

Association Between Fatty Liver Index and Epicardial Fat Thickness in Young Adults

Genç Yetişkinlerde Yağlı Karaciğer İndeksi ile Epikardiyal Yağ Kalınlığı Arasındaki İlişki

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Abstract

Objective: Hepatic steatosis refers to the fatty degeneration of liver tissue, which is associated with an increased risk of cardiovascular morbidity and mortality. Patients diagnosed with hepatic steatosis have been shown to have greater epicardial adipose tissue thickness. An increase in epicardial fat thickness (EFT) correlates with heightened cardiovascular risks. The fatty liver index (FLI) serves as a non-invasive metric for evaluating hepatic steatosis. In our study, we sought to assess whether EFT could be predicted within a apparently healthy young adult population utilising FLI. Furthermore, if such a prediction proves feasible, it aims to facilitate early diagnosis of at-risk individuals and the implementation of preventive measures to decrease future cardiovascular morbidity and mortality by furnishing pertinent information to clinicians.

Method: We conducted an observational cross-sectional study involving 258 participants randomly selected from apparently healthy young adults aged 18 to 41. The cohort was divided into two groups based on EFT (<4 mm and ≥ 4 mm). We assessed participants' EFT values using echocardiographic examination. We measured the FLI utilising variables such as body mass index, waist circumference, serum gamma-glutamyl transferase, and triglyceride level.

Results: A multiple linear regression model was constructed utilising both stepwise and enter methods, incorporating variables that demonstrated significant correlation with EFT as well as the gender variable, which has been associated with EFT in previous studies. The model significantly predicted EFT [R=0.492, R²=0.242, adjusted R²=0.233, F (3, 250) =26.662, p<0.001], accounting for 23.3% of the variance. The standard error of the estimate was 11.041, and the Durbin-Watson statistic was 2.096. Among the predictors, FLI, age,

Öz

Amaç: Karaciğer steatozu, kardiyovasküler morbidite ve mortalite riskinin artmasıyla ilişkili olan karaciğer dokusunun yağlı dejenerasyonunu ifade eder. Karaciğer steatozu tanısı konulan hastalarda epikardiyal yağ dokusu kalınlığının daha fazla olduğu gösterilmiştir. Epikardiyal yağ kalınlığındaki (EFT) artış, artan kardiyovasküler risklerle ilişkilidir. Yağlı karaciğer indeksi (FLI), hepatik steatozu değerlendirmek için kullanılan non-invaziv bir ölçüt olarak hizmet eder. Çalışmamızda, FLI'yi kullanarak görünüşte sağlıklı genç yetişkin popülasyonunda EFT'nin öngörülebilir olup olmadığını değerlendirmeyi amaçladık. Ayrıca, böyle bir öngörünün mümkün olduğu kanıtlanırsa, klinisyenlere ilgili bilgileri sunarak risk altındaki bireylerin erken teşhisini ve gelecekteki kardiyovasküler morbidite ve mortaliteyi azaltmak için önleyici tedbirlerin uygulanmasını kolaylaştırmayı hedeflemektedir.

Yöntem: On sekiz ila 41 yaşları arasında görünüşte sağlıklı genç yetişkinlerden rastgele seçilen 258 katılımcıyı içeren gözlemsel bir kesitsel çalışma yürüttük. Kohort, EFT'ye göre (<4 mm ve ≥ 4 mm) iki gruba ayrıldı. Ekokardiyografik muayene kullanarak katılımcıların EFT değerlerini değerlendirdik. Vücut kitle indeksi, bel çevresi, serum gama-glutamyl transferaz ve trigliserid düzeyi gibi değişkenleri kullanarak FLI'yi ölçtük.

Bulgular: Hem stepwise hem de enter yöntemleri kullanılarak, EFT ile anlamlı korelasyon gösteren değişkenlerin yanı sıra önceki çalışmalarda EFT ile ilişkilendirilen cinsiyet değişkenini de içeren bir çoklu doğrusal regresyon modeli oluşturuldu. Model, EFT'yi önemli ölçüde tahmin etti [R=0,492, R²=0,242, düzeltilmiş R²=0,233, F (3, 250) =26,662, p<0,001] ve varyansın %23,3'ünü açıkladı. Tahminin standart hatası 11,041 ve Durbin-Watson istatistiği 2,096 idi.

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Abstract

and platelet count were all significant independent predictors of EFT.

Conclusion: As a result, we found that EFT increased with FLI. We assert that forthcoming large-scale multicentre research involving healthy young adults will confirm that the FLI can help healthcare professionals identify at-risk individuals early and implement measures to reduce future cardiovascular morbidity and mortality.

Keywords: Cardiometabolic risk, epicardial fat thickness, fatty liver index, young adults

Öz

Yordayıcılar arasında FLI, yaş ve trombosit sayısı, EFT'nin tümüyle anlamlı bağımsız yordayıcılarıydı.

Sonuç: Sonuç olarak, EFT'nin FLI ile birlikte arttığını bulduk. Sağlıklı genç yetişkinleri içeren gelecekteki büyük ölçekli çok merkezli araştırmaların, FLI'nın sağlık profesyonellerinin risk altındaki bireyleri erken teşhis etmelerine ve gelecekteki kardiyovasküler morbidite ve mortaliteyi azaltmak için önlemler almalarına yardımcı olabileceğini doğrulayacağını iddia ediyoruz.

