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ORIGINAL ARTICLE

Can Cardiovascular Risk Be Simply Estimated in Nonalcoholic Fatty Liver Disease Patients?

Non-Alkolik Yağlı Karaciğer Hastalarında Kardiyovasküler Riski Kolayca Tahmin Etmek Mümkün mü?

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ABSTRACT

Backgrounds and Aims: In the pathogenesis of nonalcoholic fatty liver disease (NAFLD), inflammation plays a pivotal role. The presence of inflammatory cells is closely linked with epicardial adipose tissue (EAT). A recently identified prognostic indicator for cardiovascular disease (CVD) is the ratio of monocyte count to HDL-cholesterol (MHR). Our primary aim was to investigate the relationship between EAT and markers of inflammation in individuals with NAFLD, and to evaluate its predictability using straightforward diagnostic measures.
Material-Method: This retrospective study included 218 patients who underwent thoracic computed tomography angiography between 2014 and 2021. The patients were divided into the NAFLD group (HU<48 IU) and the non-NAFLD group (HU>48 IU) according to the liver attenuation ratio. 136 patients in the NAFLD group and 82 in the non-NAFLD group.
Results: The body mass index (BMI), triglyceride levels, notably the EAT volume and MHR in the NAFLD group, exhibited higher values than non-NAFLD group. Among participants in the NAFLD group, a positive correlation was observed between EAT volume and factors such as age, MHR, c-reactive protein, BMI, urea, glucose, and alanine aminotransferase. Through linear regression analysis, it was determined that MHR stood as the sole independent predictor of EAT volume in patients with NAFLD.
Conclusion: EAT volume, a risk marker for CVD, can be predicted in NAFLD patients by MHR without radiological methods. Thus, easier and earlier detection of NAFLD patients in the high-risk group for CVD will be possible.

Keywords: Epicardial adipose tissue, cardiovascular disease, monocyte-HDL ratio, nonalcoholic fatty liver disease, inflammation

ÖZ

Amaç: Enflamasyon, alkolsüz yağlı karaciğer hastalığının (NAYKH) patogenezinde önemli rol oynar. Epikardiyal yağ dokusu (EYD), enflamatuar hücrelerle yakından ilişkilidir. Monosit sayısının HDL-kolesterol'e (MHR) oranı, kardiyovasküler hastalık (KVH) için yeni bir prognostik belirteçtir. NAYKH hastalarında EYD ile inflamatuvar göstergeler arasındaki ilişkiyi ve bunun basit testlerle tahmin edilip edilemeyeceğini araştırmak istedik.
Gereç-Yöntem: Bu retrospektif çalışmaya 2014-2021 yılları arasında toraks BT anjiyografi çekilen 218 hasta dahil edildi. Hastalar karaciğer atenüasyon oranına göre NAYKH grubu (HU<48 IU) ve NAYKH olmayan grup (HU>48 IU) olarak ayrıldı. NAYKH grubunda 136 hasta ve NAYKH olmayan grupta 82 hasta.
Bulgular: NAYKH grubunun vücut kitle indeksi (VKİ), trigliserit, EYD hacmi ve MHR değerleri diğer grutan yüksekti. NAYKH grubunda ise EYD hacmi, yaş, MHR, c-reaktif protein (CRP), VKI, üre, glikoz ve alanin aminotransferaz (ALT) ile pozitif korelasyon gösterdi. NAFLD hasta grubunda MHR, EYD hacminin tek başına bağımsız ön gördürücüsüydü.
Sonuç: KVH için bir risk belirteci olan EYD hacmi, NAYKH hastalarında radyolojik yöntemler olmaksızın MHR ile tahmin edilebilir. Böylece KVH açısından yüksek risk grubunda yer alan NAYKH hastalarının daha kolay ve erken saptanması mümkün olacaktır.

