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Liver fibrosis scores can also be used as a marker of cardiovascular risk in non-alcoholic fatty liver disease

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ABSTRACT

OBJECTIVE: This study was designed to evaluate the relationship between Atherogenic Plasma Index (API), a cardiovascular event risk marker, and hepatic fibrosis level in non-alcoholic fatty liver disease (NAFLD) patients.

METHODS: 129 people over the age of 18 with NAFLD were included in the study. Fasting blood glucose (FBG), HOMA-IR, HbA1C, aspartate aminotransferase (AST), alanine aminotransferase (ALT), gamma glutamyl transferase (GGT), albumin, triglyceride (TG), total cholesterol (TC), LDL-cholesterol (LDL-C), HDL-cholesterol (HDL-C), nonHDL-cholesterol (nonHDL-C), direct bilirubin, TC/HDL-C, LDL-C/HDL-C and TG/HDL-C ratios were calculated. NAFLD fibrosis score (NFS), FIB-4, APRI scores were determinated. It was classified as low, high and intermediate. The API was calculated and its relationship with the level of hepatic fibrosis was evaluated.

RESULTS: The API level in the high group of NFS is higher than that in the low group. Similarly, the API level in the high group of FIB-4 is higher than that in the low group. The API in the low group of APRI are lower than the ID group. There is a positive correlation between API and NFS, FIB-4, APRI scores in all NAFLD subjects. There is a positive correlation between API and NFS, FIB-4, APRI scores in all NAFLD subjects in the study.

CONCLUSION: In this study, we found that there was a relationship between FIB-4, NFS, APRI scores, API and dyslipidemia in people with NAFLD, and that as the level of fibrosis increased, the risk of cardiovascular event also increased. Liver fibrosis scores can also be used as cardiovascular risk markers.

Keywords: Atherogenic plasma index; FIB-4 score; non-alcoholic fatty liver disease.

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Non-alcoholic fatty liver disease (NAFLD) is the most common liver disease in the world, with a prevalence of approximately 25% in the adult population. The disease is becoming a public health problem in the world with each passing day [1]. NAFLD is often accompanied by obesity, type 2 diabetes mellitus (DM), hyperlipidemia, hypertension and metabolic syndrome. NAFLD patients have a higher mortality than the general population. The likelihood of a non-fatal cardiovascular disease or death from cardiac events is twice as high as that of the general population. NAFLD patients' rise in morbidity and mortality is not solely caused by liver diseases, but also by cardiovascular diseases (CVD) [2–4].

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Epidemiological studies have shown that NAFLD and increased CVD risk are connected.

The histology cohort study revealed that biopsy-proven NAFLD is linked to major adverse cardiovascular events and is associated with ischemic heart disease, stroke, and congestive heart failure [1]. According to a meta-analysis, NAFLD is a factor that raises the risk of myocardial infarction, ischemic stroke, atrial fibrillation, and heart failure [5]. The relationship between NAFLD and CVD is bidirectional [6]. Considering the close relationship of NAFLD with metabolic syndrome components, CVD risk is expected to increase in those with NAFLD. However, it has also been suggested that NAFLD may be a cause of CVD independent of classical cardiovascular risk factors [7]. In addition to traditional cardiovascular risk factors, there are several secondary risk factors, such as hyperuricemia, hypoadiponectinemia, proinflammatory cytokines, and endothelial dysfunction, that are believed to be behind the increased incidence of CVD in NAFLD [8]. In the cardiovascular system in NAFLD cases; coronary artery disease (CAD), ischemic stroke, arrhythmias, diastolic dysfunction, heart failure with preserved ejection fraction, and valvular diseases [9–11].

The level of liver fibrosis is a strong finding indicating the progression of NAFLD's clinical course and the mortality associated with the liver [12, 13]. The most important finding that determines the natural history of NAFLD is this hepatic fibrosis level. In the natural course of the disease in any time period; It can be progress, stagnation or regression. The existence of risk factors increases the progression of fibrosis. It is thought that there may be a relationship between hepatic fibrosis and cardiovascular complications [14]. Liver biopsy is the gold standard for evaluating liver fibrosis. Non-invasive tests are preferred because they are easier and more economical. To evaluate hepatic fibrosis, noninvasive tests such as NFS, FIB -4, APRI, Forns are used in clinical practice.

