ROLE OF VITAMIN D IN TYPE 1 DIABETES MELLITUS IN CHILDREN

By

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ABSTRACT

Introduction: *Type 1 Diabetes mellitus (T1DM) is the most common endocrinal disorder of childhood and adolescence, with important consequences for physical and emotional development. Vitamin D has Immunomodulatory properties. Several studies suggest that vitamin D supplementation in early childhood decreases the risk of developing T1DM.*

Objective: *The aim of this study was to find the association between vitamin D and type 1 diabetes mellitus (T1DM) then to study the difference in the level of vitamin D in T1DM and healthy subjects, and to examine the influence of disease characteristics or specific treatment on vitamin D status*

Patients and methods: *This is a case-control study which was conducted from August 2012 to January 2014 and included fifty patients with T1DM matched with age (2-18years) and gender of fifty apparently healthy controls. Both studied groups were subjected to thorough history, clinical examination and investigations which include random blood sugar and serum 25 hydroxyvitamin D (25OHD) level and mean glycated hemoglobin (HbA1c) was done for diabetic group.*

Results: *our study showed that48% of diabetic and 56% of control were males and their age was 9.25 ± 3.70 year and 8.38 ± 4.26 year for cases and controls respectively. Vitamin D deficiency was more in diabetic compared to control children. There was significant vitamin D deficiency in newly diagnosed diabetes, children with diabetes duration less than 5 years, patients on conventional insulin therapy and those with higher hemoglobin A1c level. There was insignificant effect of age, sex, birth order, consanguinity, family history of diabetes, body mass index and frequency of acute complications or frequency of hospitalizations on vitamin D level in both groups.*

Conclusions: *vitamin D deficiency and low vitamin D intake are potential risk factors for developing type 1 diabetes and vitamin D deficiency is prevalent in diabetic children especially those newly diagnosed, with high hemoglobin A1c and those on conventional insulin therapy.*

Key words: *type I diabetes, vitamin D, hemoglobin A1c.*

INTRODUCTION

Type 1 diabetes mellitus (T1DM) is a common, chronic, metabolic syndrome characterized by hyperglycemia caused by deficiency of insulin secretion. T1DM is the most common endocrine-metabolic disorder of childhood and adolescence, with important consequences for physical and emotional development. Morbidity and mortality result from acute metabolic derangements and from long-term complications **(Crimmins and Dolan, 2008)**.

Type 1 Diabetes mellitus is thought to be the consequence of an autoimmune destruction of the insulin producing beta cell as a result of interactions between different susceptibility genes and environmental exposure (ADA, 2011).

The increasing incidence of type 1 diabetes mellitus strongly suggests the importance of environmental factors; the major factors being pursued include diet and viral infections **(Eisenbarth, 2007)**.

Serum 25-hydroxyvitamin D (25(OH)D) concentrations are largely determined by environmental factors, mainly through vitamin D intake and ultraviolet exposure. The main marker of vitamin D status is the metabolite 25(OH)D, which is synthesized in the liver **(Holick, 2006)**.

One of the environmental factors thought to be protective against the development of T1DM is early supplementation with vitamin D. Vitamin D has been shown to possess Immunomodulatory properties. Several studies suggest that vitamin D supplementation in early childhood decreases the risk of developing T1DM **(Mathieu et al., 2005)**. Moreover, there is a negative correlation between vitamin D intake of pregnant mother and the presence of islet antibodies in her child **(Fronczak et al., 2003)**.

Studies from different countries have shown a highly variable prevalence of vitamin D deficiency ranging from 15 to 60% among children and adolescents with T1DM **(Pozzilli et al., 2005**, **Littorin et al., 2006**, **Greer et al., 2007**, **Bener et al., 2009**, **Svoren et al., 2009**, and **Janner et al., 2010)**.

Definition of vitamin D deficiency is controversial, however, most experts agree that 25(OH)D of <20 ng/ml is considered to be vitamin D deficiency, whereas a 25 (OH)D of 20–29 ng/ml is considered to be insufficient, and a level ≥30 ng/ml is the sufficient value of vitamin D

in both children and adults **(Holick, 2009)**.

