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Vitamin D Status among Obese Children and Its Relation to Insulin Resistance

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Abstract: Background and Aims: We aimed to assess the relation between insulin resistance and serum 25-hydroxy vitamin D (25[OH]D) levels in both obese and non-obese children. **Methods and Results:** This cross-sectional study determined vitamin D levels of overweight children and their associations with insulin sensitivity, resistance, and glucose homeostasis. The study was conducted at the Childhood Obesity Clinic and Clinical Pathology Department, National Nutrition Institute, Cairo, Egypt, during January 1st 2015 to June 1st 2016. Obese participants had lower concentrations of 25(OH)D than non-obese participants but that was not notably different. The overweight group's 2-hour postprandial blood glucose level (2HRPP), fasting insulin, homeostatic model assessment of insulin resistance (HOMA-IR), and HOMA-B were all significantly higher than the control group's values, and then were linked to adiposity measures. Fasting blood sugar and hemoglobin A1c showed no statistically relevant differences in between the obese and non-obese groups. In the overweight group, 25(OH)D deficiency, insufficiency, and sufficiency (25[OH]D < 20 ng/dl, < 30, >20 ng/dl; ≥30 ng/dl, respectively) were not linked with insulin sensitivity or resistance indices. Blood pressure was positively associated with adiposity indices. **Conclusion:** The study showed no notably difference in vitamin D status among obese and normal, non-obese participants, but there was significant difference between obese and normal weight subjects regarding insulin sensitivity and resistance. Measures and 2-hour postprandial blood glucose level (insulin sensitivity and resistance indices were related to adiposity indices) and the increase in systolic and diastolic blood pressure was related to adiposity indices and insulin resistance.
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Key words: Insulin; Obese child; Vitamin D

1. Introduction

Obesity is becoming more common among kids and adolescents at an alarming rate. Obesity has reached epidemic proportions in some developing countries, exceeding that in many developed nations [1].

Over the last 30 years, overweight has more than doubled in children and quadrupled in teenagers. Obesity within 6–11-year-old children in the United States increased from 7% in 1980 to approximately 18% in 2012. Similarly, over the same time period, the number of obese adolescents aged 12–19 years increased from 5% to about 21%. In the year 2012, more than a third of children and adolescents were overweight or obese [2].

Increases in childhood hypertension, hyperlipidemia, and type 2 diabetes have coincided with the rise in obesity rates. Childhood obesity has been related to a higher risk of heart disease and type 2 diabetes in older age [3].

25-hydroxy vitamin D (25[OH]D) deficiency is one of the problems that has been linked to obesity [4].

Vitamin D production in the skin is influenced by exposure to sunlight, latitude, skin-covering clothing, sunscreen, and skin color. Despite the fact that the Mediterranean region has a relatively sunny climate, European and Mediterranean countries have increased rates of hypovitaminosis D [5]. The correlation between vitamin D deficiency and obesity is now recognized to be a bidirectional link. Serum 25(OH)D level was reported to be directly proportional to one's body mass index (BMI) [6]. The greater sequestration of vitamin D in body fat is thought to be the cause of the apparent decrease in vitamin D bioavailability with increased adiposity [7]. The other point of view is that vitamin D can cause obesity and can cause weight gain over time [8]. Several studies have related lower 25(OH)D levels to a higher incidence of impaired glucose tolerance and type 2 diabetes, but not all of them [9].

Vitamin D's direct impacts on pancreatic-cell insulin release have been a major focus of possible explanations for the association between low 25(OH)D levels and impaired glucose tolerance. Vitamin D receptors and vitamin D-binding proteins have been reported in pancreatic tissue, and calcium plays a major part in -cell insulin secretion [10].

2. Methods

We obtained approval from our Research Ethics Committee and informed written consents from the patients' guardians.