Anahtar kelimeler: Epikardiyal yağ kalınlığı, genç yetişkinler, yağlı karaciğer indeksi, kardiyometabolik risk

Introduction

Hepatic steatosis is characterized by the accumulation of triglycerides in hepatocytes above normal levels. It is generally defined as fat accounting for at least 5% of liver weight (1).

The fatty liver index (FLI) is a widely used, simple, and non-invasive score developed to assess the risk of hepatic steatosis. The FLI is calculated by an algorithm based on body mass index (BMI), waist circumference, triglyceride level, and gamma-glutamyl transferase values (2-4). High FLI is associated with cardiometabolic risk factors, including insulin resistance, abdominal and cardiac adiposity, hypertension, and hyperlipidemia, and is also independently associated with all-cause mortality and cardiovascular disease risk (5-8).

Epicardial fat is a visceral adipose tissue surrounding the heart's outer surface and lying between the myocardium and the pericardium. It is metabolically active and secretes proinflammatory substances that can directly affect the heart and blood vessels (9). The functional complexity of epicardial fat thickness (EFT) is unclear. However, it has been shown to affect cardiac morphology and function, preserving heart contractility and repolarization under physiological conditions (10). Increased EFT is associated with greater visceral adiposity and a higher risk of metabolic syndrome.

EFT can be easily measured using transthoracic echocardiography (11,12). EFT is also associated with the severity of liver steatosis and fibrosis (13). In various studies, EFT above 4 mm is considered thick in healthy individuals (14,15).

Studies have indicated that as EFT increases, the risk of non-alcoholic liver disease also rises, and there is a significant and independent association between FLI and EFT within

the general population (16,17). There are no studies in the current literature focusing on healthy pediatric or young adult populations. Our study is the first to examine this age group. According to our hypothesis, EFT will increase with increasing FLI in the apparently healthy young adult population.

In our study, we aim to determine whether FLI, a non-invasive technique, can predict EFT, recognized as an independent predictor of cardiovascular morbidity and mortality, in an apparently healthy young adult population.

Should our findings indicate that such a prediction is feasible, we aim to furnish clinicians with pertinent information, thereby facilitating early identification of at-risk individuals and the implementation of measures to mitigate future cardiovascular morbidity and mortality.

Materials and Methods

Study Population

We conducted an observational, cross-sectional study that included 258 apparently healthy young adults between the ages of 18 and 41 who presented to Battalgazi State Hospital's Cardiology Clinic with complaints such as chest pain, shortness of breath, or palpitations, and who were considered healthy after physical examination, laboratory tests, and clinical evaluation.

A total of 258 participants were initially recruited for the study. However, 3 participants were excluded from the final analysis due to missing EFT scores. Consequently, statistical evaluations were conducted with the remaining 255 valid cases.

The cohort was divided into two groups based on EFT (<4 mm and ≥4 mm).

In our study, we included patients aged 18-41 years who had no history of chronic disease, were not using medications, and had no active infection, obesity, or cachexia.

We excluded patients who were taking active medication, who had a history of acute and/or chronic disease, were obese, or who were over 41 years of age. We excluded participants with laboratory measurements suggesting acute infection.

The research was conducted in accordance with the Declaration of Helsinki and was approved by the Local Ethics Committee of İnönü University, as documented in decision number 2025/8194 (protocol no: 2025/8194; date: 21-08-2025). Written and signed consent was obtained from each participant.

Laboratory Measurements

We assessed participants' EFT values by echocardiography using a 3.2-MHz transducer on a Philips Affinity 50 ultrasound system (Philips, Andover, MA, USA). We measured EFT in the two-dimensional parasternal long-axis view by positioning the M-mode cursor perpendicular to the aortic annulus, along the free wall of the right ventricle, at end-diastole. We measured the FLI using BMI, waist circumference, serum gamma-glutamyl transferase, and triglyceride level (2). We calculated the BMI as weight in kilograms divided by the square of the height in meters, expressed as kg/m^2 . Using a flexible, non-metallic tape measure, we measured waist circumference at the level of the navel and immediately above the iliac crest after a normal exhalation. After an 8 to 10-hour fast, we collected blood samples from the right or left antecubital vein for analysis. We obtained the history of coronavirus disease-2019 (COVID-19) infection from medical records.

Statistical Analysis

Data were analysed using SPSS software (IBM Corp., IBM SPSS Statistics for Mac, version 27.0; Armonk, NY: IBM Corp., 2020). By analysing the data in their recorded state, we have avoided any potential errors that could have been introduced by imputing missing values. A descriptive analysis was conducted to characterize the study population. The normality of the variables was tested using the Kolmogorov-Smirnov test. For pairwise comparisons, the independent-samples t-test was used for normally distributed variables and the Mann-Whitney U test for non-normally distributed variables. In this study, we considered results to be statistically significant at $p < 0.05$.