AIM OF WORK

The aim of this study was to find the association between vitamin D and type 1 diabetes mellitus (T1DM) then to study the difference in the level of vitamin D in T1DM and healthy subjects, and to examine the influence of disease characteristics or specific treatment on vitamin D status.

PATIENTS AND MATERIALS

Study Design:

This is a case-control study which was designed to determine the relationship between vitamin D and Type 1 diabetes mellitus (T1DM) in young Egyptian population from 2-18 years of age. The survey was conducted over a period from August 2012 to January 2014. Our study included fifty patients with T1DM matched with age and gender of fifty apparently healthy controls.

Ethical consideration:

1. Approval of ethical committee, Faculty of Medicine Al-Azhar University.
2. Written consents from parents of the patients.
3. The patients have the right to withdraw from the study at any time.
4. All the obtained data are confidential, and the patients have the right to keep them.
5. The authors declare that there is no any financial support regarding the research and publication.
6. No conflict of interest regarding the study and publication.

Inclusion criteria for diabetic group:

1. Known type 1 diabetic patients attending diabetic clinics under insulin therapy.
2. Age from 2 to 18 years.
3. Both sexes.
4. Both newly diagnosed and old patients.

Exclusion criteria for diabetic group:

1. Patients with significant chronic illness other than diabetes, e.g renal or hepatic diseases.
2. Patients on vitamin D therapy.
3. Patients with signs of rickets.

Control group:

Included fifty apparently healthy children of the same age and sex group.

Methods:

Both groups were subjected to the following:

1. **Thoroughly history with stress on:** Vitamin D supplementation in early life. For diabetic group diabetes history included: age of onset, duration of the disease and regimen of insulin therapy.
2. **Thoroughly general examination and stress on:** anthropometric measures (Weight, length and body mass index) and systemic examination for the heart, chest, abdomen and CNS of both groups.
3. **Investigations:**

- Random blood sugar using Bionime GM100 glucotest.
- Serum 25 hydroxyvitamin D (25OHD) level: Venous blood samples were collected into plain tubes, and serum was separated and stored at -70˚C until analysis. Levels of 25OHD were measured with ELISA(enzyme linked immunosorbent assay) method (Using ELISA kit No KAP 1971 manufactured by DIAsource Immuno-assays, S.A. company, Belgium).
- Fordiabetic group Mean glycated hemoglobin (HbA1c) also was done using spectrophotometer at 415nm with reagent manufactured by Stanbio laboratory, Boerne, Texas, USA (Nathan et al, 1984). The patients were classified accordingly into three groups:
- Good control (6-7.9).
- Fair control (8-9.9).
- Poor control (≥ 10) **(Alemzadeh and Ali, 2011)**.

Statistical analysis of data:

Statistical analysis of our results were conducted using the mean, standard deviation, student t- test, Chi-square, Linear Correlation Coefficient and Analysis of variance [ANOVA] tests by using Microsoft Excel 2010 and SPSS v19.0 for Microsoft Windows 7.

Sample size was calculated using G. power softwere.

A total sample of 100 children (50 in each group) was required to estimate on effect size of sample = 0.298 with significance level of 5% (two side test) that will provide power of 90% .

RESULTS

Our result will be demonstrated in the following tables and figure.

Table (1): General Characteristics of the Studied Diabetic and Control Groups

variables	Diabetic group		Control group		Chi-square X^2	T -test	P-value
	N = 50	%	N = 50	%			
Age (years)							
Mean ± SD	9.25 ± 3.70		8.38 ± 4.26		-----	1.090	>0.05
Sex							
Male	24	48	28	56	0.642	-----	>0.05
Female	26	52	22	44			
Birth order							
Mean ± SD	2.30 ± 1.28		2.18 ± 1.10		-----	0.503	>0.05
Vitamin D supplements in first year*							
No	40	80	25	50	11.753	-----	<0.05*.
Yes	10	20	25	50			
Consanguinity							
Negative	36	72	44	88	4.921	-----	> 0.05
Positive	14	28	6	12			
Family history of diabetes							
Negative	18	36	41	82	22.891	-----	<0.001*
Positive	32	64	9	18			
Type of diabetes in the family							
Type 1	11	22	3	6	22.894	-----	<0.001*
Type 2	21	42	6	12			
Body mass index*							
Normal	35	70	46	92	7.930	-----	<0.05**
Overweight	10	20	3	6			
Obese	5	10	1	2			
Mean ± SD	18.27 ± 3.47		17.06 ± 2.45		-----	2.014	<0.05**