Our study was a cross-sectional study to evaluate the status of vitamin D in obese children and its relation with insulin sensitivity, resistance and glucose homeostasis. The study was carried out at Childhood Obesity Clinic and Clinical Pathology Department of National Nutrition Institute, Cairo, Egypt, during the period from January 1st 2015 to June1st 2016.

The Study population:

The study was conducted on 47 obese children (> +2 SD BMI for age and gender), that was selected according to the inclusion and exclusion criteria. This study was compared to 42 healthy children with matched age and gender as controls.

Inclusion criteria: Children eligible for the study had the following inclusion criteria: Children with simple obesity (BMI >+2 SD) according to the World Health Organization (WHO) Z score Child Growth charts based on weight, height, BMI and age. Age was between (6-16) years old.

Exclusion criteria: Identified or diagnosed causes of obesity like genetic, endocrinal disorders. Drugs that might affect both body weight and vitamin D level if used for long time as glucocorticoids, vitamin D supplements or anticonvulsant medications. Identified factors that might affect vitamin D metabolism or absorption like: Hepatic or renal disorders, metabolic disorders or mal-absorption syndromes and children that have Diabetes mellitus.

Our study group was subjected to the following:

(A) History taking:

Personal history: Name, age (date of birth), gender, address., sun-exposure: Timing and Duration of sun-exposure. Medical history: Any current or previous condition (renal-hepatic-endocrine) and drug consumption history (Steroids-Vitamin D-Anticonvulsants). Dietary history: Skipping breakfast meal, 24-hour recall, total caloric intake in relation to the patient's recommended dietary allowance, sugar –sweetened drink intake as sweetened juice and soda intake, milk intake and less healthy dietary pattern as less vegetables and fruit intake, more drinks, more fried. Saturated fat and high glycemic index food. Family history: F/HO of diseases (Obesity-Diabetes-Hypertension).

(B) Physical examination:

1. General examination: Skin Color (White-Light brown -Dark Skinned), presence of acanthosis nigricans (AN) and blood pressure.

2. Complete physical examination: Signs of any disease and pubertal assessment.

3. Anthropometric measurements: Weight (kg): Weight was measured using Weighing Scale with Height and Weight –Dial Type present at the Childhood Obesity Clinic at National Nutrition Institute. The measures were plotted by age on WHO gender-specific Z Score Growth charts for weight [11]. Height (cm): Using a height board (also known as a stadiometer) placed at a right angle between a level floor and a straight, vertical surface such as a wall or pillar to measure height. The BMI (kg/m^2) is a measure of a person's weight. Weight in kilograms divided by the square of height in meters (weight (kg)/height $(m)^2$) was used to measure BMI, which was then plotted by age on WHO gender-specific z score growth charts for BMI [11]**.** Circumferences: Waist circumference (WC) (cm): Waist circumference was calculated and plotted using percentiles for age and gender by Fernandez and colleagues, who released the first percentile tables for WC for US children in 2004 based on data from the Third National Health and Nutrition Examination Survey [12]**.** The following are the most important anthropometric measurements of the upper arm: MUAC (mid-upper arm circumference) (cm): Measuring the mid-upper arm circumference and plotting it on the Centers for Disease Control and Prevention's MUAC charts for age and gender for children and adolescents in the United States [13]. Triceps-skin fold thickness (TSF)(mm): Measuring triceps-skin fold thickness and it was plotted on Center for Disease Control and Prevention for TSF charts for age and gender for US Children and Adolescents [13]. BMI percentiles as median skinfold thicknesses of children who are overweight (≥85th percentile) or obese (≥95th percentile) [14].