The atherogenic Plasma index (API), a new quantitative index used to evaluate lipid levels, is a well-known predictive and prognostic biomarker for atherosclerotic cardiovascular diseases [15, 16]. The initial description of AIP was given by Dobiárco and Fröhlich in 2001 [17]. API has been shown to be a strong predictor in diseases that cause major cardiovascular death, such as CAD and acute coronary syndrome. Many studies have shown that API has a high level of accuracy in estimating the risk of atherosclerosis, CAD, myocardial infarction [18, 19] and hyperuricemia [20, 21].

Highlight key points

- There is a relationship between FIB-4, NFS, APRI scores, and API in people with NAFLD.
- There is a positive correlation between API and APRI, FIB-4, NFS.
- As the level of fibrosis increases in NAFLD, the risk of cardiovascular events also increases.
- Fibrosis scores may also predict the risk of cardiovascular events.

Although numerous studies recently examined the relationship between NAFLD and API, no studies examined the relationship between liver fibrosis level and API [22]. This study was designed to examine the correlation between the level of hepatic fibrosis and the API to determine cardiovascular risk.

MATERIALS AND METHODS

Study Population

Between January 2019 and September 2022 in the internal medicine department of our hospital, 129 people over the age of 18 with ultrasonographic fatty liver were included in the research. While determining the people to be included in the study, as the exclusion criteria; viral hepatitis, autoimmune hepatitis, toxic hepatitis, cirrhosis, biliary tract diseases, pregnancy, lactation, cancer, those who use drugs that will affect lipid profile and liver enzymes, and those who use alcohol (20 g/day for women, 30 g/day for men) were determined. This study was conducted in accordance with the Declaration of Helsinki, ethics committee approval was obtained from the Istanbul University of Health Sciences Umraniye Training and Research Hospital Clinical Research Ethics Committee (date: 24.11.2022, number: 2022/354).

Measurements

Demographic information (age, gender, etc) of the participants in the study was recorded. The use of standardized equipment was employed to measure weight and height. Body mass index (BMI) was calculated. A venoz blood sample was taken after a 12-hour fast, and the required parameters were analyzed by using the Abbott i8000 device and Abbott kits in our central laboratory.Fasting blood glucose (FBG) levels were measured

by hexokinase method and aspartate aminotransferase (AST), alanine aminotransferase (ALT) were evaluated by enzymatic method. Triglyceride (TG), and HDL-cholesterol (HDL-C) were evaluated by photometric method. The insulin levels were studied using the Abbott i16000 device and the chemiluminescent microparticle immunoassay (CMIA) method.

HOMA-IR was calculated using the formula:

HOMA-IR=[fasting insulin (μIU/mL)×fasting glucose (mg/dL)]/405] [23].

Liver Fibrosis Scores

The fatty liver was determined by a skilled radiologist using ultrasonography. To determine the level of NAFLD, four parameters (vascular blur, deep attenuation, liver brightness, echo contrast) in the scoring system proposed by Hamuguchi et al. [24] were examined.

The NAFLD fibrosis score (NFS) was determined using the formula.

NFS: -1.675+0.037 × age (years) + 0.094×BMI (kg/m^2)+1.13 pre-diabetes /diabetes (yes=, no=0)+ 0.9×AST [IU/L]/ALT[IU/L]−0.013×platelet count (109/L)−0.66×albumin(g/dL) [25].

The probability of fibrosis was low NFS<-1.445.

The probability of fibrosis was high NFS>0.676.

The probability of fibrosis was Indeterminated NFS -1.445- 0.676.

The FIB-4 score was determined using the formula.

FIB-4 score: (Age [years]×AST[U/L])/(platelet [109] X√ALT [U/L]) [26].

The probability of fibrosis was low FIB-4<1.30.

The probability of fibrosis was high FIB-4>2.67.

The probability of fibrosis was indeterminate FIB-4 between 1.30-2.67.

The APRI score was determined using the formula.

APRI score: [(AST/upper limit of normal)/Platelet Count (109/l)]×100.

The probability of fibrosis was low APRI<0.5.

The probability of fibrosis was high APRI>1.5.

The probability of fibrosis was indeterminate APRI between 0.5-1.5.

Atherogenic Plasma Index (API)

API was calculated [log(TG/HDL-C)] by taking the logarithm of the ratio of TG to HDL-C [27].