This table shows no statistically significant difference between diabetic and control groups as regard age, sex and birth order, but there was statistically significant difference between them as regard vitamin D supplements being more in control group and family history of diabetic group and also body mass index in diabetic group.

Table (2): Serum glycated hemoglobin (HBA1c) level for diabetic patients

HemoglobinA1c (%)*	No	%
Good control (6-7.9)	39	78
Fair control (8-9.9)	10	20
Poor control (≥ 10)	1	2
Range	5.13 – 10.5	
Mean ± SD	7.28 ± 1.06	

*According toAlemzadeh and Ali, 2011

This table shows that most of our diabetic patients had good to fair glycemic control.

Table (3): Serum 25 hydroxyvitamin D levels of the diabetic and control groups

Serum 25 (OH) vitamin D level (ng/ml)	Diabetic group		Control group		Chi-square X^2	t	P-value
	N=50	%	N=50	%			
Deficient (<20)	37	74	24	48	13.852		<0.05*
Insufficient (20-29)	10	20	14	28			
Sufficient (≥30)	3	6	12	24			
Mean ± SD	15.08 ± 7.69		19.84 ± 8.00			3.033	<0.05*

This table shows that there was statistically significant difference between both diabetic and control groups as regard vitamin D level being more deficient in the diabetic group ($p < 0.05$).

Table (4): Comparison among the three groups of vitamin D status and degree of glycemic control {HBA1c (%)}

HBA1c (%)	Vitamin D level			ANOVA	
	Deficient n = 37	Insufficient n =10	Sufficient n = 3	F	P-value
Range	5.13-10.5	6.13 - 7.820	5.4 - 7.7	3.889 R=-0.38	<0.05*
Mean ±SD	7.515 ± 1.062	6.750 ± 0.931	6.573 ± 0.764		

This table shows that there was statistically significant negative correlation betweenHbA1c levelandvitamin D status.patient with higher HbA1c had lower mean 25OHD.

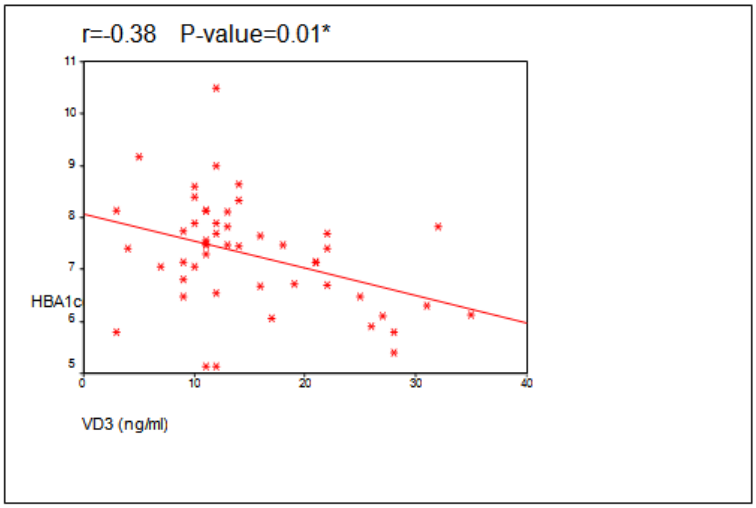

Figure (1): Correlation between vitamin D level and HBA1c in the diabetic group

This figure shows that there was statistically significant negative correlation between HbA1c level and vitamin D status. Patients with higher HBA1c had lower mean 25OHD.