(C) Laboratory tests:

Laboratory investigations include measurement of (fasting blood sugar [FBS], 2HRPP, Serum 25-hydroxy vitamin D and Fasting Insulin). Specimen Collection: 5ml venous blood was collected by venipuncture from fasting patient overnight (8hour), the sample was divided into 2 tubes. Plain tubes to obtain serum after allowing to clot and separating serum by centrifugation at room temperature. The collected serum was divided into 3 epinedorf and kept – 20 c until assay for (FBS, serum Vitamin D, Fasting Insulin). Another serum sample was collected for assay of 2-hour post-prandial blood sugar. Anti-coagulant coated tubes e.g. (EDITA) was used for assay of hemoglobin A1c

Statistical analysis:

Quantitative and qualitative approaches will be used in the statistical research. Statistical Qualitative data will be viewed as a percentage and as a number. Statistical Package for Social Science Program, version 21 will be used to conduct qualitative research, which will include descriptive and comparative methods.

3. Results

Table 1 shows that the percentage of pre-hypertensive and hypertensive children in obese subjects was more than normal non-obese with a statistical significance of<0.001. There was a significant difference regarding AN between obese and normal non –obese subjects with a P-value of <0.001.

There was a significant difference regarding TSF, MUAC and WC between obese and normal non-obese subjects where percentiles that diagnose overweight and obesity were higher in the obese group than the non-obese group with a statistical significance of<0.001. It showed that the comparison between both groups showed that all parameters showed a significant statistical difference between the median of anthropometric measurements and laboratory data except for FBS, HbA1c, 25(OH)D and Quantitative insulin sensitivity check index (Table 2).

Table 3 shows that BMI has a statistically significant positive association with systolic blood pressure and diastolic blood pressure of <0.001. There was a weak to moderate positive correlation between BMI and 2HRPP, fasting insulin, homeostatic model assessment of insulin resistance (HOMA-IR), and HOMA-B with a statistical significance of <0.05respectivly. There were no correlations between BMI and FBS, HbA1c, quantitative insulin sensitivity check index (QUICKI) or 25(OH)D.

Table 4 shows that there was a positive relationship between HOMA-IR and anthropometric measurements (Weight, BMI, BMI z score, WC, MUAC and TSF) which was statistically significant with a P- value <0.05. There was a moderate positive correlation between HOMA-IR and systolic blood pressure and diastolic blood pressure which was statistically highly significant with a P-value <0.05. There was a moderate positive correlation between HOMA-IR and FBS which was statistically significant with a P-value <0.05. There no relationship between HOMA-IR, 2HRPP, HbA1c and 25(OH)D.

Table 5 shows that there was no correlation between 25(OH)D and anthropometric measurements (except for height), glucose homeostatic indices or insulin resistance (IR) indices.

Our results demonstrated that increased number of meals more than 3 meals /day was associated with obesity. There was a significant difference regarding increased total caloric intake, total carbohydrate intake, protein intake and the way of meat cooking (fried and grilled way versus boiled way) between obese and normal weight subjects. There was no statistical difference between the main meal, taking breakfast meal, the way of vegetable cooking, healthy and unhealthy snacks or fat intake between cases and control. There was no statistical difference between total daily vitamin D, calcium and fiber intake between cases and control (Table 6).

Table (1): Comparison of general examination and anthropometric measurements between cases and control.

	Cases	Control		
	%	%	P-value	Significance
Blood pressure				
Systolic blood pressure			<0.001	S
Normotensive	46.8%	97.7%		
Pre-hypertension	42.5%	2.3%		
Hypertension	10.7%	0.0%		
Diastolic blood pressure			<0.001	S
Normotensive	27.6	80.9		
Pre-hypertension	46.8	19.1		
Hypertension	25.6	0.0		
Acanthosis nigricans			<0.001	S
Yes	59.6%	0.0%		
No	40.4%	100.0%		
Triceps-skin fold thickness			<0.001	S
Normal (5-85th)	0.0%	11.9%		
Overweight (85-94th)	27.7%	88.1%		
Obese (≥95 Percentile)	72.3%	0.0%		
Mid-upper arm circumference			<0.001	S

Wasted (<10th)	0.0%	31%		
Normal (10-89th)	30%	69%		
Obese (≥90 Percentile)	70.0%	0.0%		
Waist circumference			<0.001	S
<10th (Wasted)	0.0%	42.9%		
Normal (10-90th)	42.6%	57.1%		
Obese (>90th percentile)	57.4%	0.0%		

Table (2): Comparison between the obese subjects and the control.

