate N=12	Good Control N=8	
zWt	-0.23(-0.80- 0.31)	-0.65(-0.87 - - 0.38)	-0.55 (-0.64 - 0.67)	0.586
zHt	-0.52(-1.23- 0.19)	-1.35(-2.15 - - 0.60)	-0.47(-0.71 - 0.06)	0.904
Waist C	67.19±7.92	64.71±10.03	66.19±7.33	0.654
Hip C	80.55±11.48 A	75.92±10.09 B	63.94±24.11 C	0.008*
zMUAC	-0.24(-0.84- 0.30)	-0.60 (-1.11 - 0.20)	-0.24(-0.90 - 0.94)	0.708
Biceps	7.00(6.00- 10.15)	6.25(5.00 - 11.25)	8.25(6.25 - 10.55)	0.688
Triceps	14.50(11.00-19.25)	15.25(12.00 - 19.88)	14.75(9.88 - 21.30)	0.969
SubscapSF	11.00(7.75- 15.70)	9.50(7.00 - 15.25)	11.50(7.25 - 14.00)	0.534
SupraIlSF	11.00(7.00- 14.00)	8.60(6.05 - 13.00)	10.00(6.65 - 15.25)	0.484
zBMI	- 0.08 (- 0.57- 0.57)	- 0.34 (-0.56 - 0.02)	-0.10 (- 0.60 - 0.89)	0.727
Waist/Hip	0.84 (0.80- 0.88) B	0.90 (0.83 - 0.93) A	0.91(0.84 - 1.23) A	0.016*
Fat Percent	22.45±6.74	20.45±3.57	22.01±6.06	0.618

IQR: Interquartile range,Using: One way Analysis of Variance (ANOVA) test was performed for Mean±SD & Multiple comparison between groups through Post Hoc test: Tukey's test, Kruskal–Wallis was performed for Median (IQR) & Multiple comparison between groups through Mann-Whitney test. Different capital letters indicate significant difference at ($p<0.05$) among means in the same row. *p-value <0.05 is significant.

or metabolic parameters ($p > 0.05$). A weak negative correlation was noted between vitamin D and body fat % ($r = –0.234$, $p = 0.068$), and a weak positive but non-significant correlation with HbA1c ($r = 0.210$, $p = 0.102$), table (9).

DISCUSSION

Vitamin D deficiency (VDD) is highly prevalent among children with type 1 diabetes mellitus (T1DM), although reported rates vary widely across populations. Despite growing global interest, Egyptian data remain limited. The present study evaluated vitamin D status in Egyptian pediatric T1DM patients and explored its relationship with anthropometric parameters and glycemic control. To our knowledge, this is the first Egyptian study to simultaneously investigate these associations in both children and adolescents with T1DM.

The main findings of this study were: (i) a remarkably high prevalence of vitamin D deficiency (90.3%), with no participants showing sufficiency; (ii) vitamin D–deficient patients had significantly higher BMI Z-scores, greater body-fat percentages, and lower height Z-scores compared with those who were insufficient; and (iii) poor glycemic control was common (67.7% with HbA1c > 8%), although no significant correlation was observed between vitamin D levels and HbA1c.

Table 8. Comparison of laboratory findings according to glycemic control

Laboratory data	HbA1C Level			p-value
	Poor	Inadequate	Good Control	
Vitamin D level (ng/ml)	4.58±2.46	6.58±2.98	7.45±3.80	0.109
HbA1C (gm%)	9.46±1.12 A	7.29±0.28 B	6.18±0.56 C	<0.001**

Using: One way Analysis of Variance test was performed for Mean±SD & Multiple comparison between groups through Post Hoc test: Tukey's test. Different capital letters indicate significant difference at ($p<0.05$) among means in the same row. *p-value <0.05 is significant.