Table (5): Comparison between patients on conventional and those on basal bolus intensive insulin therapy regarding vitamin D and HBA1c levels

Variable	Conventional regimen n =32	Basal bolus regimen n =18	t	P-value
25OHvitamin D (ng/ml) mean ± SD	13.469 ± 7.121	17.944 ± 8.018	-2.039	<0.05*
HBA1c (%) mean ± SD	7.615±1.033	6.685±0.855	3.243	<0.05*

This table shows that patients on conventional insulin therapy have significantly lower serum 25OHvitamin D and higher HBA1c than those on basal bolus regimen.

Table (6): Correlation between mean serum vitamin D level and demographic data, dietetic history and family history of the diabetic group

Variables		N	%	VD3 (ng/ml)			ANOVA or T-test	
				Mean	±	SD	F or t	P-value
Age (years)	2-	10	20	13.3	±	6.325	0.417	>0.05
	6-	25	50	15.12	±	7.965		
	≥12y	15	30	16.2	±	8.291		
Sex	Male	24	48	13.083	±	5.208	-1.843	>0.05
	Female	26	52	16.923	±	9.139		
Birth order	1-2	33	66	14.515	±	6.624	0.441	>0.05
	3-4	12	24	16.917	±	10.202		
	≥5	5	10	14.400	±	8.562		
Vitamin D supplements in 1st 2 years	Yes	7	14	15.902	±	8.049	2.675	<0.05*
	No	40	80	11.333	±	4.330		
Consanguinity	Yes	14	28	15.167	±	7.959	-0.542	>0.05
	No	36	72	16.700	±	7.718		
Feeding history	Breast feeding	44	88	15.841	±	7.838	1.923	>0.05
	Formula feeding	4	8	10.250	±	0.957		
	Mixed	2	4	8.000	±	5.657		
Family history of diabetes	Negative	18	36	12.833	±	5.864	1.611	>0.05
	Type 1	11	22	14.727	±	6.769		
	Type 2	21	42	17.190	±	9.130		

This table shows that the mean serum level of 25OHVD was not affected by the age, sex, birth order, consanguinity, type of feeding and family history of diabetes but it was affected by vitamin D supplements in 1st 2 years of life being higher in those who received vitamin d than those who did not.

Table (7): Correlation between mean serum vitamin D level and duration of diabetes, frequency of complications and regimen of insulin therapy

Variables		N	%	VD3 (ng/ml) Mean ± SD	ANOVA or T-test	
					F or t	P-value
Duration of diabetes (years)	<5	43	86	10 ± 6.028	2.985	<0.05*
	≥5	7	14	18.865 ± 7.465		
Frequency of hospital admission (last year)	No	23	46	18.188 ±7.960	1.675	>0.05
	One	16	32	13.750 ± 7.820		
	Two	4	8	9.667 ± 1.155		
	Three or more	7	14	14.857 ± 6.466		
Frequency of DKA (last year)	No	25	50	15.400 ± 8.426	0.304	>0.05
	One	14	28	15.500 ± 8.103		
	Two	4	8	11.500 ± 3.786		
	Three or more	7	14	15.143 ± 6.309		
Frequency of Hypoglycemia (last year)	No	38	76	15.045 ± 7.844	-0.085	>0.05
	Yes	12	24	15.333 ± 7.062		
Newly diagnosed/ established cases	Newly diagnosed	16	32	11.94 ± 6.855	-2.046	<0.05*
	Established cases	34	68	16.56 ± 7.704		

This table shows that vitamin D level was significantly lower in newly diagnosed patients and patients with duration less than five years. No statistically significant effect of frequency of complications on mean serum vitamin D level.

DISCUSSION

The present study comprised 50 children having type 1 diabetes and 50 apparently healthy children as control. The latter were age and sex matched. Of the total number of children surveyed, 48% of diabetic and 56% of healthy children were males and 52% of diabetic and 44% of healthy children were females. The mean age (in years) for patients versus controls was 9.25 ± 3.70 versus 8.38 ± 4.26.

Regarding birth order we did not find significant difference between diabetic and control groups **(Table 1)**, this finding comes in agreement with **Robertson and Harrild, (2010)** and disagree with **Ramachandran et al., (1993)** who reported that the incidence of diabetes increases with higher birth order.