	Obese subjects		Control			
	Median	**±Interquartile Range**	**Median**	**±Interquartile Range**	**P-value**	**Sig**
Age(Years)	9	±2	9	±3	>0.05	NS
Anthropometric Data						
Weight (kg)	49.250	±22.3	26.000	±6.5	<0.001	S
Height (cm)	135.600	±16.3	131.000	±14.5	<0.05	S
Body mass index (kg/m^2)	27.300	±6.40	15.000	±2.30	<0.001	S
Body mass index-z score	.64369	±.879	-.9889	±.3365	<0.001	S
Waist circumference (cm)	81.000	±12.1	56.000	±5.7	<0.001	S
Hip circumference (cm)	88.500	±14.3	65.500	±8.5	<0.001	S
Waist:Hip ratio	.9150	±.08	.8600	±.08	<0.05	S
MUAC (cm)	28.250	±4.3	19.000	±2.2	<0.001	S
TSF (mm)	25.000	±5.8	7.000	±3.0	<0.001	S
General examination						
Systolic blood pressure (mmHg)	100	±16	80.00	±20	<0.001	S
Diastolic blood pressure (mmHg)	70.00	±20	50.00	±10	<0.001	S
Laboratory Data						
25-hydroxy vitamin D (ng/ml)	27.700	±16.0	28.200	±13.9	>0.05	NS
Glucose homeostasis indices						
Fasting glucose (mg/dl)	88.500	±14.3	83.700	±16.0	>0.05	NS
2HRPP (mg/dl)	101.000	±17.8	98.000	±14.0	<0.05	S
Hemoglobin A1c	5.250	±1.3	5.800	±1.4	>0.05	NS
Insulin Sensitivity and Resistance Indices						
Fasting insulin (µIU/mL)	13.900	±12.67	6.480	±3.55	<.0.001	S
Glucose/Fasting insulin	6.2500	±6.10	12.370	±7.95	<0.001	S
HOMA-IR	2.9050	±2.698	1.4600	±1.285	<0.001	S
HOMA-B	296.45	±277	118.000	±175	<.0.001	S
QUICKI	0.5182	±0.518	0.522	±0.190	>0.05	NS

MUAC: mid-upper arm circumference; TSF: triceps skin fold thickness; 2HRPP: 2-hour postprandial blood glucose level; HOMA-IR: homeostatic model assessment of insulin resistance; QUICKI: quantitative insulin sensitivity check index

Table (3): Correlation between body mass index and other variables in studied subjects.

	Body mass index		
	R	**P**	**Significance**
Systolic blood pressure	.740**	.000	S
Diastolic blood pressure	.646**	.000	S
25-hydroxy vitamin D	.046**	.672	NS
Fasting blood sugar	.099*	.358	NS
2HRPP	.277**	.009	S
Hemoglobin A1c	. -.061-**	.570	NS
Fasting insulin	.320**	.002	S
1/Fasting insulin	-.450-**	.000	S
Glucose/Fasting insulin	-.441-**	.000	S

QUICKI	-.133	.215	NS
HOMA-IR	.266	.012	S
HOMA-B	.343**	.001	S

2HRPP: 2-hour postprandial blood glucose level; HOMA-IR: homeostatic model assessment of insulin resistance; QUICKI: quantitative insulin sensitivity check index

Table (4): Correlation between homeostatic model assessment of insulin resistance and other variables in studied subjects.