Anahtar Kelimeler: Epikardial yağ dokusu, kardiyovaskuler hastalık, monosit-HDL oranı, nonalkolik yaplı karaciğer hastalığı, inflamasyon

Introduction

Nonalcoholic fatty liver disease (NAFLD) is distinguished by elevated lipid accumulation within hepatocytes, resulting in liver cell damage attributed to inflammation in the portal-lobular area. Its incidence has surged to 40%, with an increasing prevalence, as documented in recent studies (1). Patients with NAFLD have an elevated susceptibility to cardiovascular disease. The complex origin of NAFLD, commonly referred to as the multiple-hit hypothesis, encompasses factors like insulin resistance, oxidative stress, and persistent inflammation. Furthermore, persistent inflammation plays a role in fat accumulation, contributing to the development of steatosis in the context of NAFLD (2). Various cell types, including monocytes and macrophages, play key roles as inflammatory mediators leading to hepatic infiltration. This intricate inflammatory cascade significantly contributes to the advancement of NAFLD and the subsequent development of liver fibrosis (3).

The accumulation of adipose tissue located between

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the myocardium's outer wall and the pericardium's visceral layer is called epicardial adipose tissue (EAT). EAT's volumetric component constitutes roughly 20% of the overall heart volume, while its mass constitutes approximately 1% of the aggregate adipose tissue mass. Age, ethnicity, waist circumference, and cardiac mass emerge as distinct indicators for EAT volume (4). During the early stages of mild accumulation, EAT exerts anti-inflammatory effects; however, as its quantity escalates during later phases, it shifts toward pro-atherogenic and pro-inflammatory outcomes, precipitating cardio-metabolic events (5).

Monocytes are a pivotal cellular subset accountable for the secretion of cytokines with proinflammatory and pro-oxidant properties. The anti-inflammatory characteristics of high-density lipoprotein cholesterol (HDL-C) are evidenced in its capacity to shield endothelial cells against the adverse effects of low-density lipoprotein (LDL) and hinder the oxidation process. Within cardiovascular disease (CVD), the ratio of monocyte count to HDL-C (MHR) has arisen as an emerging predictive marker with prognostic implications (6,7). MHR is an easy-to-calculate, low-cost, and highly predictive determinant of CVD estimation, which has become increasingly important in recent years. MHR, easily calculated through routine and cost-effective tests, serves as a valuable predictor for certain diseases (8).

Both hepatosteatosis and EAT cause an increased risk of CVD, and the relationship of both local adipose tissue deposition patterns with inflammatory cells is intriguing for the relationship between inflammatory markers and EAT accumulation in NAFLD patients. Although ultrasonography stands as the prevalent diagnostic approach for hepatosteatosis, a standardized measurement technique for EAT remains absent. Transthoracic echocardiography is frequently favored for EAT assessment, primarily due to its attributes of easy accessibility, non-invasiveness, and cost-effectiveness. The predominant methodology for quantifying EAT involves gauging its volumetric content (9). EAT volume can be quantified through the utilization of computed tomography (CT) and magnetic resonance (MR) imaging. These radiological methods provide superior precision and accuracy in measuring EAT compared to transthoracic echocardiography. Furthermore, both CT and MR approaches exhibit augmented sensitivity and specificity, albeit at the cost of increased expense and procedural complexity (9). Therefore, predicting the amount of EAT using routine and easily accessible laboratory tests becomes crucial. The primary aim of this study was to predict EAT volume in NAFLD patients using simple diagnostic methods.

Material-Methods

This retrospective study was conducted with ethical approval from the hospital medical ethics committee (Approval Date: 25.03.2020, Ethical Committee Number: 14567952-050/460). This study included 218 consecutive patients who underwent thorax computer tomography (CT) angiography for any medical indication (suspected pulmonary embolism, suspected aortic aneurysm, etc.) between 2014 and 2021. A comprehensive retrospective examination of medical records was conducted utilizing the hospital's information system. This encompassed a range of data points including gender, age, body mass index (BMI), medication history, disease duration, the existence of cardiovascular disease (CVD), familial predisposition to CVD, as well as detailed biochemical profiles of blood lipid levels.

The following criteria were determined as inclusion criteria for the study. 1) age >18 years old 2) not pregnant 3) no history of diabetes mellitus or inflammatory disease 4) not using glucocorticoids, anti-inflammatory/immunomodulatory drugs and lipid-lowering therapy 5) not being diagnosed with any infective disease at the time of CT scan. Patients with a history of diabetes mellitus, cardiac operations such as coronary bypass surgery or heart valve surgery, patients with chronic liver disease, primary liver tumor or liver metastasis that will cause changes in the liver parenchyma, and patients who use alcohol were excluded from the study. In addition, volunteers with insufficient clinical-laboratory data were not included in the study.