Furthermore, 95% confidence intervals for differences that did not include zero were deemed statistically significant. The categorical variables were analysed using the appropriate chi-square test and were expressed as percentages and absolute numbers. Means and standard deviations were used to express continuous variables for normally distributed data, while medians and interquartile ranges were used for non-normally distributed data. The correlation between continuous variables was evaluated using Pearson's correlation test for normally distributed variables and Spearman's correlation test for non-normally distributed variables. Additionally, multiple linear regression analysis was conducted to forecast the EFT. We assessed the study's effect size using Cohen's *d*. A receiver operating characteristic (ROC) analysis evaluated the FLI's efficacy in predicting significant EFT.

Results

The study included 258 participants with a mean age of 29.31 ± 6.86 years, of whom 39.50% were male (Table 1). When participants were stratified by EFT (EFT < 4 mm vs. EFT ≥ 4 mm), the higher EFT group demonstrated significantly greater age (32.07 ± 6.39 vs. 27.31 ± 6.51 years, $p < 0.001$), BMI (26.64 ± 4.25 vs. 23.69 ± 4.18 kg/m^2 , $p < 0.001$), and FLI (43.92 ± 29.83 vs. 22.59 ± 23.33 , $p < 0.001$) (Table 2).

Laboratory analysis showed significantly higher C-reactive protein (CRP) levels [0.20 (0.10 - 0.40) vs. 0.20 (0.10 - 0.30) mg/dL , $p = 0.009$] (although median values were similar, the distribution differed significantly), triglycerides [102 (72 - 151) vs. 81.50 (63.50 - 116.50) mg/dL , $p = 0.001$], and low-density lipoprotein (LDL) (112.97 ± 28.28 vs. 99.63 ± 28.88 mg/dL , $p < 0.001$) in the higher EFT group.

No statistically significant differences were found between the groups regarding history of COVID-19 infection, haemoglobin, platelet count, total leukocyte count, monocyte count, basophil count, platelet distribution width, red cell distribution width, or thyroid-stimulating hormone levels ($p > 0.05$ for all).

Statistically significant positive correlations between EFT and the variables were found using Pearson or Spearman correlation analysis. The strongest positive correlations were observed with age ($r = 0.376$, $p < 0.001$) and FLI ($r = 0.413$, $p < 0.001$) (Figure 1). Additionally, statistically significant correlations were identified between EFT and LDL ($r = 0.255$, $p < 0.001$), platelet count ($r = 0.140$, $p = 0.026$), CRP ($\rho = 0.180$, $p = 0.004$), and eosinophils ($\rho = 0.136$, $p = 0.030$). A weak

positive statistical relationship was observed between the EFT and the FLI (Figure 2).

We constructed a multiple linear regression model using stepwise and enter methods, including variables that showed significant correlations with EFT, together with the gender variable, which has been associated with EFT in previous studies. The model significantly predicted EFT [R=0.492, R²=0.242, adjusted R²=0.233, F (3, 250)=26.662, p<0.001], accounting for 23.3% of the variance. The standard error of the estimate was 11.041, and the Durbin-Watson statistic was 2.096. Among the predictors, age, FLI, and platelet count were all significant independent predictors of EFT (Table 3).

Cohen's d was used to assess the magnitude of differences between groups. The study revealed moderate-to-large effect sizes for age, BMI, and FLI, with d =0.74, 0.70, and 0.81, respectively.

Table 1. Demographic and laboratory data of participants

Characteristics (258 participants)	n (%)/mean (SD) or median (IQR)
Age (years) (SD)	29.31 (6.86)
Male n (%)	102 (39.51)
Smoking n (%)	98 (38)
COVID-19 (%)	92 (35.71)
BMI kg/m ² (SD)	24.91 (4.44)
Fatty liver index (SD)	31.75 (28.38)
Haemoglobin g/dL (SD)	13.89 (1.81)
Platelet 10 ³ /mL (SD)	276.78 (61.74)
White blood cell 10 ³ /mL (SD)	7.31 (1.69)
Eosinophil 10 ³ /mL (IQR)	0.13 (0.08-0.22)
Monocyte 10 ³ /mL (SD)	0.57 (0.18)
Basophil 10 ³ /mL (IQR)	0.03 (0.02-0.04)
PDW % (SD)	12.01 (2.04)
RDW % (IQR)	12.75 (12.20-13.40)
TSH mU/L (IQR)	1.68 (1.24-2.21)
CRP mg/dL (IQR)	0.20 (0.10-0.40)
Triglyceride mg/dL (IQR)	90 (67-133.75)
LDL mg/dL (SD)	105.21 (29.58)
Epicardial fat thickness mm (SD)	4.15 (1.26)

The categorical variables were expressed as percentages (%) and absolute numbers. Means and standard deviations were used to express continuous variables for normally distributed data, while medians and interquartile ranges were used for non-normally distributed data. BMI: Body mass index, PDW: Platelet distribution width, RDW: Red-cell distribution width, TSH: Thyroid-stimulating hormone, CRP: C-reactive protein, LDL: Low-density lipoprotein, SD: Standard deviation, COVID-19: Coronavirus disease-2019, GFR: Glomerular filtration rate, IQR: Interquartile range. The demographic data is based on 258 individuals, whilst the epicardial fat thickness analyses are based on 255 individuals due to data loss

ROC analysis assessed the FLI's ability to predict substantial EFT. The results demonstrated a statistically significant area under the curve [(AUC) =0.727; standard error =0.032; 95% confidence interval (0.665, 0.789); p<0.001], indicating moderate discriminative ability (Figure 3). The analysis revealed that an FLI threshold of 14 yielded the highest Youden index (0.498), providing moderate discriminative performance, with sensitivity of 79.9% and specificity of 69.9% (18).