	Homeostatic model assessment of insulin resistance		
	r	**P**	**Significance**
Age	-.114-	.288	NS
Weight	.224*	.035	S
Height	.091	.397	NS
Body mass index	.266*	.012	S
Body mass index-z score	.307**	.004	S
Waist circumference	.272*	.010	S
Hip circumference	.191	.073	NS
Waist:Hip ratio	.084	.436	NS
MUAC	.259*	.014	S
TSF	.289**	.006	S
Systolic blood pressure	.303**	.004	S
Diastolic blood pressure	.333**	.001	S
25-hydroxy vitamin D	.188*	.079	NS
Fasting blood sugar	.316	. 003	S
2HRPP	.198**	.063	NS
Hemoglobin A1c	-.009-**	.933	NS

2HRPP: 2-hour postprandial blood glucose level; MUAC: mid-upper arm circumference; TSF: triceps skin fold thickness

Table (5): Linkage between 25-hydroxy vitamin D and othervariables in studied subjects.

	25-hydroxy vitamin D		
	r	**P**	**Significance**
Age	.064	.554	NS
Weight	.117	.280	NS
Height	.215*	.044	S
Body mass index	.046	.672	NS
Body mass index-z score	,030	.785	NS
Systolic blood pressure	.175	.103	NS
Diastolic blood pressure	.178	.097	NS
Waist circumference	.084	.436	NS
Hip circumference	.154	.151	NS
Waist:Hip ratio	.003	.981	NS
MUAC	.090	.405	NS
TSF	.085	.432	NS
Fasting blood sugar	.067	.535	NS
2HRPP	.031	. .777	NS
Hemoglobin A1c	.134	.212	NS
Fasting insulin	. .146	.177	NS
1/Fasting insulin	-.026-*	.810	NS
QUICKI	-.031	.774	NS
Glucose/Fasting insulin	-.024-	.823	NS
HOMA-IR	.188	.079	NS
HOMA-B	.066	.546	NS

MUAC: mid-upper arm circumference; TSF: triceps skin fold thickness; 2HRPP: 2-hour postprandial blood glucose level; HOMA-IR: homeostatic model assessment of insulin resistance; QUICKI: quantitative insulin sensitivity check index

Table (6): Comparison of caloric intake, macronutrient and some micronutrient intake between cases and control (24-hour –recall).

	Cases		Control			
	Median	**Interquartile ± Range**	**Median**	**Interquartile±range**	**P-value**	**Sig.**
T.kcal	1845.150	±831.93	1504.000	±464.75	<0.05	S
RDA T.kcal (%)	98.922	±48.18	79.869	±24.66	<0.001	S
Carbohydrate (gm)	241.250	±138.2	207.300	±68.0	<0.05	S
Fat (gm)	64.450	±46.7	53.900	±34.8	>0.05	NS
Protein (gm)	61.800	±12.3	53.900	±17.0	<0.05	**S**
RDA protein (%)	157.222	±38.94	141.165	±38.49	<0.05	**S**
Vitamin D (ug)	2.0150	±3.00	2.4500	±3.81	>0.05	NS
RDA vitamin D (%)	40.200	±61.40	46.100	±71.70	>0.05	NS
Calcium (mg)	513.750	±363.4	551.200	±497.0	>0.05	NS
RDA calcium (%)	57.553	±42.18	56.5154	±54.23	>0.05	NS
Fiber (gm)	7.100	±5.4	6.400	±4.8	>0.05	NS
RDA fiber (%)	26.800	±19.63	25.948	±17.49	>0.05	NS

T. kcal: RDA: recommended dietary allowance

4.Discussion

The study's aim was to compare vitamin D status in a group of obese kids and adolescents with a group of apparently healthy normal weight children attending the childhood clinic at National Nutrition Institute and examine the connection between 25(OH)D and IR, sensitivity, secretion and glucose homeostasis within the studied groups.

The study was conducted on 47 obese children (21 males & 26 females) aged 6 to 16 years and 42 age and gender matched children with ordinary weight and normal BMI (25 males & 17 females) as controls. The enrolled subjects were subjected to full history taking (Medical, Family and Dietary H/O), general examination, anthropometric measurements and laboratory evaluation.