Table 9. Correlation between vitamin D levels with different parameters among the T1D patients

Parameter	Vitamin D level	
	R	P
Duration	-0.029	0.823
Age (years)	-0.148	0.252
zWt	0.006	0.965
zHt	-0.044	0.736
Waist C	-0.054	0.678
Hip C	-0.073	0.575
MUAC	-0.002	0.987
Biceps SF	0.021	0.872
Triceps SF	0.068	0.600
Subscap SF	-0.077	0.554
SupraIl SF	-0.117	0.367
zBMI	-0.011	0.931
Waist/Hip	0.138	0.284
Fat Percent	- 0.234	0.068
HbA1C	0.210	0.102

Using Spearman's rank correlation coefficient (r), $p< 0.05$= significant.

Previous studies have reported variable rates of VDD among diabetic children. One study found combined deficiency and insufficiency in 64.2% of newly diagnosed cases versus 41.6% in established patients. t [1] Similarly, others reported significantly lower mean vitamin D levels in pediatric T1DM patients than in controls, t [2] consistent with earlier evidence linking low vitamin D levels to increased T1DM risk. t [3] Conversely, a Finnish cohort found no relationship between serum 25(OH)D and diabetes development. t [4]

Marked geographic differences have also been observed. In Turkey, deficiency reached 42.9% among adolescents, with vitamin D levels declining with age and being lowest in late-adolescent females. t [5] In contrast, our cohort showed universal deficiency or insufficiency, with no

significant sex or age differences. Such discrepancies likely reflect differences in sunlight exposure, seasonal variation, diet, and lifestyle. Supporting this, a Ukrainian study reported predominantly normal vitamin D levels, t [6] while data from the UAE showed deficiency to be more frequent among controls than diabetic patients, likely due to routine supplementation in diabetics. t [7] In contrast, reports from Kuwait and Egypt consistently demonstrate markedly higher deficiency rates, [48Ò49] although some investigators found comparable prevalence in diabetic and non-diabetic children .[50] Additional mechanisms, such as urinary loss of vitamin D-binding protein, may also contribute. [51]

The relationship between vitamin D and glycemic control remains controversial. In our study, most participants exhibited poor glycemic control, consistent with the Diabetes Control and Complications Trial (DCCT) findings that achieving optimal control in adolescents is particularly challenging. [52] Longer diabetes duration was significantly associated with poorer HbA1c values, supporting prior evidence that glycemic control tends to worsen over time due to â-cell exhaustion and behavioral factors. Despite widespread deficiency, vitamin D levels were not significantly correlated with HbA1c, suggesting that glycemic control is more influenced by adherence to insulin therapy and lifestyle factors than by vitamin D status alone.

Our findings are consistent with several reports showing no significant relationship between serum 25(OH)D and HbA1c. [45,53,54] Conversely, other studies have documented an inverse correlation between vitamin D and HbA1c, implying a contributory role of deficiency in poor glycemic control. [55-57] Interventional studies have also yielded mixed results, some reporting improved HbA1c after vitamin D supplementation, [58-60] while others observed no significant change. [61] These discrepancies likely reflect variations in baseline vitamin D status, study design, dosage, duration, and ethnic or genetic background.

The relationship between vitamin D and adiposity is equally complex. In our cohort, vitamin D–deficient patients had higher BMI Z-scores ($p = 0.024$) and greater body-fat percentages ($p = 0.024$), although no direct correlation between vitamin D levels and adiposity indices was detected. Similar inverse relationships have been described in both diabetic and non-diabetic populations, [62, 63] while some studies reported no association.[64Ò65] Proposed mechanisms include sequestration of vitamin D in adipose tissue, reducing its bioavailability, [66] and modulation of adipocyte differentiation and metabolism. [67] Meta-analyses have shown inconsistent outcomes, some indicating inverse associations with fat mass, [68] and modest benefits of supplementation on BMI and waist circumference, [69] whereas others found little or no effect. [70,71]

Vitamin D status may also influence growth and body composition in T1DM. In this study, children with deficiency had significantly reduced height Z-scores compared with those with insufficiency ($p = 0.016$), and females demonstrated higher body-fat percentages, in line with previous observations. [53,72] These alterations may be attributed to the combined effects of vitamin D deficiency, insulin therapy, nutritional intake, and pubertal hormonal changes. Given vitamin D's role in skeletal development and bone-mass accrual, maintaining optimal vitamin D status is crucial for long-term growth and bone health in pediatric T1DM.