According to the liver attenuation rate, the patients were divided into the NAFLD group with the liver density below 48 HU and the non-NAFLD group with the liver density above 48 HU (10).

Radiological Evaluation

CT examinations were executed utilizing identical scanning parameters on a Siemens dual-energy Computed Tomography scanner (Somatom Drive; Siemens Healthcare in Germany). The scanning configuration encompassed a tube voltage of 120 kV, a tube current of 80 mAs, a high-pitch spiral acquisition mode, and a reconstructed slice thickness of 1.5 mm. The process was prospectively triggered

by electrocardiography, and the scanning scope spanned from the tracheal carina to the level of the liver's portal vein.

The heart rate of all patients was controlled with a β-blocker so that the heart rate was less than 65 beats/min. A radiologist blinded to the study protocol evaluated EAT and hepatosteatosis measurements.

Hepatic CT attenuation was evaluated through the delineation of three distinct regions of interest (ROI) positioned at the level of the portal vein within the liver. These ROIs were strategically located within the hepatic parenchyma, deliberately excluding the biliary, vascular, and extrahepatic structures from consideration (as illustrated in Figure-1).

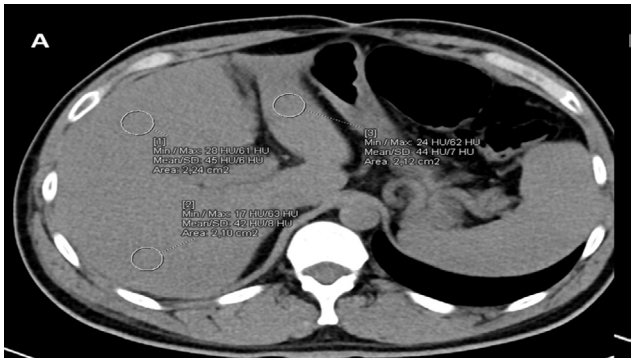

Figure 1. An unenhanced transverse CT image depicts a circular region of interest (ROI) measuring 1.5 x 1.5 cm (white circle), deliberately placed to avoid major blood vessels. The ROI is strategically positioned across the right anterior-posterior and left hepatic lobes.

A semi-automatic assessment of EAT volume was executed using non-contrast CT images, employing the subsequent approach. Initially, the observer identified the upper and lower boundaries of the pericardial sac, delineated respectively as the bifurcation point of the pulmonary trunk and the slice positioned caudally to the posterior descending artery. Subsequently, the pericardial sac's contour was automatically delineated and subject to manual adjustments by the observer, as necessary. The EAT volume (depicted in green) in milliliters (ml) was computed automatically by encompassing all contiguous 3D voxels with CT attenuations ranging between the designated upper threshold (in this case, -40 HU) and the lower threshold of -200 HU (as demonstrated in Figure-2 and 3).

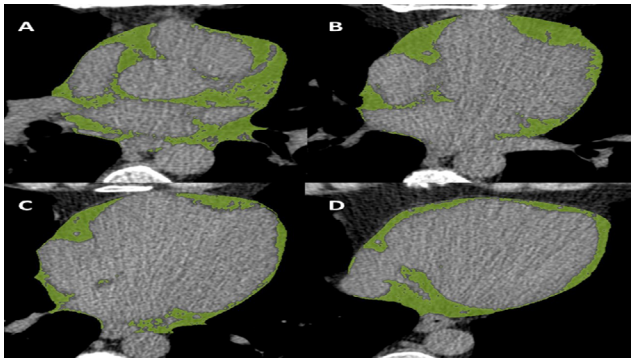

Figure 2. Panels (A-D) portray the process of semiautomatically identifying the epicardial adipose tissue area (depicted in green) within an unenhanced transverse CT image.

