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Original Research Article.

Relation Between Non-Alcoholic Fatty Liver Disease and Vitamin D Level

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ABSTRACT

Introduction: Nonalcoholic fatty liver disease (NAFLD) ranging from simple steatosis to nonalcoholic steatohepatitis (NASH) to cirrhosis. Patients with NAFLD usually meet the diagnostic criteria for the metabolic syndrome and have a greater risk for cardiovascular disease. Our primary goal is to assess the prevalence of the NAFLD and its association with the cardiovascular risk factors and vitamin D level.

Method: The study was conducted on 121 male patients attending the primary care offices in Taif University and asked to participate in the study after their consent, aged 20-63 years. Patients with known CAD, peripheral artery disease, or stroke were excluded from the study. Baseline characteristics and anthropometrics measures were obtained by the researchers. Laboratory test were done for all the participants.

Results: Compared to patients without NAFLD, those with NAFLD were younger in age (P 0.535), had lower SBP (P 0.087), higher DBP (P 0.530), lower WC and BMI (P > 0.05), and significantly higher FRS (P 0.036).

Regarding the laboratory data and when compared those without NAFLD, those with NAFLD had non-statically significant better lipid profiles except for higher TG level (P 0.479) but had higher FBS (P 0.812) and Hs-CRP (0.789).

Those with NAFLD were non-significantly more likely to have vitamin D deficiency and were less likely to have vitamin D sufficiency.

Conclusion: 46.3% of the participants have NAFLD and despite having lower SBP and better lipid profiles, they have higher FRS and inflammatory markers when compared to those without NAFLD.

Keyword: Vitamin D, NAFLD, Obesity, Fatty, Liver.

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INTRODUCTION

Nonalcoholic fatty liver disease (NAFLD) ranging from simple steatosis to nonalcoholic steatohepatitis (NASH) to cirrhosis. NAFLD has reached an epidemic nature and is the most common cause of chronic liver disease in Western countries.[1] 20-30% of the adult whom are living in the western countries have NAFLD, with an increased incidence from 70 to 90% with obesity and diabetes mellitus (DM). These patients are also at increased risk for the development of liver cirrhosis.[2]

Patients with NAFLD usually meet the diagnostic criteria for the metabolic syndrome (i.e., hypertension, dyslipidemia, hyperglycemia and abdominal obesity) and therefore have a greater risk for cardiovascular disease.[3] Young patients with NAFLD who are not obese and do not have diabetes or hypertension have an echocardiographic features of early left ventricular dysfunction and impaired left ventricular energy metabolism.[4] One community-based cohort study of 2088 male workers found that ultrasonographically diagnosed NAFLD was associated with an increased prevalence of ischemic heart disease.[5] NAFLD was associated with more severe coronary artery disease independently of established risk factors in patients consecutively referred for elective coronary angiography.[6] Cardiovascular disease was the most frequent cause of death in 173 patients with biopsy-proven NAFLD disease who were followed for 13 years.[7]

The main dietary source of vitamin D is fatty ocean fish and other marine foods. In the high latitude area during the winter months, very little previtamin D is formed.[8] It is estimated that one billion people worldwide are vitamin D deficient or insufficient.[9] The prevalence of hypovitaminosis D is ranging from 5-30% in the adult population, but it reaches a peak of 75% in patients with metabolic syndrome (MetS).

Coronary artery disease (CAD) is a major public health problem worldwide. It accounted for 25-30% of deaths in most industrialized countries. Data on the prevalence of CAD in Saudi

is limited. Lifestyle changes as increasing overweight and obesity, tobacco use, along with the rapid increase in the incidence of diabetes are the major risk factors for CAD.[10] The relationship between excess body weight and the risk of CAD is complex. Abdominal obesity is considered to play a fundamental role in the etiology of CAD through adversely affecting several established risk factors.[11] Recently, many risk score as the Framingham risk score (FRS) is designed to assess the risk of CAD using different clinical anthropometric and laboratory parameters.[12]

Our primary goal is to assess the prevalence of the NAFLD and its association with the cardiovascular risk factors and vitamin D level among Taif University male.