Statistical Analysis

Statistical analysis was performed using the MedCalc® Statistical Software version 19.7.2 (MedCalc Software Ltd, Ostend, Belgium; https://www.medcalc.org; 2021). The Shapiro-Wilk test was used for continuous variables. Frequency (n) and percentage (%) were used to express categorical variables. The Ki-Square (or Fisher's Precise Test, where appropriate) test was used to compare categorical variables. The Kruskal Wallis test was used to compare more than two independent non-normal distribution group continuous variables. The Bonferroni-tuned Mann Whitney test was used for post-hoc comparisons. An ANOVA test was used to compare two or more independent normally distributed group continuous variables. For Post Hoc Comparisons Bonferroni adjusted Tamhane's T^2 test was used. The correlation between two normally distributed variables was used by Pearson Correlation coefficient, while for non-normal data, spearman Correlation coefficient was used.

RESULTS

Of the 129 participants in the study, 68.2% (n=88) were female and 31.8% (n=41) were male. Their mean age was 53.06 (53.6+10.9) years. According to their blood glucose, 58.9% them were normal, 28.7% were prediabetic, and 12.4% were DM (Table 1). According to the participants' FIB-4 scores, 17% are in the high fibrosis(HF) groups, 31% Indeterminated fibrosis (IDF) groups, and 52 % low fibrosis (LF) groups.

According to the NFS score, 17.1% HF, 28.7% IDF, and 54.3% LF group. According to the APRI score, 7% are in the IDF group and 93% are in the LF group. There are no participants with a High group. (Table 2).

In terms of all parameters, all three groups determined by FIB-4 scoring were compared.

The HF group has a higher level of API, TG, TC, non HDL-C, non HDL-C/ HDL-C, TC /HDL-C, LDL-C/HDL-C compared to the LF group (p<0.001).

In terms of all parameters, all three groups determined by NFS classification were compared. The HF group has a higher level of API, TG, TC, non HDL-C, non HDL-C/ HDL-C, TC /HDL-C, LDL-C/HDL-C compared to the LF group(p<0.001).

In terms of all parameters, all three groups determined by APRI classification were compared. Significantly lower API and TG were found in the LF group than in the IDF group (p<0.01) (Table 3).

Table 1. Demographic characteristicsand laboratory parameters

	n	%
Gender		
Female	88	68.2
Male	41	31.8
DM status		
Normal	76	58.9
Prediabetes	37	28.7
DM	16	12.4
	Mean±SD	Med (min-max)
Age (year)	53.6±10.9	54 (27–84)
BMI (kg/m^2)	28.9±4.3	27 (22.5–43)
FBG (mg/dl)	104±25.5	98 (77–241)
AST (IU/l)	25.4±10.9	22 (7–60)
ALT (U/l)	25.5±13.8	21 (8–62)
Platelet (×10^9/l)	255.4±63.4	255 (118–500)
Albumin (gr/dl)	4.7±2.9	4.5 (3.4–37)
Homa-IR	3.9±3.9	2.8 (1.4–38)
HBA1c (%)	5.8±1	5.5 (4.6–11)
API	0.6±0.3	0.6 (0–1.3)
TG (mg/dl)	204.5±113.3	165 (51–536)
TC (mg/dl)	204.6±39.6	203 (102–299)
Non-HDL-C	158.4±41.7	157 (54–242)
HDL-C (mg/dl)	45.8±10.5	44 (25–77)
Non-HDL/HDL	3.7±1.4	3.6 (0.7–8.5)
TC/HDL-C	5.2±5.3	4.5 (1.7–62.5)
LDL-C (mg/dl)	125.8±33.7	124 (46–211)
LDL-C /HDL-C	2.9±1.1	2.9 (0.9–6.7)
GGT (U/l)	43.3±51.9	26 (7–336)

DM; Diabetes mellitus; SD: Standard deviation; BMI:Body mass index; FBG: Fasting blood glucose; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; API : Atherogenic plasma index; TG: Triglyceride; TC: Total cholesterol; Non-HDL-C: Non-HDL- cholesterol; HDL-C: HDL- Cholesterol; LDL-C: LDL-cholesterol; GGT: Gamma glutamil transferase

Table 2. Fibrosis scoring and classifications (n=129)

