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Research Article

The Relationship Between Non-alcoholic Fatty Liver Disease and Epicardial Adipose Tissue Thickness, Carotid Intima Media Thickness, and Aortic Propagation Velocity

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ABSTRACT

Objective: Cardiovascular disease is prevalent among individuals diagnosed with non-alcoholic fatty liver disease (NAFLD). Epicardial adipose tissue (EAT), carotid intima-media thickness (CIMT), and aortic propagation velocity (APV) are associated with atherosclerosis and cardiovascular disease (CVD). The aim of this study is to determine whether NAFLD is associated with EAT, CIMT, and APV.

Methods: Individuals aged ≥18 and <65 years who presented for routine follow-up were included. The study population comprised 75 healthy controls (40 females, 35 males) and 141 patients with NAFLD (68 females, 73 males). Epicardial adipose tissue thickness, carotid intima-media thickness, and aortic propagation velocity were evaluated using transthoracic echocardiography in patients who were diagnosed with hepatosteatosis by ultrasound during routine check-ups at the internal medicine clinic.

Results: NAFLD was positively associated with epicardial adipose tissue thickness and carotid intima–media thickness, and negatively associated with aortic propagation velocity. Patients with NAFLD had significantly higher EAT and CIMT values and lower APV values than controls. Logistic regression analysis identified body mass index (BMI) and EAT as independent predictors of hepatosteatosis. Each unit increase in BMI was associated with a 1.29-fold increase in the risk of hepatosteatosis, whereas each unit increase in EAT was associated with a 5.49-fold increase.

Conclusion: Given the established associations of CIMT, APV, and EAT with atherosclerosis and cardiovascular disease, assessment of patients with NAFLD using these noninvasive parameters may help identify subclinical cardiovascular risk.

Keywords: Non-alcoholic fatty liver disease, epicardial adipose tissue thickness, carotid intima-media thickness, aortic propagation velocity

INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) is characterized by excessive fat accumulation within hepatocytes, affecting more than 5% of liver cells in individuals without significant alcohol intake or secondary causes of hepatic steatosis such as steatogenic drugs, prior surgery, or other liver disorders.[1] There are insufficient noninvasive parameters demonstrating the association between NAFLD and the risk of atherosclerosis and cardiovascular disease.[2,3] The gold standard for the accurate diagnosis of NAFLD is the pathological evaluation of liver biopsy specimens; however, numerous biomarkers of NAFLD have been investigated to avoid such an invasive

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technique.[4,5] Because NAFLD is associated with the risk factors of metabolic syndrome, treatment should primarily focus on the management of hypertension, hyperlipidemia, insulin resistance, and diabetes mellitus.[6]

Epicardial adipose tissue thickness (EAT) and volume have been associated with visceral adiposity, coronary artery disease, subclinical atherosclerosis, insulin resistance, metabolic syndrome, and cardiac structural and functional alterations.[7] EAT thickness is associated with the presence and progression of NAFLD. Individuals with increased EAT may require closer monitoring with regard to NAFLD.[8]

Atherosclerosis is a systemic and chronic disease, characterized by the transendothelial infiltration of low-density lipoprotein cholesterol (LDL-C), its accumulation within the intima, and subsequent oxidative and enzymatic processes.[9] Therefore, the intima–media complex of the arterial wall plays a crucial role in the pathogenesis of atherosclerosis and may reflect different stages of disease development. In the early stages of atherosclerosis, a hypertensive and hypertrophic response of medial cells can be observed, which is assessed by measuring carotid intima–media thickness (CIMT).[10]

Arterial stiffness and atherosclerotic changes within the aorta contribute to vascular wall thickening and reduced elasticity, ultimately increasing vascular resistance. As resistance increases, blood flow propagation within the arterial lumen decreases.[11] In this context, APV has emerged as a practical, reproducible, and non-invasive parameter for assessing arterial stiffness.[12]

The present study aimed to investigate the relationship between NAFLD and atherogenic parameters, including epicardial adipose tissue thickness, CIMT, and APV and to determine which of these parameters is the most valuable predictor of NAFLD.

MATERIAL AND METHODS

This descriptive study included individuals aged 18-64 years who presented to the Internal Medicine outpatient clinic of Erzincan Binali Yıldırım University Mengücek Gazi Training and Research Hospital for routine follow-up between October 25, 2022, and April 25, 2023. A total of 75 healthy individuals (40 females, 35 males) and 141 individuals with NAFLD (68 females, 73 males) were enrolled in the study. Among participants whose stage of hepatic steatosis was determined by ultrasonography, anthropometric measurements and hematological and biochemical tests (including liver and renal function tests, lipid profile, etc.) were evaluated.