METHOD

The study was conducted on 121 male patients attending the primary care offices in Taif University and asked to participate in the study after their consent, aged 20-63 years. Patients with known CAD, peripheral artery disease, or stroke were excluded from the study. Participated physicians measured body weight, height, blood pressure, and waist circumference (WC) according to written, standardized instructions given in a manual. Systolic and diastolic blood pressure was measured by indirect cuff sphygmomanometer after several minutes of rest in sitting position with the use of an appropriate cuff size. Weight made by digital scales without heavy clothing; height barefoot by portable stadiometer; the waist was measured at the end of a gentle expiration midway between the lowest rib and iliac crest, with the patient standing. BMI (weight in kg divided by the square of height in meters) was calculated.

After their consent, all studied population was subjected to:

1. Full history taking with particular emphasis on age, history of any systemic diseases, e.g., HTN, DM, dyslipidemia or history of any associated diseases and drug intake.

2. Thorough clinical examination. 3- Blood sampling and analysis: 6 ml venous blood sample was collected between 8-9 AM after overnight fasting. 2 ml was collected in an EDTA tube with gentile mix, and then preserved at refrigerator not more than one week for glycated hemoglobin analysis. The other 4 ml was collected in plain tube, incubated for 30 minutes at 37 °C, and then centrifuged at 3000 rpm for 10 minutes. Serum was separated in a dry clean tube and stored at -20 °C till assay time for doing the other analysis parameters. Another 2 ml of venous blood was collected in plain tube, serum separated as previous and preserved for 2 hours post prandial blood glucose analysis. At the assay time, serum samples were left to take room temperature for doing the following parameters:

a) Fasting and two hours postprandial blood glucose, according to a glucose-oxidase method using kits from UDI Diagnostic.
b) Glycated hemoglobin A1c was measured using column chromatography method by commercial Kit from Biosystem diagnostic.
c) Serum TG, TC was estimated according to (GPO-POD) method using kits from Spinreact Diagnostics.
d) Serum HDL level was estimated by "enzymatic colorimetric method using kits from Spinreact. Serum LDL level was calculated using Friedewald's formula if the triglycerides were less than 4.5 mmol/l, as following: LDL= total cholesterol – HDL-cholesterol – triglycerides/5.
e) High sensitivity CRP was done using a fully automated analyzer IMAGE from Bechman coulter Diagnostics g-Serum 25-(OH) Vitamin D was measured by Abbott Architect i1000 Chemiflex device (kits manufactured by Abbott#3L52-25).

Statistical Analysis

Values were expressed as mean ±SD. The statistical analysis of the results was performed based on the conventional standard statistical procedures using computed statistical analysis by SPSS, version 20.0 of Microsoft windows 7. All variables were tested for normality of distribution. Paired-samples t test was applied to compare between parametric values; Pearson's correlation with correlation coefficient was applied for parametric results. The significant difference was considered at $p<0.05$.

Table 1: Clinical characteristics based on the NAFLD status

	NAFLD	Non-NAFLD	P
Age	34.6±10.6	35.9±12.6	0.535
SBP	129.6±14.4	133.9±13.7	0.087
DBP	76.1±10.2	74.9±9.7	0.530
WC	93.4±22.2	96.1±20.9	0.488
Weight	79.4±16.9	82.7±15.7	0.259
BMI	27.2±5.6	28.9±5.0	0.070
FRS	5.14±4.5	3.51±3.5	0.036

Table 2: Biochemical characteristics based on the NAFLD status

	NAFLD	Non- NAFLD	P
LDL	114.9±30.3	125.7±51.1	0.169
HDL	44.8±8.3	42.9±6.7	0.191
TG	139.4±113.6	126.4±75.5	0.479
TC	190.2±28.4	194.3±53.0	0.615
Non-HDL cholesterol	145.5±28.5	151.1±51.3	0.474
FBS	98.5±28.7	97.4±17.7	0.812
Hs-CRP	0.45±0.68	0.42±0.37	0.789
Vitamin D	17.45±6.14	17.6±5.9	0.886
TSH	2.1±1.2	1.9±1.9	0.428