In the current study, hypovitaminosis D was found in 55.1% of the whole sample (Vitamin D insufficiency was found in 39.3% of people, while vitamin D deficiency was found in 15.8%.), 55.3%of the obese (40.4% had vitamin D insufficiency and 14.9% had vitamin D deficiency), while 54.8%of control subjects (38.1% had vitamin D insufficiency and 16.7% had vitamin D deficiency).

There was no marked difference in vitamin D levels in our current study in between obese and normal weight subjects with the median of vitamin D in the obese group was (27.700± 16.0 ng/ml) and in normal non -obese group was (28.200±13 ng/ml), 55.3% females in obese group and 40.5% in non-obese group.

Our findings corroborate with the results of the study conducted by Poomthavorn et al. [15] in Thailand on 150 obese with (mean BMI = 28.6 ± 4.8 kg/m2; mean age 11.2 ± 2.6 years) and 29 non-obese with (mean BMI = 17 ± 2.7 kg/m^2; mean age 8.7± 1.5 years), 49.3% females in obese group and 86.2% females in non-obese group and found that the mean of 25(OH)D was(70.4 ± 16.5 nmol/l in obese (=28.16±6.6ng/ml); 68.9 ± 13.7 nmol/l (=27.56±2.64ng/ml)) in non- obese and 25(OH)D <50 nmol/L (<20ng/ml) present in 11.3% of obese and 10.3%of non-obese .

Also a study by Roth et al. [16] reported that 125 obese (mean BMI-Z = 2.7 ± 0.6) and 31 non-obese in Germany, age range (6-16 years) and found that 25(OH)D <75 nmol/L (<30ng/ml) present in 96%,<50 nmol/l (<20ng/ml) in 76% of participants and that vitamin D status was not significantly different between obese and non-obese subjects.

The same results were found by Erdönmez et al. [17] in Turkey who enrolled in their study 310 obese and non-obese (BMI ranged = 19.3–40.3 kg/m^2), mean age14 ± 2 years and 59% female and found that 25(OH)D = 25–50 nmol/l(10-20ng/ml) present in 53%, <25 nmol/l (<10 ng/ml) and there was no important association between vitamin D status and obesity in 12% of individuals.

Moreover in study of Stanley et al. [18] in USA on 15 obese (mean BMI = 34.4 ± 7.1 kg/m^2) and 15 matched non-obese females, age range 12–18 years and found that 25(OH)D there was no discernible difference between obese and non-obese (56.4 ± 28.9nmol/L)(22.56±11.56ng/ml)and(54.9 ±20.9nmol/l)(21.96±8.36ng/ml) respectively) and also Ashraf et al. [19] found the same results that vitamin D status was not significantly correlated with BMI.

Our results are in disagreement with Zakharova et al. [20] who mentioned that a substantial difference in vitamin D deficiency prevalence was observed among obese patients in a meta-analysis that included 15 studies (3,867 obese participants and 9,342 healthy individuals), with an odd ratio (95 percent) of 3.70 (2.33–5.06), and the prevalence of vitamin D deficiency among children and adolescents with obesity is extremely high: 96.0 percent in Germany and 78.4 percent in the US.

Also, Kelly et al. [21] conducted a study in USA on 85 obese and non-obese, age 4–18 years

with (BMI-Z = -1.2–4.1), and reported that 25(OH)D <75 nmol/L (<30ng/ml) present in 74%, <50 nmol/l (<20ng/ml) in 47% of participants and higher BMI-Z was negatively associated with 25(OH)D.

Moreover, Turer et al. [22] discovered that as compared to normal-weight children, overweight, obese, and extremely obese children had substantially higher adjusted odds of vitamin D deficiency, and that the incidence of vitamin D deficiency in healthy-weight, overweight, obese, and severely obese children was 21% (20%–22%), 29% (27%–31%), 34% (32%–36%), and 49% (45%–53%), respectively.