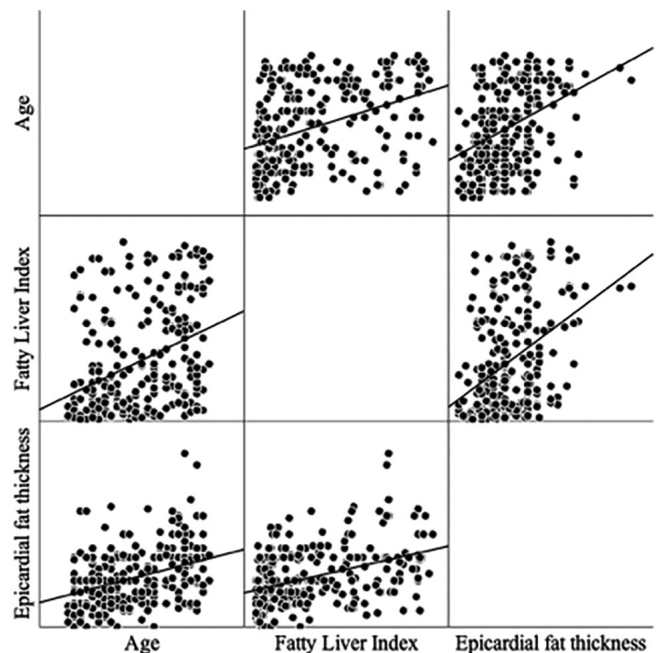


Figure 1. Scatterplot matrix showing the relationships between epicardial fat thickness, age, and fatty liver index

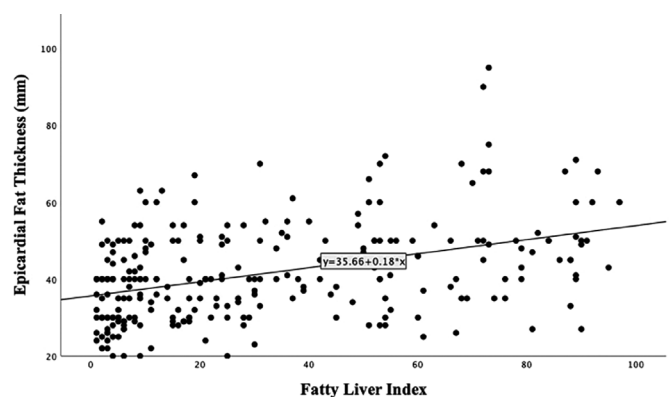


Figure 2. Correlation between epicardial fat thickness and fatty liver index

Table 2. Comparison of demographic and clinical data of both groups

Characteristics	EFT <4 mm (n=146)	EFT ≥4 mm (n=109)	p
Age (years) (SD)	27.31 (6.51)	32.07 (6.39)	<0.001**
Male n (%)	50 (50.51)	49 (49.51)	0.083
Smoking n (%)	34 (23.29)	62 (56.88)	0.069
COVID-19 (%)	49 (53.81)	42 (46.21)	0.412
BMI kg/m ² (SD)	23.69 (4.18)	26.64 (4.25)	<0.001**
Fatty liver index (SD)	22.59 (23.33)	43.92 (29.83)	<0.001**
Haemoglobin g/dL (SD)	13.73 (1.81)	14.04 (1.77)	0.127
Platelet 10 ³ /mL (SD)	271 (55.11)	284.31 (70.21)	0.102
White blood cell 10 ³ /mL (SD)	7.27 (1.71)	7.41 (1.71)	0.543
Eosinophil 10 ³ /mL (IQR)	0.13 (0.08-0.21)	0.14 (0.09-0.22)	0.140
Monocyte 10 ³ /mL (SD)	0.56 (0.18)	0.59 (0.19)	0.148
Basophil 10 ³ /mL (IQR)	0.03 (0.02-0.04)	0.03 (0.02-0.04)	0.227
PDW % (IQR)	12.05 (10.75-13.60)	11.70 (10.35-13.20)	0.078
RDW % (IQR)	12.75 (12.20-13.50)	12.80 (12.20-13.25)	0.604
TSH mU/L (IQR)	1.70 (1.21-2.34)	1.57 (1.21-2.12)	0.549
CRP mg/dL (IQR)	0.20 (0.10-0.30)	0.20 (0.10-0.40)	0.009*
Triglyceride mg/dL (IQR)	81.50 (63.50-116.50)	102 (72-151)	0.001**
LDL mg/dL (SD)	99.63 (28.88)	112.97 (28.28)	<0.001**