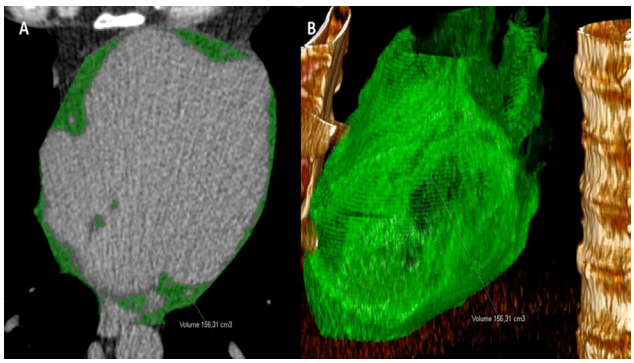

Figure 3. Subfigure (A) showcases the delineation of the epicardial adipose tissue area (depicted in green) via the tracing of a singular region of interest on an unenhanced transverse CT image. Adipose voxels are discerned utilizing an attenuation range threshold spanning from -200 to -40 HU. Subfigure (B) illustrates the automated computation of the epicardial adipose tissue volume (in cm3), arrived at by summing the areas of adipose tissue for the entire heart.

Biochemical Analyses

Serum C-reactive protein (CRP) levels and routine blood paramethers were measered.

Statistical Analyses

The Statistical Package for Social Sciences for Windows 21.0 (SPSS Inc. Chicago, Illinois, USA) used for data analysis. Descriptive statistics were computed for each variable, with data presented as mean ± standard deviation. The $\chi 2$ test was utilized to assess statistically significant differences among categorical variables. Both nonparametric methods (Mann-Whitney U) and parametric methods (independent sample t-test) were employed for continuous variables. Correlations between variables were explored through Spearman's rho test. To identify independent predictors for EAT, binary logistic regression analysis was executed. In the regression analysis, factors with a p-value of <0.2 were included in the initial univariate analysis, and those exhibiting significance in the univariate phase were subsequently incorporated into the multivariable evaluation. The backward elimination approach was employed in the stepwise regression analysis, utilizing a p-value threshold of >0.1 for elimination criteria. A statistically significant distinction was recognized when the p-value was <0.05.

Results

The demographic characteristics and laboratory parameters of the study's cohort, encompassing 218 patients (113 females, 105 males), are outlined in Table-1. These patients were categorized into distinct groups based on their liver density, yielding the NAFLD and non-NAFLD groups. The NAFLD group consisted of 136 patients (72 females, 64 males), while the non-NAFLD group comprised 82 patients (41 females, 41 males). Within the NAFLD group, there existed higher BMI and triglyceride levels in comparison to the non-NAFLD group. While the white blood cell count and levels of CRP exhibited similarity between the two groups in terms of inflammatory markers, the MHR was notably elevated in the NAFLD group (p:0.03) (as presented in Table-1). The mean MHR was observed to be 12.24(6.41) among patients with NAFLD and 10.3(5.08) in the non-NAFLD cohort, with a statistically significant distinction noted between these groups (p:0.03) (as depicted in Figure-4). Furthermore, the volumes of epicardial adipose tissue (EAT) exhibited a significant increase within the NAFLD group (mean: 82.32) as opposed to the non-NAFLD group (mean: 62.07), with a p-value of 0.002 (Figure-4).

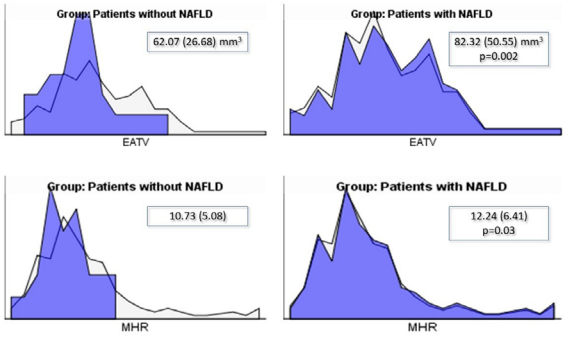

Figure 4. A comparative representation of EAT volume and the MHR in patients afflicted by NAFLD against those without NAFLD.

