6-15 Years Vitamin D Levels in Normal and Obese Children

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Abstract

Purpose: Healthy living is number one priority of human beings. One of the obstacles awaiting urgent solution, on the way to healthy living is obesity. There are numerous reports regarding obesity and vitamin D relationship in the literature. Researchers tried to investigate obesity and vitamin D link in a low income district between normal and obese children.

Material and methods: Retrospectively BMI and vitamin D values of 149 children were compared in one year period. Children under 5 years could not be included because they took vitamin D supplements regularly. Both to reduce bias and nullify seasonal effects on vitamin D research was conducted all year long. Statistical analysis was conducted in each group separately to observe the relation between BMI and vitamin D values.

Results: An inverse relation was observed between BMI and vitamin D values. Results were similar to previous reports in the literature.

Key Words: BMI, Vitamin D, Obesity

Introduction

Being a widespread problem, obesity is on the agenda. To solve health problems efforts are focusing on solutions for obesity. In Turkey obesity prevalence of underweight, overweight and obesity among adolescent’s girls was 11.1%, 10.6% and 2.1% respectively. The overall prevalence of obesity in adult’s was 18.6% in the year 1990. In year 2000 with an increase rate of 17.7%, prevalence reached 21.9% (1). As is true for most of the countries overweight is more common in men and obesity is more prevalent among women also in Turkey (2).
Obesity associated diseases are progressively increasing. Association of obesity with diabetes mellitus, lipid metabolism disorders, hypertension, inflammatory diseases and some cancers is a well-known fact. Mechanisms in this association are under scrutiny. A prominent research field in this respect is vitamin D and its effects on immune system and inflammatory mediators. Deficiency of vitamin D levels are linked with a rising incidence of immune diseases like diabetes mellitus, multiple sclerosis, hypertension, cardiovascular disease and some common cancers (3-5).

Obesity-associated vitamin D insufficiency is likely due to the decreased bioavailability of vitamin D3 from cutaneous and dietary sources because of its deposition in body fat compartments(6). As we see interrelated and complicated mechanisms more clearly we come to a new understanding in this relation. Furthermore researchers observed the correlation between BMI and vitamin D levels in a low income area to test this relation.

**Material and Methods**

This study enrolled 91 normal 58 obese totaling 149 children and adolescents between 6-15 years of age who did not take vitamin D supplements beforehand and applied to Kağıthane State Hospital in the period from 1/1/2015 to 1/2/2016 .Data regarding body mass indexes and vitamin D levels were gathered from previous databases and results analyzed statistically.

BMI is calculated with weight/height$^2$ formula. Obese and normal are defined using BMI percentiles; children>2 years old with BMI>95th percentile meet the criterion for obesity (7)

In children, promotion of growth in different rates is determined and also measured by BMI. BMI changes substantially with age, rising steeply in infancy, falling during the preschool years, and then rising again into adulthood. For this reason, child BMI needs to be assessed using age related reference curves (8,9).

Venous blood samples were drawn from the antecubital region between 8.00-8.30 am. Serum 25 (OH) D levels were measured by liquid chromatography (Spektra 2000Sr).

**Statistical analysis**

This study is intended to observe the relation between BMI and vitamin D. In this context vitamin D data collected from categorically obese 58 and 91 normal patients. Collected data was analyzed in each group as to distribution incrementally. Because obese and normal BMI groups were different, this two groups’ comparison was done using Student’s t test.

**Results**

In table 1 distribution of obese and normal children in any age group is seen. Because vitamin D supplementation is given to all preschool children we did not include this age group .Due to methodic restriction we also did not include children who took vitamin D in the past 1 year. Because of this
restrictions and to prevent bias we included in a serial manner all subjects who met the necessary criteria of the research. In a one year period the distribution of subjects were haphazard so in some age groups there was elation an accumulation of subjects. But this fact was not a disadvantage for statistical analysis. For instance we see an accumulation of obese subjects in 9-12 age groups. Children with normal BMI consisted of 41 female and 50 male subjects. Obese group included 31 female and 27 male children. Gender difference is not detected-reported in the literature so we did not compare vitamin D between genders.

Table 1. Distribution of obese and normal BMI individuals in each age

<table>
<thead>
<tr>
<th>Age group</th>
<th>Number of obese individuals</th>
<th>Number of normal individuals</th>
</tr>
</thead>
<tbody>
<tr>
<td>6</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>7</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>8</td>
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<td>6</td>
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<tr>
<td>9</td>
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<tr>
<td>10</td>
<td>4</td>
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<tr>
<td>11</td>
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<tr>
<td>12</td>
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<td>18</td>
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<td>13</td>
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<tr>
<td>14</td>
<td>7</td>
<td>8</td>
</tr>
<tr>
<td>15</td>
<td>9</td>
<td>6</td>
</tr>
</tbody>
</table>