Venous blood samples were collected following a fasting period of 10-12 hours.

After excluding, by ultrasonography, liver pathologies that could affect the study outcomes (such as malignancy, cholestasis, and liver cirrhosis), hepatic steatosis was classified into four stages: normal liver, stage 1 (mild),stage 2 (moderate), and stage 3 (severe).

Epicardial adipose tissue thickness measurements were performed by a cardiology specialist using transthoracic echocardiography (Philips EPIQ 7C) with an S4-2 transducer (2-4 MHz).

For CIMT evaluation, participants were examined in the supine position with slight neck extension, achieved by placing a pillow beneath the neck. Bilateral common carotid arteries were visualized using a Philips EPIQ 7C ultrasonography device with a 7.5 MHz linear probe. Three separate measurements were obtained from each side, and the average value was recorded.

For the assessment of APV, participants were positioned supine, and M-mode recordings were obtained by a cardiology specialist using a Philips EPIQ 7C echocardiography system with a 3.0 MHz transducer. The mean of at least three measurements was recorded as the APV value.

Statistical Analysis

Statistical analyses were performed using the Statistical Package for the Social Sciences (SPSS) version 22.0 (IBM Corp., Armonk, NY, USA). The normality of data distribution was assessed using the Kolmogorov-Smirnov test. Descriptive statistics are presented as mean (standard deviation) in the results and in the tables. Comparisons between two groups were performed using the Student's t-test for normally distributed variables and the Mann-Whitney U test for non-normally distributed variables. For comparisons among more than two groups, one-way analysis of variance was used for normally distributed data, while the Kruskal–Wallis test was applied for non-normally distributed data. Categorical variables were compared using the chi-square test.

Logistic regression analysis was conducted to identify factors affecting the risk of hepatic steatosis, including variables found to be significant in univariate analysis. Age and sex were included in the model as biological variables using the enter method; subsequently, significant variables were identified using the forward likelihood ratio method. Results were expressed as odds ratios with 95% confidence intervals.

A P value of <0.05 was considered statistically significant.

RESULTS

Among the 216 individuals included in the study, NAFLD was detected in 141 (65.3%); the remaining 75 individuals (34.7%) comprised the control group.

The study population consisted of 108 women (50%) and 108 men (50%). NAFLD was identified in 68 women (63.0%) and 73 men (67.6%). No statistically significant difference in sex distribution was observed between individuals with and without NAFLD (P = 0.47).

The mean epicardial adipose tissue thickness was 3.9 ± 0.8 mm in individuals without NAFLD and 5.1 ± 0.7 mm in those with NAFLD, demonstrating a statistically significant difference between the groups (P <0.001). The mean carotid intima–media thickness (CIMT) was 0.82 ± 0.12 mm in participants without NAFLD and 1.01 ± 0.12 mm in those with NAFLD (P < 0.001). The mean APV was 56.4 ± 7.9 cm/s in individuals without NAFLD and 43.8 ± 7.9 cm/s in those with NAFLD, with the difference being statistically significant (P < 0.001).

Comparison of laboratory parameters between individuals with and without NAFLD revealed statistically significant differences in the levels of glucose, total cholesterol, high-density lipoprotein cholesterol (HDL-C), LDL-C, triglycerides, alanine aminotransferase (ALT), aspartate aminotransferase (AST), gamma-glutamyl transferase (GGT), and body mass index (BMI) (Table 1).

When demographic, clinical, and laboratory characteristics were compared across levels of hepatic steatosis severity, significant differences were observed in BMI, EAT, CIMT, APV, triglycerides, fasting plasma glucose, ALT, and AST (Table 2).

To identify factors associated with hepatic fat accumulation, logistic regression analysis was performed using variables that were significant in the univariate analysis; age and sex were retained in the model as biological covariates. According to the regression analysis, BMI and EAT were found to be significant independent predictors of NAFLD. Each unit increase in BMI was associated with a 1.29-fold increase in the risk of NAFLD, whereas each unit increase in EAT was associated with a 5.49-fold increase in the risk of NAFLD (Table 3).

DISCUSSION

In this study, laboratory parameters, radiological imaging findings, and anthropometric measurements possibly associated with nonalcoholic fatty liver disease (NAFLD) were evaluated.

When individuals with and without NAFLD were compared, statistically significant associations were observed between NAFLD status and body mass index (BMI), epicardial adipose tissue thickness (EAT), carotid intima–media thickness (CIMT), APV, total cholesterol, high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), triglycerides, fasting plasma glucose, ALT, AST, and GGT.