Table 1. Demographic data and laboratory measurements of the patient and control groups

Parameters	NAFLD (n=136) Mean±SD or Median (IQR)	Non-NAFLD (n=82) Mean±SD or Median (IQR)	**p Value**
Gender (M/F)	64/72	41/41	0.258
Age (years)	50.21 (12.16)	40.81 (9.05)	0.001
Body Mass Index (kg/m^2)	29.41 (5.02)	25.32 (1.75)	0.001
Hemoglobin (g/dl)	14.15 (1.62)	14.17 (1.57)	0.96
White blood count (103/uL)	7881.54 (2303.05)	7322.31 (1655.89)	0.24
Platelet count (103 /mm3)	263 (82)	250.50 (69)	0.45
Urea (mg/dl)	29 (13)	26 (15)	0.33
Creatinine (mg/dL)	0.82 (0.21)	0.89 (0.18)	0.001
Glomeruler filtration rate (ml/dk)	91.94 (20.28)	85.15 (28.88)	0.14
Glucose (mg/dl)	100 (23)	94 (23)	0.53
Total cholesterol (mg/dl)	203.28 (47.22)	188.71 (40.73)	0.14
Low density lipoprotein (mg/dl)	124.75 (39.32)	120.16 (32.39)	0.57
Triglyceride (mg/dl)	144.88 (100.50)	101.50 (72)	0.01
C-reactive protein (mg/L)	2.20 (2.62)	2.15 (3.35)	0.73
Alanine aminotransferase (U/L)	17 (10)	16 (7)	0.10
Aspartate aminotransferase (U/L)	18 (8)	16 (6)	0.96
Albumin (g/L)	4.23 (0.40)	4.34 (0.39)	0.20

A bivariate correlation analysis was conducted to ascertain the associations between various parameters and the volume of EAT within the NAFLD group. Age, MHR, CRP, BMI, urea, blood glucose, and alanine aminotransferase (ALT) were positive correlate with EAT volume (Table-2).

Table 2. Bivariate Correlation Analysis between epicardial adipose tissue volume and other parameters in NAFLD groups

Parameters	Rs	**p value**
Age (years)	0.193	0.024
Monocyte HDL-cholesterol ratio	0.348	0.004
C-reactive protein (mg/L)	0.187	0.038
Body Mass Index (kg/m^2)	0.297	0.008
Urea (mg/dl)	0.185	0.031
Glucose (mg/dl)	0.164	0.050
Alanine aminotransferase (U/L)	0.171	0.047

Furthermore, we conducted an assessment to determine variables that exhibited independent associations with the volume of EAT in patients diagnosed with NAFLD (as outlined in Table-3). The variables considered for inclusion in this model encompassed age, MHR, ALT, CRP, BMI, and glomerular filtration rate (GFR). Notably, the MHR emerged as the sole independent predictor of EAT volume (p-value: 0.033), as delineated in Table-3.

Table 3. Independent Variable of EAT in Patients with NAFLD

Parameters	Standardized beta	t	P-value	95% CI
Step 1				
Age (years)	0.124	1.059	0.292	-0.303 – 0.999
Monocyte HDL-cholesterol ratio	0.202	1.946	0.054	-0.18 – 2.009
Alanine aminotransferase (U/L)	0.137	1.479	0.142	-0.184 -1.267
C-reactive protein (mg/L)	0.032	0.338	0.736	-0.816 – 0.152
Body Mass Index (kg/m^2)	-0.034	-0.339	0.735	-1.550 – 1.097
Triglyceride (mg/dl)	-0.060	-0.589	0.557	-0.069 – 0.037

Glomeruler filtration rate (ml/dk)	0.053	0.476	0.635	- 0.290 – 0.474
Step 7				
Monocyte HDL-cholesterol ratio	0.194	2.153	0.033	0.077 – 1.835

Discussion

Within the context of this investigation, it was observed that the volume of EAT exhibited a noteworthy increase among patients diagnosed with NAFLD as compared to individuals devoid of hepatosteatosis. The most important result of the study is that EAT volume, which is a risk marker for CVD, can be predicted by MHR, which is a simple and frequently used laboratory test in clinical practice, even without using radiological methods in NAFLD patients. To our present understanding, this study stands as the pioneering endeavor in the existing body of literature to unveil the predictive capacity of the MHR with regard to EAT. Notably, no preceding studies have showcased the MHR as a predictor of EAT among patients afflicted with NAFLD or within distinct population cohorts. Another significant result we found was that age, CRP, BMI, blood glucose, and ALT levels were positively correlated with EAT volume in NAFLD patients.