The categorical variables were analysed using the appropriate chi-square test and expressed as percentages (%) and absolute numbers. Means and standard deviations were used to express continuous variables for normally distributed data, while medians and interquartile ranges were used for non-normally distributed data. BMI: Body mass index, PDW: Platelet distribution width, RDW: Red-cell distribution width, TSH: Thyroid-stimulating hormone, SD: Standard deviation, COVID-19: Coronavirus disease-2019, CRP: C-reactive protein, LDL: Low-density lipoprotein, IQR: Interquartile range, *: p<0.05, **: p<0.001. The demographic data is based on 258 individuals, whilst the EFT analyses are based on 255 individuals due to data loss

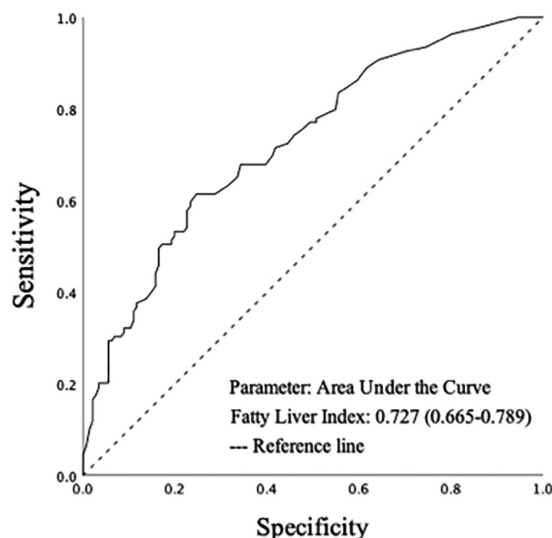


Figure 3. Receiver operating characteristic curve showing the fatty liver index's ability to discriminate between individuals with and without high epicardial fat thickness

Discussion

Our study is the first to examine the relationship between FLI and EFT in young, apparently healthy adults. We determined that as the FLI increases, the EFT also increases. Our multiple regression analysis reveals that the FLI is a significant independent predictor of EFT.

Hepatic steatosis is characterized by excessive fat accumulation, particularly triglycerides, in hepatocytes (liver cells) and is often called fatty liver disease. Hepatosteatois is the first and most common stage of both alcohol-related and non-alcohol-related liver diseases (19,20). Hepatic steatosis often co-occurs with cardiovascular disease. It is associated with higher coronary artery calcium scores, greater plaque burden, and metabolic syndrome in patients (21,22). Hepatosteatois is associated with cardiovascular disease in the general population (23). FLI is a score developed to estimate hepatic steatosis non-invasively, based on BMI, waist circumference, triglycerides, and gamma-glutamyltransferase levels. FLI is widely used to screen for the risk of non-alcoholic fatty liver disease and to predict the presence of steatosis in population studies (24).

Table 3. Multiple linear regression analysis predicting epicardial fat thickness using the enter approach

	B	SE	β	t	p	95% CI	
						Lower	Upper
Age	0.48	0.11	0.26	4.37	<0.01	0.26	0.69
Fatty liver index	0.14	0.03	0.3	5.09	<0.01	0.08	0.19
Platelet count	0.03	0.01	0.13	2.4	0.02	0.01	0.05

The dependent variable is the epicardial fat tissue thickness. CI: Confidence interval, SE: Standard error

Epicardial adipose tissue (EAT) is visceral adipose tissue located around the heart and coronary arteries, between the myocardium and the pericardium. It shares the same microcirculation as the heart and is in close anatomical proximity to it. Under normal conditions, EAT performs protective functions such as cushioning the heart against mechanical stress, regulating myocardial temperature, and maintaining fatty acid balance. It also serves as an energy source and secretes various cytokines that nourish the heart muscle (25).

EFT is typically measured by echocardiography or CT and serves as an independent indicator of visceral obesity, metabolic syndrome, type 2 diabetes, hypertension, and increased cardiovascular disease risk (26). Reviews and meta-analyses show that EFT and cardiovascular risk increase in conditions such as fatty liver disease and metabolic syndrome (27,28).

Research suggests that an increase in hepatic steatosis is associated with a corresponding increase in EFT, thereby increasing the risk of cardiometabolic complications. This connection is evident in conditions such as diabetes, obesity, and metabolic syndrome (29,30).

The FLI is a standard biochemical score that indicates the level of liver steatosis. Research shows that as FLI rises, EFT also increases significantly. Findings from an extensive study involving 512 patients with non-alcoholic fatty liver disease demonstrated a significant and independent association between FLI and EFT. Multivariate analysis indicated that higher FLI was associated with greater EFT. Additionally, as the severity of liver steatosis escalated, EFT also showed a corresponding rise (17).

In recent years, studies have examined the relationship between EFT and carotid intima-media thickness. Karakurt et al. (31) found a significant relationship between the presence and severity of erectile dysfunction in newly diagnosed hypertensive patients and EFT and carotid intima-media thickness, and Ardahanlı et al. (32) found significant reductions in EFT and carotid intima-media

thickness with empagliflozin treatment in patients with diabetes mellitus.

No studies have directly examined the relationship between the FLI and EFT in apparently healthy young adults. Our study is the first to examine this issue.