When data were analyzed according to the degree of hepatosteatosis, statistically significant relationships were found between steatosis severity and BMI, APV, EAT, CIMT, triglycerides, fasting plasma glucose, AST, and ALT.

MAIN POINTS

- Patients with Non-alcoholic fatty liver disease (NAFLD) have significantly increased epicardial adipose tissue (EAT) thickness and carotid intima-media thickness (CIMT), and reduced aortic propagation velocity (APV).
- The severity of hepatic steatosis is associated with progressive increases in EAT and CIMT and a progressive decrease in APV.
- Noninvasive cardiovascular imaging markers, particularly EAT, may facilitate early identification of subclinical cardiovascular risk in patients with NAFLD.
- Routine assessment of EAT together with CIMT and APV may improve cardiovascular risk stratification and support earlier preventive interventions in patients with NAFLD.

Several mechanisms have been proposed to explain the link among NAFLD, various stages of the atherosclerotic process, and alterations in cardiac structure and function. These mechanisms include endothelial dysfunction, atherogenic dyslipidemia, insulin resistance, and impaired cardiac mechanics.[13,14]

An association between obesity and NAFLD has been well established.[15] In a study by Almeida et al. involving 3,841 participants, the prevalence of NAFLD was shown to increase

Table 1. Demographic, Clinical, and Laboratory Characteristics of the Groups

		Control group (n=75)	Study group (n=141)	*P* value
Sex	Female	40 (53.3)	68 (48.2)	0.47
	Male	35 (46.7)	73 (51.8)	
Age (year)*		42.8 (13.2)	46 (11)	0.11
Body mass index (kg/m^2)*		25.9 (3.9)	31.7 (5.4)	**<0.001**
Waist circumference (cm)**		91.3 (12.4)	106 (12.7)	**<0.001**
Hip circumference (cm)*		103.5 (9.7)	113.5 (11.8)	**<0.001**
NAFLD Fibrosis Score**		-2.42 (1.04)	-1.83 (1.38)	**0.001**
Epicardial adipose tissue thickness (mm)*		3.9 (0.8)	5.1 (0.7)	**<0.001**
Carotid intima media thickness (mm)*		0.82 (0.12)	1.01 (0.12)	**<0.001**
Aortic propagation velocity (cm/sn)*		56.4 (7.9)	43.8 (7.9)	**<0.001**
Total Cholesterol (mg/dL)**		189.1(40.9)	203.6 (38.6)	**0.011**
HDL Cholesterol (mg/dL)**		48.4 (10.3)	45.2 (8.8)	**0.019**
LDL Cholesterol (mg/dL)**		126.9 (33.1)	140.1 (29.9)	**0.003**
Triglycerides (mg/dL)*		127.2 (66.3)	188.7 (106.5)	**<0.001**
Fasting Plasma Glucose (mg/dL)*		90.3 (9.7)	110.7 (42.4)	**<0.001**
ALT (U/L)*		21.7 (15)	35.8 (24.6)	**<0.001**
AST (U/L)*		22.7 (9.2)	27.6 (12.4)	**<0.001**
GGT (U/L)*		23.8 (17.1)	37.5 (22.6)	**<0.001**
Creatinine (mg/dL)**		0.85 (0.15)	0.85 (0.14)	0.94
Albumin (g/dL)**		43.7 (2.5)	43.2 (2.7)	0.26
TSH (µU/mL)*		1.84 (1.04)	1.89 (1.06)	0.62
Uric Acid (mg/dL)**		4.5 (1.2)	5.1 (1.4)	**0.001**
Platelet count (10^3/µL)**		244.6 (51.6)	250.6 (53.7)	0.43

*Mann-Whitney U test was applied. ** Student's t-test was applied. *** Chi-square test was applied. (The data in the table are presented as mean (standard deviation) values.)
NAFLD, non-alcoholic fatty liver disease; HDL, high-density lipoprotein; LDL, low-density lipoprotein; ALT, alanine aminotransferase; AST, aspartate aminotransferase; GGT, gamma-glutamyl transferase; TSH, thyroid-stimulating hormone.

with rising BMI, indicating that hepatic steatosis is primarily related to excess adiposity (higher BMI).[16] In a meta-analysis conducted by Yuan et al.[17], an increase in BMI was associated with a 1.33-fold increase in the risk of NAFLD. In the present study, the mean BMI was 25.9 ± 3.9 kg/m^2 in individuals without NAFLD and 31.7 ± 5.4 kg/m^2 in those with NAFLD. Moreover, the severity of NAFLD increased in parallel with BMI. Logistic regression analysis demonstrated that BMI was associated with a 1.29-fold increased risk of NAFLD. These findings are consistent with previous reports.