Monocytes are the main source of proinflammatory cytokines (11). HDL-C manifests anti-inflammatory attributes through its capacity to impede monocyte activation, safeguard endothelial cells from the detrimental impacts of LDL-C, and counteract LDL oxidation. This unique functionality has led to the adoption of the MHR as a novel prognostic indicator for CVD in recent times. (6,7).

Recent investigations have revealed a notable observation: the MHR presents higher values in patients with NAFLD when contrasted with those without hepatosteatosis. Moreover, a significant correlation has been established between elevated MHR levels and an augmented risk of NAFLD (12,13). Monocyte-derived macrophages are of paramount significance in NAFLD's progression and liver fibrosis, which are also implicated in hepatic infiltration (3). Conversely, HDL-C engenders antioxidant and anti-inflammatory effects in NAFLD development by constraining the generation of oxidized LDL-cholesterol and suppressing monocyte proliferation (14). This impels consideration of the possibility that heightened MHR could potentially serve as an indicator of escalated inflammatory burden and oxidative stress within the context of NAFLD. Furthermore, escalated deposition of visceral adipose tissue fosters the secretion of proinflammatory cytokines, thereby instigating chronic systemic low-grade inflammation. This pathophysiological continuum proceeds to encompass insulin resistance and an elevated incidence of CVD. Is worth noting that MHR is a valuable marker for metabolic syndrome, an entity encompassing an array of risk factors (8).

Both the inflammatory cytokines causing EAT accumulation and the inflammatory properties of EAT led to the idea that EAT could be associated with an inflammatory parameter such as MHR. While EAT has anti-inflammatory properties when there is mild accumulation in the early stages, it has pro-inflammatory, pro-atherogenic, and oxidative stress-increasing effects that cause cardio-metabolic events due to the change in adipokines secreted in the later periods when the accumulation increases (5). As the biological composition of EAT undergoes alteration, there ensues an augmentation in the production of proinflammatory adipokines, notably including tumor necrosis factor-a (TNF-a), interleukin 1-β (IL1-β), interleukin-6 (IL1-6), and resistin (15). The primary mechanisms through which EAT contributes to CVD are rooted in pathogenic processes driven by inflammation and oxidative stress CVD (16). EAT serves as a source of proinflammatory adipocytokines that are released into the systemic circulation, thereby inciting a systemic inflammatory response. This, in turn, triggers a positive feedback loop that fosters an expansion of EAT (15). While previous studies have explored the correlation between diverse inflammatory parameters and EAT, it is noteworthy that our study will introduce, for the first time in the existing literature, the association between the MHR and EAT (17,18).

The initial evidence pointing to the correlation between EAT and NAFLD was established in the year 2014 (19). Iacobellis et al. highlighted that individuals classified as obese and afflicted by NAFLD exhibited higher EAT levels compared to obese individuals without hepatosteatosis. Their findings indicated that epicardial fat holds predictive potential for identifying NAFLD among obese individuals. Additionally, the echocardiographically assessed measurement of EAT showcased superior predictive accuracy for liver fat compared to traditional metrics such as BMI or waist circumference (19). After this seminal research, numerous subsequent studies have consistently underscored the augmented EAT accumulation among patients diagnosed with NAFLD, further

elucidating its connection to cardiovascular risk factors (17,19,20). A recent meta-analysis, compiling data from 13 studies probing the relationship between EAT and NAFLD, corroborated that EAT tends to be elevated in individuals harboring liver fat. Furthermore, this increase in EAT was notably linked to adiposity, fibrosis, and the severity of cardiovascular disease within the NAFLD patient cohort (21). The collective insights gleaned from these investigations illuminate the paramount significance of ectopic fat accumulation and accentuate the necessity for easily accessible markers in the clinical assessment of these conditions.