In our study, FLI, age, and platelet count were predictive of EFT. The possible physiological reasons for this are as follows:

a) FLI

Both fatty liver and epicardial fat accumulation are indicators of insulin resistance and metabolic syndrome. Insulin resistance causes fat to accumulate in both the liver and around the heart (16). Ectopic fat tissues (such as hepatic and epicardial fat) increase systemic and local inflammation by secreting proinflammatory cytokines and adipokines. This condition increases both liver damage and cardiovascular risk (33).

b) Age

With ageing, body fat distribution changes and visceral fat, especially epicardial fat, increases. This increase contributes to an elevated cardiometabolic risk (34). Ectopic accumulation of adipose tissue in organs such as muscles, liver, and heart increases with age. Fibrotic and apoptotic changes in epicardial fat also become more pronounced with ageing (35).

c) Platelet count

EAT contributes to heightened local and systemic inflammatory responses by secreting proinflammatory cytokines and adipokines. Elevated inflammation may lead to increased platelet activation and platelet count (36). In conditions such as metabolic syndrome, obesity, and insulin resistance, both platelet count and EFT are elevated. This common increase is indicative of underlying inflammatory and metabolic disorders (26).

In our study, the echocardiographic EFT cut-off value was set at 4 mm. Previous studies have reported a wide range of EFT thresholds depending on the population studied

and the clinical outcome assessed. In patients with acute ischaemic stroke, ROC analysis identified 3.75 mm as the optimal cut-off value, while a threshold of 4 mm was adopted for practical clinical use (37). In contrast, studies evaluating coronary artery disease severity have generally reported higher cut-off values, ranging from 4.5 to 5.5 mm, for predicting the presence of coronary artery disease or more extensive multivessel involvement (38). Similarly, an EFT value of ≥ 5 mm has been associated with increased coronary artery disease risk in diabetic populations (39). Therefore, the selection of a 4 mm cut-off in our study is consistent with thresholds reported in acute ischaemic stroke populations while remaining below those described in higher-risk cardiovascular cohorts, potentially providing a more sensitivity-oriented approach for risk stratification (37-39).

Study Limitations

Our study has some limitations. Due to its cross-sectional design, we can't establish a cause-and-effect relationship. Instead, the study shows only the relationship between the variables at a single point in time, without explaining how they interact. Although the participants were deemed healthy based on their physical examination, laboratory results, and clinical assessment, they were apparently healthy but symptomatic young adults, which may limit the generalisability to community-dwelling asymptomatic young adults. Limitations of generalizability: The study population was limited to young adults aged 18-41. Therefore, the findings cannot be applied to older populations or to children. This limits the study's generalisability. The explanatory power of the multivariate regression model used in our study was limited. This suggests that the factors determining EFT cannot be explained solely by the variables included in our study and that additional metabolic, lifestyle, and behavioural determinants should also be considered. Since FLI incorporates BMI and triglycerides, which are themselves related to EFT, the observed association between FLI and EFT may be partly driven by these shared components. Therefore, our findings should be interpreted to indicate a composite cardiometabolic risk profile captured by FLI, rather than a fully independent effect of liver steatosis on EFT. The literature indicates that EFT is closely associated with insulin resistance and impaired glucose metabolism, and that EFT is significantly increased in both obese individuals and non-diabetic insulin-resistant patients (40). Excluding parameters that directly reflect insulin resistance from our model may have contributed to

unexplained variance in EFT. EFT is influenced not only by metabolic parameters but also by lifestyle factors. A cohort study conducted in the general population showed that physical activity is inversely related to EFT, while red meat consumption is directly related; alcohol consumption and heavy drinking have been reported to be associated with increased EFT, particularly in women (41). The absence of variables such as physical activity level, dietary pattern, and alcohol consumption in our analyses may significantly limit the model's explanatory power. Therefore, the limited explanatory power of our regression model indicates that additional variables, such as well-defined indicators of insulin resistance affecting EFT, detailed physical activity measurements, and dietary habits, should be integrated into future studies. The study indicated that the optimal threshold value for the FLI to predict significant EFT is 14. Although this value offers the best performance, characterized by a high Youden index (0.498) and a moderate AUC of 0.727, further research is needed to assess its effectiveness and reliability in clinical practice.

Conclusion

We found that EFT was positively associated with FLI. In addition, the participant group with higher EFT was significantly older and had a significantly higher BMI and FLI than the group with lower EFT. In this group, levels of CRP, triglycerides, and LDL were also significantly higher. Longitudinal follow-up studies may be conducted to determine whether FLI is associated with EFT in healthy young adults.

We assert that forthcoming extensive multicenter research involving healthy young adults will substantiate that the FLI can aid healthcare professionals in early identification of at-risk individuals and in the implementation of measures to reduce future cardiovascular morbidity and mortality.

Ethics

Ethics Committee Approval: The research was conducted in accordance with the Declaration of Helsinki and was approved by the Local Ethics Committee of İnönü University, as documented in decision number 2025/8194 (protocol no: 2025/8194; date: 21-08-2025).

Informed Consent: Written and signed consent was obtained from each participant.