Table 2 The mean BMI and vitamin D value for each age

<table>
<thead>
<tr>
<th>Age</th>
<th>Number of obese individuals</th>
<th>Number of normal individuals</th>
<th>Obese</th>
<th>Normal</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean ± SD BMI</td>
<td>Mean Vit D±SD</td>
<td>Mean BMI</td>
<td>Mean vit D</td>
</tr>
<tr>
<td>6</td>
<td>4.4±6.78</td>
<td>18.9±5.12</td>
<td>14.9±3.45</td>
<td>13.4±3.45</td>
</tr>
<tr>
<td>7</td>
<td>5.5±6.61</td>
<td>17.8±4.22</td>
<td>16.6±4.44</td>
<td>15.3±4.01</td>
</tr>
<tr>
<td>8</td>
<td>5.9±7.03</td>
<td>26.2±6.99</td>
<td>18.1±5.01</td>
<td>13.1±3.98</td>
</tr>
<tr>
<td>9</td>
<td>5.6±5.59</td>
<td>20.8±5.24</td>
<td>17.9±4.98</td>
<td>15.6±4.22</td>
</tr>
<tr>
<td>10</td>
<td>6.7±6.67</td>
<td>13.2±4.45</td>
<td>18.9±4.47</td>
<td>11.8±3.44</td>
</tr>
<tr>
<td>11</td>
<td>7.8±7.04</td>
<td>14.8±4.89</td>
<td>17.8±4.87</td>
<td>11.4±3.15</td>
</tr>
<tr>
<td>12</td>
<td>8.6±6.66</td>
<td>14.7±4.73</td>
<td>18.9±5.00</td>
<td>10.3±3.02</td>
</tr>
</tbody>
</table>
We compared the relation of BMI with vitamin D values in each group separately. When we calculate the correlation between BMI and vitamin D values the correlation is -0.792. This shows an inverse relation between BMI and vitamin D in normal BMI children. Namely the higher the BMI the lower the vitamin D value.

In obese individuals the correlation coefficient is -0.401. This value also shows an inverse relation between this two variables but strength of relation is not as strong as is the case in normal BMI children. The difference between normal and obese groups is 2.262 with a significance of 0.017 according to T-test.

Discussion

Two pandemic diseases of the era which are obesity and deficiency of vitamin D show a high association (10). Association of these two entities began by epidemiological studies. Today this association is a well-known fact. In the light of epidemiological studies supporting this accompaniment, came animal and genetic studies and also the mechanisms to elucidate this relation were investigated. To date vitamin D supplementation did not increase weight loss when compared to placebo thus interventions through vitamin D supplementation is still uncertain and its influence on well-developed obesity is questionable (11). In clinical practice of obesity before bariatric surgery vitamin D status of individuals are screened according to current recommendations to identify individuals for postoperative deficiency (12).

We can discuss vitamin D and obesity relationship under multiple headings.

Vitamin D is fat soluble and thus stored in adipose tissue. It is shown by radioactive vitamin D₃ studies. (13) Vitamin D nuclear receptor is present in adipocyte. Also we observe 1 hydroxylase, 25 hydroxylase and vitamin D degrading 24 hydroxylase enzyme activity in adipose tissue. This three vitamin D metabolizing enzymes might influence the circulating 25OH Vitamin D levels with increasing bulk of adipose tissue in the body. Both subcutaneous and intraabdominal visceral fat possess these enzymatic activity. Both presence of VDR and enzymatic activity support paracrine and autocrine activity of 1.25 (OH)₂D in adipose tissue. In obese women researchers showed a reduced hydroxylase activity in subcutaneous fat but degrading enzyme activity did not change (14).

There is a strong relation between angiogenesis, angiogenesis inhibitors and obesity in experimental obesity studies. Vascular endothelial growth factor (VEGF) is secreted from adipocytes and foster fat tissue formation through the regulation of angiogenesis. The expression of VEGF has been shown to be reduced by vitamin D and analogues in multiple studies (15).

Increased inflammation and inflammation markers are another facet of obesity. Toll like receptors (TLR) have been incriminated in obesity associated inflammation Dynamic
role of adipose tissue in regulating inflammation and innate immunity through TLR has been proposed. It was observed that TLR-4 deficient mice were protected against obesity when given a high fat diet. On the other hand VDR null mice had less body fat and triglyceride and cholesterol levels when compared wild type mice. Also they did not show a propensity toward obesity and they are protected against diet induced obesity (16).

Vitamin D deficiency has long been associated with obesity, hypertriglyceridemia, insulin resistance and higher renin levels. In VDR knockout mice under expression of VDR resulted in high blood pressure and cardiac hypertrophy through over activation of renin angiotensin system (RAS) with increased renin and angiotensin II levels (17). Hypertriglyceridemia effects were also shown to be trough effect of angiotensin II on expression of apolipoprotein E in adipocyte which is a key regulator of cholesterol and lipid metabolism (18). Activation of RAS is a proven fact in obese patients and it is thought that mechanism is through the role of vitamin D in modulation of RAS. Vitamin D therapy in obese hypertensives modified mean arterial pressure similar to ACE inhibitors (19).

When considering vitamin D we must not put aside the role of the active metabolite of vitamin D; calcitriol. Calcitriol has been shown to increase matrix metalloproteinases (MMP) that have an important role in the pathogenesis of obesity. Also MMPs selectively inhibits Cox-2 activity and cause a decrease in prostaglandin synthesis. Calcitriol also inhibits the production of 25(OH)D production in the liver. It is this active metabolite of vitamin D is implemented in animal and genetic studies on obesity (20).

The vital question is whether vitamin D is the cause or the result of obesity (21). There is a report in favor of high prevalence of vitamin D deficiency among Spanish obese children and adolescents (Gutierrez-Medina S 2014) A Dutch study in multiethnic obese children reported a high prevalence of vitamin D insufficiency/deficiency (22). Researchers observed an inverse relation between BMI and vitamin D levels in both normal and obese children. Because of the small sample size we did not compare results between obese and normal children.
References