FIB-4, mean+SD	1.2+0.7
	%
High	17
ID	31
Low	52
NFS, mean+SD	-1.6+1.6
	%
High	17.1
ID	28.7
Low	54.3
APRI, mean+SD	0.3+0.2
	%
High	-
ID	7
Low	93

SD: Standard deviation; FIB-4: <1.3low, 1.3-2.67 ID, >2.67 high; NFS: <-1.445 low -1.445-0.676 ID, >0.676 High; APRI: <0.5low, 0.5-1.5I D, >1.5 high; NFS: NAFLD fibrosis score

The correlation between fibrosis scores of people with NAFLD and API was investigated. There is a positive, high correlation between API and FIB-4 score (r=0.825, p<0.001) (Fig. 1). There is a positive, high correlation between API and NFS (r=0.753 p<0.001) (Fig. 2). There is a positive, moderate correlation between API and APRI (r=0.666, p<0.001) (Fig. 3).

DISCUSSION

In this study, we found that there is a relationship between FIB-4, NFS, APRI scores, API and dyslipidemia in people with NAFLD. In other words, as the level of fibrosis increases, the risk of CVD also increases. Liver fibrosis scores can also be used as cardiovascular risk markers.

The most common liver disease in the world is NAFLD, with a prevalence of approximately 25% in among the adults. The disease is becoming a public health problem in the world with each passing day [1]. The increase in morbidity and mortality in NAFLD patients is not just due to liver diseases, but also to CVD [2–4].

The relationship between NAFLD and CVD is bidirectional [6]. Considering the close relationship of NAFLD with metabolic syndrome components, it is expected that CVD risk will increase in those with NAFLD. However, it has also been suggested that NAFLD may be a cause of CVD independent of classical cardiovascular risk factors [7]. In addition to traditional cardiovascular risk factors like DM, hypertension, and dyslipidemia, there are several secondary risk factors like hyperuricemia, hypoadiponectinemia, proinflammatory cytokines, and endothelial dysfunction that are believed to be behind the rise in the frequency of CVD in

TABLE 3. Fibrosis scores and parameters

	FIB- 4 Mean±SD				NFS Mean±SD				APRI Mean±SD			
	High (n=22)	ID (n=40)	Low (n=67)	p	High (n=22)	ID (n=37)	Low (n=70)	p	ID (n=9)	Low (n=120)	p
API	1.1±0.1	0.7±0.1	0.4±0.2	**<0.001**[1]	1±0.1	0.7±0.2	0.4±0.2	**<0.001**[1]	0.9±0.3	0.6±0.3	**0.011**[2]
TG	393.6±69	242±64	120.1±31.8	**<0.001**[1]	380.5±82.8	226.8±87	137.4±56	**<0.001**[1]	334.7±150	194.8±105	**0.010**[2]
TC	222±35	211.6±36	194.8±40.7	**0.012**[1]	222.3±35	206.2±38	198.3±41	**0.041**[1]	209.2±34.9	204.3±40	0.721[3]
Non-HDL	187.4±36	167.8±34	143.2±41.5	**<0.001**[1]	187.5±35.6	162.6±36	147±42	**<0.001**[1]	168.7±33.3	157.6±42	0.222[3]
HDL-C	34.6±6	43.8±6.5	50.7±10.6	**<0.001**[1]	34.8±5.8	43.7±8	50.4±10	**<0.001**[1]	40.6±8.4	46.2±11	0.154[2]
Non-HDL/HDL	5.6±2	3.9±0.9	3±1	**<0.001**[1]	5.6±1.5	3.8±1	3.1±1	**<0.001**[1]	4.4±1.1	3.7±1.4	0.117[2]
TC/ HDL	6.6±2	6.3±9.2	4±1	**<0.001**[1]	6.6±1.5	6.4±10	4.1±1	**<0.001**[1]	5.3±1.2	5.1±6	0.164[2]
LDL	135.6±34	131.2±30	119.3±34.6	0.071[1]	136.1±33.7	126.3±32	122.3±34	0.247[1]	124.4±32.2	125.9±34	0.902[3]
LDL/ HDL	4±1	3±0.8	2.4±0.8	**<0.001**[1]	4±1.2	3±0.9	2.5±0.8	**<0.001**[1]	3.1±0.9	2.9±1	0.548[2]

[1]: Kruskal Wallis Test; [2]: Mann-Whitney U; [3]: Independent Samples T-Test. API: Atherogenic plasma index; TG: Triglyceride; TC: Total cholesterol; Non-HDL: Non-HDL cholesterol; HDL-C: HDL cholesterol; LDL: LDL cholesterol

FIGURE 1. There is a positive, high and statistically significant correlation between API and FIB-4 score.