Epicardial adipose tissue thickness is an easily measurable marker of visceral adiposity. Epicardial adipocytes secrete

Table 2. Comparison of Demographic, Clinical, and Laboratory Data of Groups According to the Degree of Hepatosteatosis

		Grade 1	Grade 2	Grade 3	*P*
Age (year)*		44.5(11.1)	47.1(10.6)	46.7(11.4)	0.43
Sex	Female	26 (50.9)	27 (50)	15 (44.1)	0.85
	Male	27 (49.1)	27 (50)	19 (55.9)	
BMI (kg/m^2) **		29.9 (4.3)^a	31.5 (5.9)^a	34.7 (4.7)^b	**<0.001**
Waist circumference (cm)*		101.8 (12.2)^a	105.2 (11.1)^a	113.8 (12.4)^b	**<0.001**
Hip circumference (cm)*		109.6 (10.7)^a	113.2 (10.8)^a	120.1 (12.4)^b	**<0.001**
NAFLD Fibrosis Score*		-2.25 (1.21)^a	-1.77 (1.31)	-1.26 (1.57)^b	**0.004**
Epicardial adipose tissue thickness (mm)**		4.7(0.5) ^{a}	5.2 (0.75)^b	5.7 (0.6)^c	**<0.001**
Carotid intima media thickness (mm)**		0.94 (0.09)^a	1.02 (0.13)^b	1.09 (0.1)^c	**<0.001**
Aortic propagation velocity (cm^2 .dyn^{-1}X10^{-6})**		49 (5.3)^a	42.6 (7.8)^b	37.5(5.7)^c	**<0.001**
Left ventricular ejection fraction (%)**		56.2 (4.4)	56.6 (4.2)	57.1 (4.3)	0.65
Total Cholesterol (mg/dL)*		199.8 (34.9)	206.9 (38.4)	204.2 (44.6)	0.63
HDL Cholesterol (mg/dL)*		44.1 (8.7)	46.7 (8.9)	44.5 (8.4)	0.27
LDL Cholesterol*		138.2 (28.6)	141.2 (29.9)	141.2 (32.6)	0.85
Triglycerides (mg/dL)**		160.3 (101.1)^a	196.9 (106)	219.7 (107.3)^b	**0.005**
Fasting Plasma Glucose (mg/dL)**		96.7 (19.4)^a	115 (49.5)	125.8 (50.4)^b	**0.009**
ALT (U/L) **		26.5 (15.3)^a	38.2 (24.3)^b	46.7 (31.3)^b	**<0.001**
AST (U/L)*		22.6 (6.6)^a	28.5 (12.7)^b	33.8 (15.7)^b	**<0.001**
GGT (U/L) **		32.4 (18.1)	37.2 (19.9)	45.6 (30.1)	0.08
Kreatinin (mg/dL) *		0.84 (0.14)	0.85 (0.14)	0.86 (0.14)	0.77
Albümin (g/dL) *		43 (2.7)	43.8 (2.8)	42.7 (2.7)	0.12
TSH (µU/mL) *		1.98 (1.1)	2 (1.07)	1.58 (0.92)	0.14
Uric Acid (mg/dL)**		4.8 (1.2)^a	5.1 (1.4)	5.7 (1.3)^b	**0.011**
Platelet count (10^3/µL)*		257.7 (49.7)	246 (55)	246.8 (57.8)	0.48

*One Way ANOVA test was used. **Kruskal Wallis test was used. *** Chi-square test was applied. a,b,c: Groups marked with the same letter are statistically similar, and there is a statistically significant difference at the level of <0.05 between groups with different letters.
BMI, body mass index; NAFLD, non-alcoholic fatty liver disease; HDL, high-density lipoprotein; LDL, low-density lipoprotein; ALT, alanine aminotransferase; AST, aspartate aminotransferase; gamma-glutamyl transferase; TSH, thyroid-stimulating hormone.