The size of the sample of our study and the high percentage of breast feeding in both diabetic and control groups (88% and 76% in diabetic and nondiabetic children respectively) may explain our finding.

No significant difference was found between diabetic and control groups regarding consanguinity, both groups have low percentage of consanguinity (28% and 12% for diabetic and control groups respectively) **(Table 1)**. In contrast to this result **Bener et al, (2009)** found higher consanguinity in parents of healthy children (64.1%) when compared to diabetic children (48.8%), ($P<0.05$). On the other hand **Lebenthal et al., (2012)** reported that consanguineous marriages may contribute to an increased genetic predisposition for the development of T1D since the two parents may share similar susceptibility genes that could be transmitted to their offspring.

Regarding body mass index (BMI), we found that diabetic patients have significantly higher BMI than control (in diabetic group, 20% overweight and 10% obese, while in control group 6% overweight and 2% obese with the mean BMI was 18.27 ± 3.47 and 17.06 ± 2.45 for diabetic and control groups respectively).

Bener et al., (2009) and **Svorin et al., (2009)** found no significant difference between diabetic and control groups as regard to BMI. On the other hand, **Kaminski et al., (2013)** found that BMI scores for the diabetic patients were slightly lower than the control population. Our result goes with the Accelerator Hypothesis of **Wilkin, (2012)** who states that obesity is driving the rising incidence of both type 1 and type 2 diabetes. Obesity causes increased insulin resistance, and the resulting high glucose levels accelerate destruction of the beta cells.

Regarding family history of diabetes we found that the diabetic children had significantly higher percentage of positive family history of type 1 and type 2 diabetes than control children **(Table 1)**, this result comes in agreement with **Mustuura et al., (1998)** who stated that the first degree relatives of patients with T1D are at increased risk compared to the general population.

The main finding of our study was the significant higher frequency of vitamin D deficiency and insufficiency in diabetic (74%) than control (48%) children. Also a significant difference was noted between diabetic and healthy children in

the mean value of 25 hydroxyvitamin D (15.08 ± 7.69 for diabetic patients and 19.84 ± 8.00 for control children). Although the vitamin D deficiency was prevalent in both groups, it was much higher in diabetic children.

Similar finding was reported in different contries, **Pozzilli et al., (2005)** found low 25-OHD levels in 88 newly diagnosed children and adolescents in Italy. **Littorin et al., (2006)** found low 25-OHD levels in 459 Swedish patients who were newly diagnosed with T1DM compared with age-matched and place-matched controls. **Greer et al., (2007)** reported similar finding in Australian children.

On the contrary, **Bierschenk et al., (2009)** found the 25-OH vitamin D levels were similar among control subjects, new-onset type 1 diabetic patients, established type 1 diabetic patients, and first-degree relatives in their cross-sectional study on 415 individuals residing in Florida: 153 control subjects, 46 new-onset type 1 diabetic patients, 110 established type 1 diabetic patients (5 months from diagnosis), and 106 first-degree relatives of the diabetic patients. . However, the mean 25-OH vitamin D levels were less than the optimal World Health Organization level which is 30ng/ml in all study groups.

These overall differences might be explained by the variability of geographical environment, ages of the subjects, duration of diabetes and glycemic control **(Svoren et al., (2009)**.

The protective effects of vitamin D are mediated through the regulation of several components such as the immune system and calcium homeostasis. However, an increasing amount of evidence suggests that vitamin D also affects beta cells directly thereby rendering them more resistant to the types of cellular stress encountered during T1D and T2D **(Wolden-Kirk et al., 2011)**.

However, in contrast to all these findings, **Simpson et al., (2011)** reported that neither vitamin D intake nor 25(OH)D levels throughout childhood were associated with the risk of islet autoimmunity or progression to type 1 diabetes in population.

Evidence from basic, clinical and epidemiological studies provides a rationale for this hypothesis. Observations in animal models of diabetes and human studies have implicated vitamin D deficiency in the impairment of insulin synthesis and secretion **(Zeitz et al, 2003)**, while vitamin D supplementation

has been demonstrated to attenuate cytokine-mediated pancreatic beta-cell destruction **(Gysemans et al., 2005)**. Receptors for 1, 25(OH)2-vitamin D3 are expressed in antigen-presenting cells and T-cells as well as in pancreatic beta cells **(Adams et al., 2007)**.

