



NON-ALCOHOLIC FATTY LIVER DISEASE AND SERUM VITAMIN D

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ABSTRACT

Background: Non-alcoholic fatty liver disease (NAFLD) is associated with metabolic syndrome and insulin resistance. The frequency of NAFLD and vitamin D deficiency are increasing, worldwide. The aim of this study was to evaluate the relationship between vitamin D deficiency and NAFLD.

Methods: 148 patients who underwent abdominal ultrasonography (USG) were included in the study. The presence of NAFLD was determined by standard 2-dimensional abdominal ultrasonography. 25 OH vitamin D levels of the patients were measured. Vitamin D levels above 30 ng/ml were sufficient, between 20 and 30 ng/ml were insufficient and below 20 ng/ml were defined as deficiency. All the data provided from the study were recorded and evaluated by using SPSS (Windows 16.0). A p value ≤ 0.05 or confidence interval of 95% was considered statistically significant.

Results: The number of patients with adequate vitamin D levels was 14, the number of those with vitamin D deficiency was 12, and the number of those with severe vitamin D deficiency was 102. Of the 148 patients, 33 had hepatosteatosis according to the abdominal USG. 25 of patients with hepatosteatosis had severe vitamin D deficiency.

Conclusion: In our study, serum vitamin D levels were decreased in the majority of patients. Therefore, there was no statistical relation between NAFLD and serum vitamin D levels. Vitamin D deficiency was found to be severe in our hospital population.

KEYWORDS : Non-alcoholic fatty liver disease, serum vitamin D**INTRODUCTION**

Non-alcoholic fatty liver disease (NAFLD) is defined as fat accumulation in the liver in patients without chronic alcohol consumption (1). NAFLD associates with insulin resistance, diabetes, obesity and metabolic syndrome. NAFLD contains a broad spectrum of liver damage from simple liver fat to steatohepatitis, fibrosis and cirrhosis (2). Vitamin D is a prohormone and is converted into active form in the kidney. The most important effect of vitamin D is on regulation of calcium, phosphorus metabolism and bone mineralization. Some studies suggested a relationship between vitamin D deficiency and chronic inflammatory process (3,4). There is an evidence that vitamin D deficiency is related to insulin resistance (5,6). In this study we aimed to evaluate the association between NAFLD and vitamin D deficiency.

METHODS

This was a retrospective study and consisted of 148 patients who were admitted to the Haseki Training and Research Hospital's outpatient clinic for routine control. In this study, NAFLD was determined by abdominal ultrasound (USG) and serum vitamin D levels were measured. NAFLD was characterized by the presence of hepatic brightness, hepatorenal echo contrast, deep attenuation and vascular blurring on USG (106). Serum biochemical parameters were measured after an 8-hour fast using an Abbot Architect Analyzer System (IL, USA). Patients with an endocrinological disease and alcohol consumption were excluded from the study. Serum 25 OH vitamin D levels of the patients were analyzed. Serum vitamin D levels above 30 ng/ml were defined as sufficient, 20-30 ng/ml were insufficient and below 20 ng/ml were deficient. Data was evaluated by using SPSS (Windows 16.0) program. Statistical variables were expressed as mean and standard deviation and categorical variables were expressed as in percentage. The distributions were

evaluated by Kolmogorow-Smirnow test. To compare 2 groups, the normal distribution of the numerical data was evaluated by the student's t test. If the distribution was not normal then Mann withney u test was used for binary comparisons. When more than two group comparisons of the numerical variables were provided for the normal distribution condition, One Way Anova was performed with Kruskal Wallis test when not provided. Sub-group analyzes were interpreted according to Bonferroni correction in parametric test. Categorical variables were evaluated by chi-square test. Pearson's Spearman correlation test was used to compare two numerical data. A confidence interval of ≤ 0.05 or 95% was considered statistically significant.

RESULTS

The number of patients with adequate vitamin D levels was 14, with deficiency was 12, with severe deficiency was 102. A comparison of demographic and laboratory parameters of patients according to vitamin D level is shown in Table 1. There was no significant difference in terms of gender and age. There was no significant difference between the levels of urea, creatinine, lipid profile, electrolytes and serum vitamin D. There was severe vitamin D deficiency in 25 patients in this study (Table 2). There was no statistically significant difference in vitamin D levels between patients with and without hepatosteatosis.

Table 1. Comparison of patient characteristics and laboratory values.