Table 3. The Results of the Logistic Regression Analysis Conducted to Determine the risk Factors Affecting Liver Steatosis

	P value	OR	95% CI for OR	
			Lower limit	Upper limit
Gender*	0.96	0.978	0.439	2.175
Age*	0.62	0.966	0.932	1.002
BMI	**<0.001**	1.294	1.152	1.453
EAT	**<0.001**	5.492	3.121	9.664
APV	0.37	1.101	0.893	1.357
CIMT	0.98	1.011	0.374	2.732

*Gender and age were kept constant in the model as biological factors. OR: Odds-ratio; CI: Confidence interval; BMI, body mass index; EAT, epicardial adipose tissue thickness; APV, aortic propagation velocity; CIMT; carotid intima-media thickness.

numerous cytokines and vasoactive peptides, all of which may independently contribute to increased cardiovascular risk.[18] In a study by Oğuz et al.[19] including 78 participants, mean EAT was 5.1 ± 2.5 mm in individuals with NAFLD, compared with 2.9 ± 0.9 mm in healthy controls. In a large multivariate logistic regression analysis conducted by Meng et al.[2] in 2,238 individuals, a significant association between NAFLD and EAT was demonstrated. Participants with higher EAT were older and exhibited greater waist and hip circumferences, lower HDL-C levels, higher body weight and BMI, elevated blood pressure, higher LDL-C, fasting plasma glucose, triglyceride levels, increased CIMT, and poorer overall cardiovascular health.[2] In the present study, the mean EAT was 3.9 ± 0.8 mm in individuals without NAFLD and 5.1 ± 0.7 mm in those with NAFLD, and EAT increased in parallel with the severity of steatosis. Regression analysis revealed that EAT increased the risk of NAFLD by 5.49-fold. EAT was identified as the parameter most strongly associated with NAFLD and therefore may serve as a valuable imaging marker to predict hepatic steatosis.

CIMT is a widely used and reliable method for the assessment of subclinical atherosclerosis.[20] Increased CIMT has been shown to be associated with established atherosclerotic risk factors, including age, sex, BMI, systolic blood pressure, HbA1c, and hyperlipidemia.[21] In a study by Kumari et al.[22] involving 80 participants, CIMT was 0.52 mm in individuals without NAFLD and 0.86 mm in those with NAFLD, and a statistically significant relationship between NAFLD and CIMT was reported. Furthermore, a meta-analysis by Sookoian et al. including 3,497 participants demonstrated a 13% increase in CIMT among individuals with NAFLD compared with those without.[23] In the present study, the mean CIMT was 0.82 ± 0.12 mm in individuals without NAFLD and 1.01 ± 0.12 mm in those with NAFLD. This difference was statistically significant, and CIMT values also differed significantly across steatosis severity groups. However, CIMT was not identified as an independent predictor of NAFLD in the regression analysis.

APV, a parameter reflecting aortic stiffness and arterial resistance, has been shown to be associated with coronary artery disease.[24] In the study by Oğuz et al.,[19] patients with NAFLD exhibited significantly lower APV values and significantly higher epicardial adipose tissue thickness compared with controls.[19] Similarly, Gunes et al.[11] demonstrated that APV, a simple, practical, and non-invasive measure of arterial resistance, was associated with coronary atherosclerosis and significantly lower in individuals with NAFLD than in controls. Additionally, APV was found to decrease in individuals with increased waist circumference, higher BMI, and elevated fasting plasma glucose levels.[11] In our study, the mean APV was 56.4 ± 7.9 cm/s in individuals without NAFLD and 43.7 ± 7.9 cm/s in those with NAFLD. Although APV differed significantly between individuals with and without NAFLD and across steatosis severity groups, it was not identified as an independent predictor of NAFLD in the regression analysis.

Study limitations

This study has several limitations. First, due to its single-center, descriptive design, establishing causal relationships based on the findings is challenging. Second, participants could not be followed longitudinally after the diagnosis of NAFLD; therefore, the duration required for the development of cardiovascular risk could not be determined.

CONCLUSION

Our findings demonstrate significant associations between NAFLD and atherogenic parameters, including EAT, CIMT, and APV. Among these, BMI and EAT were identified as independent predictors of NAFLD. Given the increased cardiovascular risk associated with NAFLD, incorporating noninvasive vascular assessments such as EAT, CIMT, and APV into routine evaluation may facilitate early detection of subclinical cardiovascular disease.

Further multicenter studies with larger cohorts are required to validate these findings and clarify the clinical utility of these parameters.

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Ethics

Ethics Committee Approval: The study was approved by the Erzincan Binali Yıldırım University Clinical Research Ethics Committee (approval no.: 02/08, dated: 29.09.2022).

Informed Consent: Written informed consent was obtained from all participats.

Footnotes

Author Contributions

Concept Design – E.Ö., F.Ö.; Data Collection or Processing – E.Ö., M.S.C., S.A., G.K.Ö.; Analysis or Interpretation – E.Ö., F.Ö., S.H., G.K.Ö.; Literature Review - E.Ö., F.Ö., S.A.; Writing, Reviewing and Editing – E.Ö., F.Ö.

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