Our study revealed that there was statistically significant difference among the three groups of vitamin D status (deficient, insufficient and sufficient groups) regarding mean glycated hemoglobin (HbA1c) level with higher HbA1c in the deficient group and the correlation was negative (r = -0.38). This negative correlation was reported by **Alemzadeh et al., (2008)** in their cross-sectional study of 127 adolescents (mean age: 13 years) which showed an inverse relationship between 25(OH)D and hemoglobin A1c levels.

As regard management, 64% of patients use intermediate + short acting insulin and 36% use basal long acting insulin analogue (insulin glargine) + rapid acting insulin analogue (insulin glulisine), this is because insulin analogues are more expensive and not routinely dispersed by health insurance. Our study revealed that patients were on conventional regimen had significant lower vitamin D level and higher hemoglobinA1c level than those on basal bolus intensive regimen **(Table 6)**.

Conventional insulin therapy (CIT) usually refers to two injections of insulin daily, most commonly a mixture of regular and NPH insulin injected twice daily before breakfast and dinner. This technique is no longer standard of care **(Paris et al., 2009)**.

So, American Diabetes Association (ADA) recommends that people with type 1 diabetes should be treated with MDI injections (three to four injections per day of basal and prandial insulin) or continuous subcutaneous insulin infusion (CSII) and insulin analogs should be used to reduce hypoglycemia risk **(ADA, 2014)**.

However data from studies in experimental diabetic nephropathy indicate that vitamin D insufficiency may also be involved in the pathogenesis of albuminuria. In the general population, an inverse association is found between the level of vitamin D and the prevalence of albuminuria **(de Boer et al., 2007)**, and limited data from clinical trials in nondiabetic chronic kidney disease (CKD) patients suggest that treatment with paricalcitol (vitamin D

receptor analog [VDRA]) may reduce proteinuria **(Alborzi et al., 2008)**.

In our study, we did not find any significant effect of age, sex and birth order on vitamin D level. **Bener et al., (2009)** and **Huynh et al., (2009)** reported a similar finding, but **Ataie-Jafari et al., (2012)** found that girls had higher prevalence of vitamin D deficiency than boys but they found no significant effect of age on vitamin D level.

Also we did not find any significant effect of consanguinity, feeding history, family history of diabetes and body mass index on serum vitamin D level. This coincides with **Svoren et al., (2009)** but in contrast to this finding, **Dong et al., (2010)** found significant inverse correlations between 25- hydroxyvitamin D levels and all adiposity measurements, including BMI percentile ($P< 0.05$), waist circumference ($P<0.01$), total fat mass ($P< 0.01$), percentage of body fat ($P < 0.01$), visceral adipose tissue ($P <0.015$), and subcutaneous abdominal adipose tissue ($P <0.05$).

Also we found that the three groups of vitamin D deficiency were similar regarding age of onset of diabetes, the finding which is supported by **Huynh et al., (2009)** who studied the relationship between measured 25(OH)-vitamin D3 levels and the degree of acidosis in children presenting with new-onset T1DM and found that no effect of age at presentation on vitamin D level.

CONCLUSION

Vitamin D deficiency is more in diabetic than non-diabetic children. Among diabetic patients, vitamin D deficiency is more in children who did not receive vitamin D supplements in early life, newly diagnosed diabetes, children with diabetes duration less than 5 years and those with higher hemoglobin A1c level. Patients on conventional insulin therapy have lower serum vitamin D and higher hemoglobin A1c level than those on basal bolus intensive regimen.

RECOMMENDATION

Supplementing infants with vitamin D might be a safe and effective strategy for reducing the risk of T1DM.For diabetic patients, multiple daily intensive regimen (or basal bolus regimen) is preferable for better glycemic control and adequate vitamin D level. Future studies are needed to confirm our findings of vitamin D inadequacy in T1DM. However the results of this study, combined with previous researches, may have considerable importance for

preventive medicine regarding type 1 diabetes.