API: Atherogenic plasma index.

FIGURE 2. There is a positive, high and statistically significant correlation between API and NFS.

FIGURE 3. There is a positive, moderate and statistically significant correlation between API and APRI score.

NAFLD [8]. It should not be forgotten that NAFLD is closely associated with cardiovascular mortality as well as liver-related mortality [9–11].

NAFLD's progression depends on the level of hepatic fibrosis.The level of hepatic fibrosis is a reliable determinant of the course of NAFLD and liver-related mortality [12, 13]. It is also thought that there may be a relationship between hepatic fibrosis and cardiovascular complications [28]. In this research, we examined the correlation between hepatic fibrosis level (NFS, FIB-4, APRI scores) and cardiovascular event risk in NAFLD. As a result, we found that the risk of cardiovascular events increased as the degree of hepatic fibrosis increased. Chen et al. [14] suggested in a study that there is an association between liver fibrosis and cardiovascular mortality in people with coronary artery disease. In another meta-analysis, it was reported that there was a correlation between NAFLD and subclinical atherosclerosis, including coronary calcification, arterial stiffness and endothelial dysfunction. In the research of Neri et al. [29], a positive relationship was determined between the stages of liver fibrosis and intima-media thickness. Possible mechanisms of this increased atherosclerosis during NAFLD may be related to the secretion of certain cytokines and molecules, including adropine, selenoprotein P, fibroblast growth factor 21, sex hormone-binding globulin, and retinol-binding protein 4 [14].

API is a well-known predictive and prognostic biomarker for atherosclerotic CVD [15, 16]. API has been shown to be a strong predictor in diseases that cause major cardiovascular death, such as coronary artery disease and acute coronary syndrome.Various studies have demonstrated that API has a high level of accuracy in forecasting the risk of various situations such as atherosclerosis, CAD, myocardial infarction [18, 19] and hyperuricemia [20].

API has greater sensitivity in reflecting the relationship between atherogenic and protective lipoproteins. In a study by Frohlich et al. [21] it was stated that the API value was inversely related to the diameter of LDL-C particles and showed the sdLDL particle size. Therefore, it is thought that API can be accepted as an economical and reliable indicator of CAD. As a result of numerous studies in recent years, it is suggested that the API is a strong predictor of CAD risk [30]. In this study, we used API as a marker of CVD risk.

Obesity and dyslipidemia are the main risk factors for the development of NAFLD [31]. The relationship between traditional lipid parameters such as TC, LDL-C, TG and CAD is known. In addition to these, TG/HDL-C, TC/HDL-C, LDL-C/HDL-C ratios are thought to be predictors for CAD. In a study of more than 12 million people, it was emphasized that there was a relationship between lipid indices, especially TC/HDL-C, and the prevalence of NAFLD [30, 31]. In a study conducted by Nobili et al. [32], a positive correlation was shown between NAFLD fibrosis scores and TG/HDL-C TC/HDL-C, LDL-C/HDL-C ratios in 118 children who underwent a biopsy. We also found a significant relationship between fibrosis scores and lipid parameters in this study.

According to NFS and FIB-4 scoring, we found that HF group had higher TC, TG, nonHDL-C, non HDL/HDL-C, LDL/HDL-C and lower HDL-C than LF group. In other words, we determined that dyslipidemia was higher in the group with higher fibrosis. According to APRI scoring, we found that the TG levels of the IDF group were higher than LF group.

CONCLUSIONS

NAFLD is an important disease with a rapidly increasing prevalence in the world day by day. Noninvasive fibrosis scores are widely used to determine liver damage. In this study, we found that there is a relationship between FIB-4, NFS, APRI scores, API and dyslipidemia in people with NAFLD. In other words, as the level of fibrosis increases, the risk of CVD also increases. We think that these fibrosis scores in NAFLD patients can be used to evaluate the risk of cardiovascular events as well as to show liver damage.

Ethics Committee Approval: The Istanbul University of Health Sciences Umraniye Training and Research Hospital Clinical Research Ethics Committee granted approval for this study (date: 24.11.2022, number: 2022/354).

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