There are many studies in obese children to support that vitamin D status significantly influenced by adiposity.

Almezadah et al. [23] conducted a study in USA (Wisconsin) on 127 obese age 6–18 years with a mean (BMI = 37 ± 8.5 kg/m^2) and found that 25(OH)D <75 nmol/l (<30ng/ml) present in 74% of participants and Khadgawat et al. [24] study on 62 Indian obese children , age range between 6 and 17 years, with (BMI = 29 ± 4.8 kg/m^2), and reported that all participants are vitamin D deficient with a mean serum 25(OH)D of 8.5 ± 4.2 ng/ml.

Also, in USA (Wisconsin) Alemzadeh and Kichler [25] conducted a study on 133 obese (BMI-Z = 2.4 ± 0.4) age range3–18 years and found that fat mass negatively correlated with 25(OH)D and that lower 25(OH)D than those without metabolic syndrome.

Inadequate exposure to ultraviolet B radiation from the sun or inadequate vitamin D intake can cause vitamin D insufficiency and deficiency in otherwise healthy children. Hypovitaminosis D is more common in children with darker skin pigmentation and those who live in colder climates.

In our study hypovitaminosis D was found in 55% of apparently healthy normal weight children and this goes with the studies that conducted by Turer et al. [22] that estimated that there is prevalence of hypovitaminosis D in this group to be 21%.

Acanthosis nigricans has been described as an IR marker and a type 2 diabetes risk factor [26]. AN has been linked to IR and a much higher incidence of type 2 diabetes in children in a variety of studies [27].

The median of fasting insulin was higher in the presence of AN in our current research, but there was no substantial difference in HOMA-IR, HOMA-B, QUICKI, or glucose homeostatic measures.

Acanthosis nigricans may indicate the probability of IR, but it cannot identify it, according to Levy-Marchal et al. [28].

AN is an independent predictor for IR in obese Hispanic children at risk for type 2 diabetes, body obesity is the main determinant of insulin sensitivity, according to Kobaissi et al. [29]. Furthermore, it appears that scale scoring AN has limited clinical utility in evaluating the degree of IR.

Hirschler et al. [30] also found that AN indicates obesity but is not an independent marker of IR in the population studied, and that there was no univariate correlation between AN and the markers of IR (base insulinemia, HOMA-IR, and insulin growth factor binding protein 1), despite the fact that the community with AN had higher fasting insulin levels and HOMA-IR AN. In our current study there was no correlation between hypovitaminosis D and indices of insulin sensitivity and resistance indices (fasting insulin-1/fasting insulin, glucose/fasting Insulin, HOMA-IR or HOMA-B) and those indices correlated mostly with BMI, WC and TSF values are associated with obesity in children and adolescents, but not with 25(OH)D rates.

Our results corroborate with Torun et al. [4] who found that in obese children and adolescents, IR was primarily linked to BMI but not to 25(OH)D rates. They found a relation between obesity and certain biochemical parameters and IR but noted that varying levels of 25(OH)D in obese kids were not a reliable predictor of IR.

Our results are in disagreement with Flores Ruelas et al. [31] conducted a cross-sectional analysis on 227 obese children and adolescents ranging in age from 6 to 19 years to demonstrate the relation of hypovitaminosis D and IR and found that the average HOMA-IR rate was 3.16, with 70% of the individuals having been diagnosed with IR. Insulin levels and HOMA-IR were also greater in adolescents with hypovitaminosis. In comparison to the boys, the girls had higher insulin and HOMA-IR amounts.

Our results showed that there was a significant relation between duration of sunlight exposure and serum 25(OH)D as the subjects who had exposed to sunlight for more than 1 hour most times of the weak possessed a higher vitamin D level.