Study limitations:

The only limitation is that it was not a blind or randomized study.

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دور فيتامين (د) في مرض السكرى النوع الأول في الأطفال

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مقدمة: مرض السكري من النوع الأول (T1DM) هو اضطراب الغدد الصماء الأكثر شيوعًا في الطفولة والمراهقة، وله عواقب مهمة على النمو البدني والعاطفي. وهي متلازمة أيضا مزمنة تتميز بارتفاع السكر في الدم الناجم عن نقص إفراز الأنسولين. يشير تزايد الإصابة بمرض السكري من النوع الأول بقوة إلى أهمية العوامل البيئية؛ فيتامين د هو أحد العوامل البيئية، وله خصائص مناعية. تشير العديد من الدراسات إلى أن مكمل الفيتامين (د) في مرحلة الطفولة المبكرة تقلل من خطر الإصابة بـ T1DM.

الهدف: كان الهدف من دراسة الحالات والشواهد المتطابقة هو إيجاد العلاقة بين فيتامين د ومرض السكري من النوع الأول (T1DM) ثم دراسة الاختلاف في مستوى فيتامين د في T1DM والأشخاص الأصحاء، وفحص تأثير خصائص المرض أو علاج محدد لحالة فيتامين د.

الطرق: هذه دراسة حالة شواهد أجريت على مدى الفترة من أغسطس 2012 إلى يناير 2014 وتضمنت خمسين مريضًا مصابًا بـ T1DM متطابق مع العمر (2-18 عامًا) والجنس

من خمسين شخصًا يبدو أنهم يتمتعون بصحة جيدة. خضعت مجموعة مرضى السكر إلى تاريخ شامل وفحص وتحقيقات سريرية شملت الدم العشوائي والسكري عن الهيموغلوبين السكري (HbA1c) ومستوى المصل 25 هيدروكسي فيتامين د (25OHD). خضعت المجموعة الضابطة لتاريخ شامل وفحص وتحقيقات سريرية شملت الدم العشوائي ومستوى 25 هيدروكسي فيتامين د (25OHD) في الدم.

النتائج: في هذه الدراسة 48٪ من مرضى السكري 56٪ من مجموعة السيطرة ذكور وأعمارهم كانت 9.25 ± 3.70 و 8.38 ± 4.26 للحالات والضابطة على التوالي. كان نقص فيتامين د أكثر في مرضى السكري مقارنة بالأطفال غير المصابين بالسكري. كان نقص فيتامين (د) أكثر عند الأطفال الذين لم يتلقوا مكملات فيتامين (د) في وقت مبكر من حياتهم، والسكري الذي تم تشخيصه حديثًا، والأطفال الذين يعانون من مرض السكري لمدة تقل عن 5 سنوات، والمرضى الذين يخضعون للعلاج التقليدي بالأنسولين وأولئك الذين لديهم مستوى أعلى من الهيموجلوبين A1c. لا فرق بين الأطفال الذين يعانون من نقص فيتامين (د) والأطفال الذين لديهم كفاية فيتامين (د) فيما يتعلق بالعمر والجنس وترتيب الولادة وقرابة الأقارب والتاريخ العائلي لمرض السكري ومؤشر كتلة الجسم. كما لم يلاحظ أي اختلاف بينهما فيما يتعلق بعمر ظهور مرض السكري، وتواتر المضاعفات الحادة أو تواتر الاستشفاء.

الاســـتنتاجات: يعتبـــر نقـــص فيتـــامين د وانخفـــاض تنـــاول فيتـــامين د فـــي الحيـــاة المبكـــرة مـــن عوامـــل الخطـــر المحتملـــة للإصـــابة بمـــرض الســـكري مـــن النـــوع الأول، كمـــا أن نقـــص فيتـــامين د منتشـــر فـــي الأطفـــال المصـــابين بمـــرض الســـكري وخاصـــة أولئـــك الـــذين تـــم تشخيصـــهم حـــديثًا والـــذين يعـــانون مـــن ارتفـــاع الهيموجلـــوبين A1c والذين يخضعون للعلاج التقليدي بالأنسولين.