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Footnotes

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References

- Nassir F, Rector RS, Hammoud GM, Ibdah JA. Pathogenesis and prevention of hepatic steatosis. *Gastroenterol Hepatol (N Y)*. 2015;11(3):167-175.
- Bedogni G, Bellentani S, Miglioli L, Masutti F, Passalacqua M, Castiglione A, et al. The fatty liver index: a simple and accurate predictor of hepatic steatosis in the general population. *BMC Gastroenterol*. 2006;6:33.
- Kaneva AM, Bojko ER. Fatty liver index (FLI): more than a marker of hepatic steatosis. *J Physiol Biochem*. 2024;80(1):11-26.
- Koehler EM, Schouten JN, Hansen BE, Hofman A, Stricker BH, Janssen HL. External validation of the fatty liver index for identifying nonalcoholic fatty liver disease in a population-based study. *Clin Gastroenterol Hepatol*. 2013;11(9):1201-1204.
- Carli F, Sabatini S, Gaggini M, Sironi AM, Bedogni G, Gastaldelli A. Fatty liver index (FLI) identifies not only individuals with liver steatosis but also at high cardiometabolic Risk. *Int J Mol Sci*. 2023;24(19):14651.
- Kim JH, Moon JS, Byun SJ, Lee JH, Kang DR, Sung KC, et al. Fatty liver index and development of cardiovascular disease in Koreans without pre-existing myocardial infarction and ischemic stroke: a large population-based study. *Cardiovasc Diabetol*. 2020;19(1):51.
- Zou B, Yeo YH, Cheung R, Ingelsson E, Nguyen MH. Fatty liver index and development of cardiovascular disease: findings from the UK biobank. *Dig Dis Sci*. 2021;66(6):2092-2100.
- Chung GE, Jeong SM, Cho EJ, Yoo JJ, Cho Y, Lee KN, et al. Association of fatty liver index with all-cause and disease-specific mortality: a nationwide cohort study. *Metabolism*. 2022;133:155222.
- Salazar J, Luzardo E, Mejías JC, Rojas J, Ferreira A, Rivas-Ríos JR, et al. Epicardial fat: physiological, pathological, and therapeutic implications. *Cardiol Res Pract*. 2016;2016:1291537.
- Iacobellis G, Leonetti F, Singh N, M Sharma A. Relationship of epicardial adipose tissue with atrial dimensions and diastolic function in morbidly obese subjects. *Int J Cardiol*. 2007;115(2):272-273.
- Iacobellis G, Willens HJ, Barbaro G, Sharma AM. Threshold values of high-risk echocardiographic epicardial fat thickness. *Obesity (Silver Spring)*. 2008;16(4):887-892.
- Iacobellis G, Willens HJ. Echocardiographic epicardial fat: a review of research and clinical applications. *J Am Soc Echocardiogr*. 2009;22(12):1311-1319; quiz 1417-1418.
- Petta S, Argano C, Colomba D, Cammà C, Di Marco V, Cabibi D, et al. Epicardial fat, cardiac geometry and cardiac function in patients with non-alcoholic fatty liver disease: association with the severity of liver disease. *J Hepatol*. 2015;62(4):928-933.
- Swaroop S, Tripathy SK, Sahu NC, Das B, Satpathy M. Epicardial fat thickness and its association as a risk factor for coronary artery disease. *World Journal of Cardiovascular Diseases*. 2019;9(3):193-201.
- Shambu SK, Desai N, Sundaresh N, Babu MS, Madhu B, Gona OJ. Study of correlation between epicardial fat thickness and severity of coronary artery disease. *Indian Heart J*. 2020;72(5):445-447.
- Orci LA, Jornayvaz FR, Toso C, Gariani K. Systematic review and meta-analysis of the usefulness of epicardial fat thickness as a non-invasive marker of the presence and severity of nonalcoholic fatty liver disease. *Biomedicines*. 2022;10(9):2204.
- Fracanzani AL, Pisano G, Consonni D, Tiraboschi S, Baragetti A, Bertelli C, et al. Epicardial adipose tissue (EAT) thickness is associated with cardiovascular and liver damage in nonalcoholic fatty liver disease. *PLoS One*. 2016;11(9):e0162473.
- Fluss R, Faraggi D, Reiser B. Estimation of the Youden index and its associated cutoff point. *Biom J*. 2005;47(4):458-472.
- An L, Wirth U, Koch D, Schirren M, Drefs M, Koliogiannis D, et al. The role of gut-derived lipopolysaccharides and the intestinal barrier in fatty liver diseases. *J Gastrointest Surg*. 2022;26(3):671-683.
- Viscarra J, Sul HS. Epigenetic regulation of hepatic lipogenesis: role in hepatosteatosis and diabetes. *Diabetes*. 2020;69(4):525-531.
- Carter J, Heseltine TD, Meah MN, Tzolos E, Kwiecinski J, Doris M, et al. Hepatosteatosis and atherosclerotic plaque at coronary CT angiography. *Radiol Cardiothorac Imaging*. 2022;4(2):e210260.
- Choi KM, Han K, Park S, Chung HS, Kim NH, Yoo HJ, et al. Implication of liver enzymes on incident cardiovascular diseases and mortality: a nationwide population-based cohort study. *Sci Rep*. 2018;8(1):3764.
- Heseltine T, Murray S, Ortega Martorell S, Olier I, Lip GYH, Khoo S. Associations of hepatosteatosis with cardiovascular disease in HIV-positive and HIV-negative patients: the Liverpool HIV-heart project. *J Acquir Immune Defic Syndr*. 2021;87(5):1221-1227.
- Castellana M, Donghia R, Guerra V, Procino F, Lampignano L, Castellana F, et al. Performance of fatty liver index in identifying non-alcoholic fatty liver disease in population studies. A meta-analysis. *J Clin Med*. 2021;10(9):1877.
- Iacobellis G. Epicardial adipose tissue in contemporary cardiology. *Nat Rev Cardiol*. 2022;19(9):593-606.
- Chong B, Jayabaskaran J, Ruban J, Goh R, Chin YH, Kong G, et al. Epicardial adipose tissue assessed by computed tomography and echocardiography are associated with adverse cardiovascular outcomes: a systematic review and meta-analysis. *Circ Cardiovasc Imaging*. 2023;16(5):e015159.
- Villasante Fricke AC, Iacobellis G. Epicardial adipose tissue: clinical biomarker of cardio-metabolic risk. *Int J Mol Sci*. 2019;20(23):5989.
- Iacobellis G. Local and systemic effects of the multifaceted epicardial adipose tissue depot. *Nat Rev Endocrinol*. 2015;11(6):363-371.
- Aitken-Buck HM, Moharram M, Babakr AA, Reijers R, Van Hout I, Fomison-Nurse IC, et al. Relationship between epicardial adipose tissue thickness and epicardial adipocyte size with increasing body mass index. *Adipocyte*. 2019;8(1):412-420.
- Gruzdeva O, Uchasova E, Dyleva Y, Borodkina D, Akbasheva O, Belik E, et al. Relationships between epicardial adipose tissue thickness and adipo-fibrokinase indicator profiles post-myocardial infarction. *Cardiovasc Diabetol*. 2018;17(1):40.
- Karakurt D, Ede H, Turan Y, Gürel A, Ardahanlı İ, Sarak T. Yeni tanı konmuş hipertansiyonu olan hastalarda erektil disfonksiyon ile epikardiyal yağ dokusu kalınlığı ve karotis intima-media kalınlığı arasındaki ilişkinin değerlendirilmesi [Evaluation of the relationship between erectile dysfunction and epicardial fat tissue thickness and carotid intima-media thickness in patients with newly diagnosed hypertension]. *Turk Kardiyol Dem Ars*. 2020;48(8):746-753.