	Vit D > 30 ng/ml (n:14)	Vit D 20-30 ng/ml (n:12)	Vit D < 20 ng/ml (n:122)	P value
Gender (male/female)	7/7	4/8	62/60	0.512

Age	69.93±13.41	70.42±10.70	67.25±15.31	0.660
FBG (mg/dl)	127.92±63.83	112.87±37.51	132.97±77.92	0.733
Total cholesterol (mg/dl)	177.60±73.2	175.47±65.9	164.87±59.40	0.724
HDL cholesterol (mg/dl)	41,72±14,3	35,11±10,7	35.29±12.52	0.268
LDL cholesterol (mg/dl)	108,74±48,2	114,09±51,0	99.70±48.33	0.574
Triglyceride (mg/dl)	162,18±135,7	131,54±58,3	159.15±133.61	0.790
Urea (mg/dl)	46,19±21,0	50,27±41,4	64.86±47.03	0.221
Creatinine (mg/dl)	1,10±0,5	0,88±0,6	1.39±1.21	0.238
ALT (U/L)	41.33±48.34	49.37±111.71	68.77±181.92	0.805
AST (U/L)	50.22±48.82	66.24±152.00	72.82±174.20	0.886
ALP (U/L)	187.99±222.81	138.68±128.03	137.62±111.31	0.371
GGT (U/L)	70.10±64.02	66.47±68.81	109.34±174.13	0.503
Sedimentation (mm/hr)	41±33	55±45	45±33	0.547
CRP (mg/l)	52.97±81.33	59.99±75.64	70.87±82.22	0.692
NAFLD (+)	5	3	25	0.328

(FBG: fasting blood glucose, ALT: alanine aminotransferase. AST: aspartate aminotransferase. ALP: alkaline phosphatase. GGT: gamma glutamyl transferase, CRP: c reactive protein, NAFLD: non-alcoholic fatty liver disease)

Table 2. Comparison of the presence of NAFLD with clinical and laboratory findings

	NAFLD (-) n:115	NAFLD (+) n:33	P value
Age	68.76±15.01	64.27±13.52	0.124
Hypertensive patients (n,%)	53, 46%	16, 48%	0.808
FBG (mg/dl)	130.65±78.82	132.53±59.33	0.904
Total cholesterol(mg/dl)	163.92±60.73	175.87±60.51	0.339
HDL cholesterol(mg/dl)	35.53±12.84	36.70±12.02	0.652
LDL cholesterol(mg/dl)	100.87±49.61	104.01±44.42	0.757
Triglyceride (mg/dl)	146.47±128.53	192.31±126.11	0.078
Urea (mg/dl)	65.47±46.90	49.50±35.52	0.039
Creatinine (mg/dl)	1.40±1.21	1.05±0.81	0.112
ALT (U/L)	70.79±187.22	43.06±72.03	0.407
AST (U/L)	78.13±183.60	41.40±43.21	0.052
ALP (U/L)	143.36±128.62	139.55±121.83	0.880
GGT (U/L)	96.93±144.53	119.95±209.72	0.471
Sedimentation (mm/hr)	46±34	46±34	0.960
CRP (mg/l)	73.91±85.81	48.71±60.13	0.117
Mean of vit D levels (ng/ml)	14.49±15.04	20.19±20.12	0.103
Vit D insufficiency (n)	97	25	0.42
Diabetic patients (n,%)	56, 53%	20, 62%	0.361

(FBG: fasting blood glucose, ALT: alanine aminotransferase. AST: aspartate aminotransferase. ALP: alkaline phosphatase. GGT: gamma glutamyl transferase, CRP: c reactive protein)

DISCUSSION

Vitamin D deficiency is a global health problem and may contribute to the pathogenesis of many disorders such as obesity, metabolic syndrome and type 2 diabetes (7,8). In our study, the relationship between the presence of hepatosteatosis and age, duration of diabetes and serum vitamin D levels were evaluated. No statistically significant relationship was found between age, fasting blood glucose, HBA1c levels and duration of diabetes and hepatosteatosis. Low serum 25 (OH) D concentrations, body mass index (≥30 kg / m²) and obesity were shown to be inversely proportional (9). Vitamin D deficiency may play a role in the progression of liver fat (10). Vitamin D has immunomodulatory properties and helps explain the effect of this vitamin on the progression of liver fats (11). In our study, there was no significant relationship between vitamin D levels in patients with and without liver fatty tissue. In our study, serum vitamin D levels were found to be deficient in 84% of the patients. Only 14 patients had sufficient serum vitamin D levels. Vitamin D deficiency

is associated with insulin resistance, inflammation in the liver and increased oxidative stress. Decreased serum vitamin D leads to the formation of oxidative stress, through the production of proinflammatory cytokines (12). In our study, no statistically significant relationship was found between serum AST, ALT, GGT and ALP levels and hepatosteatosis independent of vitamin D levels. In some studies, an increase in liver enzymes has been reported in individuals developing NAFLD (13,14). As a result, there is evidence that serum vitamin D deficiency is related to insulin resistance, visceral adipose tissue increase and hepatosteatosis. In our study, there was no positive relationship between serum D vitamin level and advanced deficiency level. Vitamin D deficiency was found in our hospital population. In our study, there was no association between NAFLD and vitamin D deficiency.

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