Table 3: Vitamin D status according to the level

< 20 ng/mL	**Deficient**
20 - 30 ng/mL	**Insufficient**
> 30 ng/mL	**Optimal**

Table 4: Vitamin D diagnosis distribution in those with NAFLD

Vit D level	N	Category	%
< 20 ng/mL	42	Deficient	75
20 - 30 ng/mL	13	Insufficient	23
> 30 ng/mL	1	Optimal	2

Table 5: Vitamin D diagnosis distribution in those with non-NAFLD

Vit D level	N	Category	%
< 20 ng/mL	48	Deficient	69
20 - 30 ng/mL	16	Insufficient	23
> 30 ng/mL	1	Optimal	8

Table 6: Comparison of the vitamin D level according to NAFLD status

	Deficient Vit. D N (%)	Insufficient Vit. D N (%)	Optimal Vit. D N (%)
NAFLD (56)	42 (75)	13 (23)	1 (2)
Non- NAFLD (65)	48(69)	16 (23)	1 (8)
P	0.459	1.0000	0.138

RESULTS

Overall 46.3% of the whole cohort has NAFLD. Compared to patients without NAFLD, those with NAFLD were younger in age (P 0.535), had lower SBP (P 0.087), higher DBP (P 0.530), lower WC and BMI (P > 0.05), and significantly higher FRS (P 0.036) (Table 1).

Regarding the laboratory data and when compared those without NAFLD, those with NAFLD had lower LDL level (P 0.169), higher HDL level (P 0.191), higher TG level (P 0.479), lower TC level (P 0.615), lower non-HDL level (P 0.474), higher FBS (P 0.812), higher Hs-CRP (0.789), lower vitamin D level (P 0.886), and higher TSH level (P 0.428) (Table 2).

Vitamin D diagnosis according to the level as listed in Table 3. Those with NAFLD were more likely to have vitamin D deficiency and were less likely to have vitamin D sufficiency (Table 4, 5). The difference observed in the vitamin D level according to the NAFLD status didn't reach statistically significant (Table 5).

DISCUSSION

Almost half of our sample has NAFLD. It was reported that prevalence of NAFLD in Saudi Arabia ranging from 7-10% with around 10-20% of those patients may go on to develop advanced fibrosis and cirrhosis. DM, obesity and hyperlipidemia are all a major risk factors for NAFLD and those risk factors are extremely common in Saudi Arabia.[13]

Interestingly those in our cohort with NAFLD had better lipid profile except for TG level. In the other hand our patients with NAFLD had higher FBS, hs-CRP and FRS.

Some researchers found that patients with NAFLD have impaired flow-mediated vasodilatation and increased carotid-artery intimal thickness, two reliable markers of subclinical atherosclerosis that are independent of obesity and other established risk factors.[14] Some studies found that NAFLD diagnosed by ultrasonography is strongly associated with increased prevalence of carotid atherosclerotic plaques.[15]

Other studies have shown that there is no significant association between NAFLD and either carotid-artery intimal thickness or carotid-artery calcium.[16] Our cohort showed that 75% of the participants had vitamin D deficiency. Nowadays, vitamin D deficiency is very common in obese individuals. It is known that there is a relationship between decreased vitamin D concentrations in circulation and obesity. However, the mechanisms have not yet been clarified. Vitamin D deficiency is thought to be associated with the presence of cardiometabolic risk in obese individuals.[17] A range of serious diseases may be associated with vitamin D deficiency as cardiovascular disease, infectious diseases, autoimmune diseases, T2D, MetS and cancer.[11]

Our study limitation includes the small sample size, single center, and male limited study. Our strength includes comprehensive laboratory and clinical data.

CONCLUSION

46.3% of the participants have NAFLD and despite having lower SBP and better lipid profiles, they have higher FRS and inflammatory markers when compared to those without NAFLD. Although those with NAFLD were more likely to be vitamin D deficient but this wasn't statistically significant.

SOURCE OF SUPPORT

Taif University, Taif, Saudi Arabia.

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