Our results corroborate those of Poomthavorn et al. [15], who discovered that, while food is unlikely to be the primary source of vitamin D for Thai children, sunlight exposure is; most of their children had a daily 1–1.5-hour outdoor physical education class at school during which they were exposed to sunlight. As a result, children in Thailand, which has a relatively sunny climate during the year, are more likely than children in high-latitude countries studied in other studies to get enough sunlight exposure. In addition, all of their patients were Asian, with light-brown skin.

Our results are in disagreement with the results by Reesukumal et al. [32] who reported that other factors, such as duration of sun exposure time, was not different between children with hypovitaminosis D and vitamin D sufficiency, also Rodríguez-Rodríguez et al. [33] concluded the same results.

Our results showed that vitamin D was not affected by skin type (the same concentrations in light coloured-skinned children and in darker-skinned children).

Our results are in agreement with the results by Jamali et al. [34] who reported that there was no significant difference between vitamin D status and skin color.

Our results are in disagreement with the results by Bonilla et al. [35] who reported that fair-skinned children had higher levels of 25(OH)D (0.6 nmol/l) per unit increase in skin colour, and that taking precautions against sunburn and skin cancer does not seem to negate the beneficial impact of having a less pigmented skin on vitamin D development.

Our results showed no significant difference in vitamin D levels in children residing in urban or rural areas. Our results are in accordance with the result by Jamali et al. [34] who concluded that no significant relationship was found between serum levels of vitamin D and residency.

Our results are in disagreement with the results by Rojroongwasinkulet et al. [36] conducted a study on 6–12.9 year-old healthy Thai children from four regions (central, north, northeast, and south) and found that 52.2 percent of urban children were vitamin D deficient. (n = 101) and 29.2% in rural areas (n = 217), using a cut-off value of < 20 ng/ml.

Our results reported a significant association between increased total caloric intake, total carbohydrate and protein intake, the way of meat cooking (fried way verus boiled way) and increased body weight and obesity.

Anderson et al. [37] conducted a study on 239 obese in New Zeeland, aged 5–17 years and reported that daily energy intake was above the recommended guidelines for 54%. Also, Ledoux et al. [38] concluded that energy intake was positively related to adiposity.

But Anderson et al. [37] reported that there was an association between obesity and skipping breakfast.

Our results are in disagreement with the results by Ledoux et al. [38] who stated that all macronutrients (in our study carbohydrate and protein not fat) and sugar sweetened beverages were positively related to adiposity but in agreement in that vegetables, which are low-energy-dense foods, were also positively related to adiposity.

In conclusion, with the rise of the epidemic of obesity worldwide and its associated co-morbidities as cardiovascular morbidities and decreased quality of life raise the concern for early detection and management.

Hypovitaminosis D is primarily caused by lifestyle factors (such as decreased outdoor activities) and external conditions (such as air pollution) that reduce exposure to sunlight, which is necessary for ultraviolet B-induced vitamin D development in the skin, as well as decreased intake of the recommended dietary allowance of vitamin D.

In conclusion, the study showed no significant difference in vitamin D status between obese and normal non-obese subjects, but there was significant difference between obese and normal weight subjects regarding insulin sensitivity and resistance indices and 2-hour postprandial blood glucose level (insulin sensitivity and resistance indices were related to adiposity indices) and the increase in systolic and diastolic blood pressure was related to adiposity indices and IR.

Abbreviations:

2HRPP 2-hour postprandial blood glucose level
25(OH)D 25-hydroxy vitamin D
AN acanthosis nigricans
BMI body mass index
FBS fasting blood sugar
HBA1c hemoglobin A1c
HOMA-IR homeostatic model assessment of insulin resistance.
IR insulin resistance
MUAC mid-upper arm circumference
TSF triceps-skin fold thickness
QUICKI quantitative insulin sensitivity check index
RDA recommended dietary allowance
WC waist circumference
WHO World Health Organization

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