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.

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 June 31st 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/m²) is a measure of a person's weight. Weight in kilograms divided by the square of height in meters (weight (kg)/height (m²)) 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 by Fernandez 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 3epinedorf 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 (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.

<table>
<thead>
<tr>
<th></th>
<th>Cases</th>
<th>Control</th>
<th>P-value</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>%</td>
<td>%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blood pressure</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic blood pressure</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normotensive</td>
<td>46.8%</td>
<td>97.7%</td>
<td>&lt;0.001</td>
<td>S</td>
</tr>
<tr>
<td>Pre-hypertension</td>
<td>42.5%</td>
<td>2.3%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>10.7%</td>
<td>0.0%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diastolic blood pressure</td>
<td></td>
<td></td>
<td>&lt;0.001</td>
<td>S</td>
</tr>
<tr>
<td>Normotensive</td>
<td>27.6</td>
<td>80.9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre-hypertension</td>
<td>46.8</td>
<td>19.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>25.6</td>
<td>0.0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acanthosis nigricans</td>
<td></td>
<td></td>
<td>&lt;0.001</td>
<td>S</td>
</tr>
<tr>
<td>Yes</td>
<td>59.6%</td>
<td>0.0%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>40.4%</td>
<td>100.0%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Triceps-skin fold thickness</td>
<td></td>
<td></td>
<td>&lt;0.001</td>
<td>S</td>
</tr>
<tr>
<td>Normal (5-85th)</td>
<td>0.0%</td>
<td>11.9%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overweight (85-94th)</td>
<td>27.7%</td>
<td>88.1%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Obese (≥95 Percentile)</td>
<td>72.3%</td>
<td>0.0%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mid-upper arm circumference</td>
<td></td>
<td></td>
<td>&lt;0.001</td>
<td>S</td>
</tr>
</tbody>
</table>
Table (2): Comparison between the obese subjects and the control.

<table>
<thead>
<tr>
<th></th>
<th>Obese subjects</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age (Years)</strong></td>
<td>9</td>
<td>9</td>
</tr>
<tr>
<td><strong>Weight (kg)</strong></td>
<td>49.250±22.3</td>
<td>26.000±6.5</td>
</tr>
<tr>
<td><strong>Height (cm)</strong></td>
<td>135.600±16.3</td>
<td>131.000±14.5</td>
</tr>
<tr>
<td><strong>Body mass index (kg/m²)</strong></td>
<td>27.300±6.40</td>
<td>15.000±2.30</td>
</tr>
<tr>
<td><strong>Waist circumference (cm)</strong></td>
<td>81.000±12.1</td>
<td>56.000±5.7</td>
</tr>
<tr>
<td><strong>Hip circumference (cm)</strong></td>
<td>88.500±14.3</td>
<td>65.500±8.5</td>
</tr>
<tr>
<td><strong>Waist:Hip ratio</strong></td>
<td>.9150±.08</td>
<td>.8600±.08</td>
</tr>
<tr>
<td><strong>MUAC (cm)</strong></td>
<td>28.250±4.3</td>
<td>19.000±2.2</td>
</tr>
<tr>
<td><strong>TSF (mm)</strong></td>
<td>25.000±5.8</td>
<td>7.000±3.0</td>
</tr>
</tbody>
</table>

**Anthropometric Data**

**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 |

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

<table>
<thead>
<tr>
<th></th>
<th>R</th>
<th>P</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic blood pressure</td>
<td>.740**</td>
<td>.000</td>
<td>S</td>
</tr>
<tr>
<td>Diastolic blood pressure</td>
<td>.646**</td>
<td>.000</td>
<td>S</td>
</tr>
<tr>
<td>25-hydroxy vitamin D</td>
<td>.046**</td>
<td>.672</td>
<td>NS</td>
</tr>
<tr>
<td>Fasting blood sugar</td>
<td>.099</td>
<td>.358</td>
<td>NS</td>
</tr>
<tr>
<td>2HRPP</td>
<td>.277**</td>
<td>.009</td>
<td>S</td>
</tr>
<tr>
<td>Hemoglobin A1c</td>
<td>-.061-</td>
<td>.570</td>
<td>NS</td>
</tr>
<tr>
<td>Fasting insulin</td>
<td>.320**</td>
<td>.002</td>
<td>S</td>
</tr>
<tr>
<td>1/Fasting insulin</td>
<td>-.450-</td>
<td>.000</td>
<td>S</td>
</tr>
<tr>
<td>Glucose/Fasting insulin</td>
<td>-.441-</td>
<td>.000</td>
<td>S</td>
</tr>
</tbody>
</table>

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.

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

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 other variables in studied subjects.

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

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
Our findings corroborate with the results of the study conducted by Poonthavorn 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 = 17.1 ± 2.7 kg/m², mean age 8.7 ± 3.7 years), 49.3% females in obese group and 86.2% females in non-obese group. 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 \((\text{BMI-Z} = -1.2–4.1)\), and reported that 
\(25(\text{OH})D <75 \text{ nmol/L} (<30\text{ng/ml})\) 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 \((\text{BMI} = 37 \pm 8.5 \text{ kg/m}^2)\) 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 \((\text{BMI} = 29 \pm 4.8 \text{ kg/m}^2)\), and reported 
that all participants are vitamin D deficient with a 
mean serum 25(OH)D of \(8.5 \pm 4.2\) ng/ml.

Also, in USA (Wisconsin) Alemezadeh 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 Rodriguez- 
Rodriguez et al. [33] concluded the same results.
Our results showed that vitamin D was not affected by skin type (the same concentrations in light skinned and dark 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 color, 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 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 versus boiled way) and increased body weight and obesity.

Anderson et al. [37] conducted a study on 239 obese in New Zealand, 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 macro nutrients (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 childhood 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 environmental (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-sk. fold thickness
- QUICKI = quantitative insulin sensitivity check index
- RDA = recommended dietary allowance
- WC = waist circumference
- WHO = World Health Organization

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