32. Ardahanlı İ, Aslan R, Çelik M, Akgün O, Akyüz O. Effects of empagliflozin on carotid intima-media thickness and epicardial fat tissue volume in patients with type-2 diabetes mellitus. *Lokman Hekim Health Sci.* 2021;1(3):74-80.
33. Petta S, Craxi A. Epicardial fat in patients with non-alcoholic fatty liver disease. *J Hepatol.* 2015;62(5):1215.
34. Chen YC, Lee WH, Lee MK, Hsu PC, Tsai WC, Chu CY, et al. Epicardial adipose tissue thickness is not associated with adverse cardiovascular events in patients undergoing haemodialysis. *Sci Rep.* 2020;10(1):6281.
35. Iacobellis G. Aging effects on epicardial adipose tissue. *Front Aging.* 2021;2:666260.
36. Ustuntas G, Basat SU, Calik AN, Sivritepe R, Basat O. Relationship between epicardial fat tissue thickness and CRP and neutrophil-lymphocyte ratio in metabolic syndrome patients over 65 years. *Sisli Etfal Hastan Tip Bul.* 2021;55(3):405-411.
37. Qayyum W, Iqbal M, Khan S, Yousafzai Z, Nabi D, Ashraf S, et al. Epicardial fat thickness as a predictor of acute ischemic stroke at Rehman medical institute: a case-control study. *J Rehman Med Inst.* 2025;11(1):19-23.
38. El-Naggar HM, Abdel-Maseh JG, Hasan-Ali H, Khidr SS. Epicardial fat thickness predicts severe coronary artery disease and high mortality risk among ST-elevation myocardial infarction patients. *Echo Res Pract.* 2025;12(1):17.
39. Akhter AN, Aisha F, Moazzam AB, Khan SHB, Malik J, Parveen A. Epicardial fat thickness as a marker of coronary artery disease in diabetics: a single center study. *Clin Cardiol.* 2025;48(7):e70171.
40. Altin C, Sade LE, Gezmis E, Yilmaz M, Ozen N, Muderrisoglu H. Assessment of epicardial adipose tissue and carotid/femoral intima media thickness in insulin resistance. *J Cardiol.* 2017;69(6):843-850.
41. Gustafsson B, Rovio SP, Ruohonen S, Hutri-Kähönen N, Kähönen M, Viikari JSA, et al. Determinants of echocardiographic epicardial adipose tissue in a general middle-aged population - The Cardiovascular Risk in Young Finns Study. *Sci Rep.* 2024;14(1):11982.