Anthropometric analysis revealed that hip circumference was lowest among patients with good glycemic control, whereas the waist-to-hip ratio was highest. This may reflect differences in fat distribution patterns and their metabolic implications. Visceral adiposity is more strongly linked to insulin resistance and inflammation than subcutaneous fat, explaining why BMI alone may not sufficiently reflect metabolic risk. [73]

Taken together, our findings highlight the multifaceted role of vitamin D in T1DM. Although deficiency is nearly universal in our cohort, its association with glycemic control, adiposity, and growth remains

inconclusive. Geographic, environmental, and genetic factors likely contribute to these variations. Nonetheless, the consistently high prevalence of deficiency underscores the need for routine screening and correction as part of comprehensive care in children with T1DM.

Limitations

This study has several limitations that should be acknowledged. First, its cross-sectional design precludes establishing a causal relationship between vitamin D status and glycemic control, adiposity, or growth parameters. Longitudinal or interventional studies would be more suitable for assessing temporal associations. Second, the relatively small sample size may limit the generalizability of our findings to the wider Egyptian pediatric population. Third, seasonal variation in sunlight exposure was not accounted for, and since serum 25(OH) D levels fluctuate with sun exposure, this might have influenced the measured vitamin D concentrations. Fourth, dietary intake and supplement use were not quantitatively assessed, which could have contributed to individual variability in vitamin D levels. Finally, other relevant biochemical markers, such as parathyroid hormone (PTH), calcium, phosphorus, and bone mineral density, were not evaluated, which might have provided additional insight into vitamin D metabolism and skeletal outcomes in T1DM.

Despite these limitations, the study's strengths include a well-characterized pediatric diabetic cohort, the use of standardized anthropometric and biochemical assessments, and focus on a geographically underrepresented population; Egyptian children and adolescents with T1DM, adding valuable regional data to the global literature.

CONCLUSION

This study demonstrates an alarmingly high prevalence of vitamin D deficiency among Egyptian children and adolescents with type 1 diabetes mellitus. Vitamin D deficient patients exhibited higher BMI Z-scores, increased body fat percentage, and lower height Z-scores. However, no significant relationship was observed between vitamin D levels and glycemic control.

These findings suggest that while vitamin D deficiency is highly prevalent and associated with adverse anthropometric outcomes, its impact on glycemic regulation remains uncertain. The study underscores the need for routine screening and supplementation strategies to correct vitamin D deficiency in pediatric T1DM patients as part of comprehensive disease management. Future prospective interventional studies are warranted to clarify the causal pathways linking vitamin D status, adiposity, and metabolic control in diabetic youth.

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Conflict of interest

The authors do not have any conflict of interest.

Data Availability Statement

This statement does not apply to this article.

Ethics Statement

The study was approved by the National Research Centre Ethical Committee (Approval 16390). Written informed consents were obtained after explaining the study to the children's parents or care givers. The study was carried out accordance with national regulations, institutional guidelines, and The Declaration of Helsinki's ethical guidelines.

Informed Consent Statement

Informed consent was obtained from the patients' parents or guardians.

Clinical Trial Registration

This research does not involve any clinical trials.

Permission to reproduce material from other sources

Not Applicable.

Author contributions

Wafaa Abdel Samie Kandeel: Conceptualization, design of the study, supervision and revision of the final manuscript; Ahmed Sayed Ismail: clinical, anthropometric examination of patients and collection of data; Mona Abdelkader Mohamed Awad: laboratory investigations; Eman Refaat Youness: laboratory investigations; Hanaa Reyad Abdallah: clinical, anthropometric examination of patients and collection of data, interpretation of results, writing the original draft, reviewing and editing the final manuscript.

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