Upon scrutinizing the CT measurements of our patients' EAT volumes, accounting for the localized distribution of EAT, regional discrepancies, and variations across patients of diverse ethnic origins, we find concordance with analogous measurement methodologies employed in the relevant literature (22,23). Within our investigation, EAT volume displayed elevated levels in individuals with NAFLD who exhibited elevated blood glucose levels. Diabetic individuals tend to exhibit greater EAT volumes than their healthy counterparts, with age and waist circumference emerging as key independent determinants of EAT volume in the broader healthy population (4,24,25). In addition, it has been previously shown that BMI and diabetic status are not independent factors in the relationship between EAT and CVD and that one of the essential factors is visceral adiposity and the resulting insulin resistance or metabolic syndrome (26,27). In our study, blood glucose level and age were correlated with EAT, and the data support the literature. It can be thought that the chronic inflammatory state that continues continuously with aging causes a cumulative effect on EAT accumulation.

Our study determined that EAT volume increased as BMI increased in NAFLD patients. BMI is associated with visceral adipose tissue; if total body weight increases, visceral adipose tissue, fatty liver, and EAT also increase (28). Inflammation caused by increased adipose tissue also contributes to the formation of adipose tissue with positive feedback. Analogous to the accumulation of visceral adipose tissue observed in obese individuals, EAT likewise demonstrates an augmentation. This expansion is accompanied by alterations in EAT's biological composition and its anti-inflammatory attributes (29). In obese individuals, EAT's inflammatory properties are more prominent than its anti-inflammatory properties (26). Our data showing that BMI and MHR are higher in patients with NAFLD support these pathophysiological processes. In contrast to BMI and waist circumference, the thickness of EAT emerges as a more informative marker for hepatosteatosis (19). However, when interpreting the findings from the study that highlights the higher EAT thickness in obese patients with hepatosteatosis compared to obese individuals without hepatosteatosis, drawing a conclusive determination regarding whether the increase in EAT is solely attributed to hepatosteatosis in isolation from obesity remains elusive. However, it is not appropriate to comment only on BMI in the relationship between EAT and hepatosteatosis, and more valuable EAT markers are definitely needed (19). A wealth of evidence underscores that the distribution of adipose tissue in specific regions holds a stronger association with CVD risk compared to the overall excess of body fat resulting from obesity (30). Obesity stands as an autonomous risk factor for CVD, yet it does not serve as an independent predictor of EAT (25). In addition, EAT's inflammatory cytokine production is higher than subcutaneous adipose tissue (15). This may explain why increased visceral adiposity, such as EAT, rather than overall body weight gain, is associated with a greater risk of CVD.

The correlation between EAT and serum transaminases is independent of obesity and is mostly associated with excessive visceral adipose tissue deposition (31,32). Our dataset substantiates the correlation between ALT and EAT among individuals diagnosed with NAFLD. Undoubtedly, revealing the intricate interplay between liver fibrosis, ALT, and EAT via liver biopsy would significantly enhance the precision in elucidating the underlying mechanisms in this context.

This study has two significant limitations. Primarily, NAFLD diagnosis was based on CT rather than biopsy. While radiological techniques can determine liver density and the extent of hepatosteatosis, they may not fully account for potential factors like iron accumulation or inflammatory processes that could impact liver density (33,34). Secondly, many therapeutic medications used for managing dyslipidemia, hypertension, and diabetes mellitus have been demonstrated to influence MHR, thereby presenting potential confounding factors.

Conclusion

We propose that MHR, a simple and widely used test for assessing EAT—a crucial predictor for cardiovascular disease—can be efficiently employed in clinical practice for NAFLD patients. This approach facilitates the early and efficient identification of NAFLD patients

at risk of cardiovascular disease.

Main Points

•EAT volume is higher in NAFLD patients than in patients without hepatosteatosis.

•EAT volume, a risk marker for CVD, can be predicted by MHR, a simple and frequently used laboratory test in clinical practice, even without using radiological methods in NAFLD patients.

•This study represents the inaugural endeavor in the existing literature to unveil the MHR as a predictor of EAT.

•Age, CRP, BMI, blood glucose, and ALT levels are positively correlated with EAT volume in NAFLD patients.

Conflicts of interest: All authors declare that there is no conflict of interest in this study.

Informed consent: Ethics committee approval was obtained from the institution for the study and written consent was obtained from all patients.

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