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The role of lipid-derived indices in patients with MASLD in Turkey: a nationwide, multicenter cross-sectional study

İhsan Solmaz[1]✉, Ali Kırık[2,3], İsmail Demir[4], Oğuzhan Sıtkı Dizdar[5], Nevzat Gözel[6], Gökhan Tazegül[7], Bilgin Bahadır Başgöz[8], Cem Şahin[9], Kubilay İşsever[10], Gülali Aktaş[11], Pınar Yıldız[12], Emin Gemcioğlu[13], Enes Seyda Şahiner[14], Hilal Bektaş Uysal[15], Yasin Şahintürk[16], Ömer Faruk Alakuş[1], Mustafa Akdeniz[1], Hilmi Erdem Sümbül[17], Nizameddin Koca[18], Türkan Paşalı Kilit[19], Sibel Demiral Sezer[20], Emine Binnetoglu[21], Eşref Araç[22], Hacer Şen[2], Bahri Abaylı[23], Hale Akan[24], Canan Akkuş[13], Berrin Aksakal[25], Burçin Meryem Atak Tel[11], Ahmet Aydın[26], Sami Bahçebaşı[5], Orhan Balıkçı[20], Lale Saka Baraz[24], Muharrem Bayrak[27], Hatice Beyazal Polat[28], İfakat İrem Biçer[5], Rıfat Bozkuş[13], Fatih Coşkun[29], Cüneyt Çağatay[18], Feride Çağlar[13], Erkan Çakmak[6], Deniz Cekiç[30], Ülfet Değer[7], Ayse Kevser Demir[31], Erkan Dulkadiroğlu[32], Nur Düzen Oflas[33], Betül Erişmiş[14], Ali Erol[29], Ayşegül Ertınmaz[18], Müzeyyen Eryılmaz[34], Ahmed Bilal Genç[30], Melis Gökgöz[13], Fatih İleri[18], Uğur Can İzlimek[35], Özge Kama Başcı[2], Aynur Kamburoğlu[18], Fatih Kamış[36], Sanem Kayhan[13], İsmail Kırlı[9], Yusuf Kimyon[7], Şeref Enes Kocaman[29], Kamil Konur [28]✉, Özge Kurtkulağı[36], Celalettin Küçük[37], Mehmet Selim Mamiş[38], Hatice Metin[18], Necip Nas[35], Sibel Ocak Serin[25], Oktay Olmuşçelik[26], Alihan Oral[39], Muhammet Özbilen[40], Erkan Özdemir[41], Ensar Özmen[42], Hikmet Öztop[43], Hüseyin Ali Özturk[17], Osman Özüdoğru[44], Emel Sağlam[45], Hatice Özge Serin[7], Hasan Sözel[46], Melisa Şahin Tekin[12], Ahmet Veli Şanibaş[31], Hakan Şıvgın[47], Abdullah Tanrıkulu[38], Tuba Taslamacıoğlu Duman[11], Elif Duygu Topan[15], Hasan Tunca[9], Seyit Uyar[16], Ece Ünal Çetin[36], Nazif Yalçın[18], Demet Yalçın Kehribar[48], Selçuk Yaylacı[30], Hasan Esat Yıldırım[29], Hüseyin Yıldız[41], Hasan Esat Yücel[32], Oğuzhan Zengin[14], Ali Zeynettin[4], Fatih Atik[2], Selin Müge Aslan[2], Mert Akyıldız[17], Nurcan Aslan[14], Sare Babacan Çelikel[6], Suat Baran Bakan[34], Merve Durmuş[17], Mert Karacay[17], Çağatay Koçyiğitoğlu[17], Gökçe Paşa[17], Selvinaz Sivri[17], Tutku Naz Şahin[17], Yağmur Sena Tosun[14], Zeynep Gizem Totik [17], Rabia Ulutaş[48], Tuğçe Nur Yazıcı[40], Emrah Yılmaz[44] & Hamit Yıldız[49]

To investigate the role of lipid-derived indices—the monocyte-to-HDL cholesterol ratio (MHR), visceral adiposity index (VAI), atherogenic index of plasma (PAI), and cardiometabolic index (CMI)—in determining hepatic steatosis and fibrosis among patients with metabolic dysfunction-associated steatotic liver disease (MASLD) across Turkey. This nationwide, multicenter, retrospective cross-sectional study included 14,322 individuals from 44 internal medicine clinics in 31 provinces of Turkey. Anthropometric, clinical, and biochemical data were collected, and Participants were classified as MASLD (+) if ultrasonographic hepatic steatosis was present in the setting of ≥ 1 cardiometabolic risk factor according to current EASL–EASD–EASO guidelines; individuals without ultrasonographic steatosis were classified as MASLD (–). FIB-4 scores were calculated to assess the risk of advanced hepatic fibrosis. Cardiometabolic risk factors included obesity (BMI ≥ 25 kg/m² or increased waist circumference), type 2 diabetes mellitus, hypertension, impaired fasting glucose, and dyslipidemia as defined by international guidelines. The relationships between MHR, VAI, PAI, CMI, and MASLD presence and fibrosis severity were analyzed via nonparametric statistical tests. MASLD was detected in 10,836 participants (75.7%). The VAI, PAI, and CMI were significantly greater in the MASLD (+) group than in the MASLD (–) group ($p < 0.001$ for all), whereas the MHR was not significantly different

($p = 0.453$). Among MASLD (+) patients, 1,214 (11.2%) had high FIB-4 scores (≥ 1.3; ≥2.0 if > 65 years) yet none of the lipid indices correlated significantly with FIB-4 levels. In multivariable logistic regression analysis restricted to MASLD-positive individuals, none of the lipid-derived indices were independently associated with high FIB-4 risk after adjustment for metabolic covariates. A weak negative correlation was observed only between FIB-4 score and the MHR (r=–0.030, $p = 0.002$). VAI, PAI, and CMI are significantly associated with hepatic steatosis in MASLD, suggesting their potential role as supportive, noninvasive markers in identifying individuals at increased risk of MASLD. However, these indices are not reliable predictors of advanced hepatic fibrosis risk based on FIB-4. The combination of these tools with other noninvasive fibrosis assessment tools may increase diagnostic accuracy in MASLD management.

Keywords MASLD, lipid indices, visceral adiposity index, atherogenic index of plasma, cardiometabolic index, monocyte-to-HDL ratio, FIB-4, hepatic fibrosis

[1]Department of Internal Medicine, Diyarbakır Gazi Yasargil Training and Research Hospital, Kayapınar, 21010 Diyarbakır, Turkey. [2]Department of Internal Medicine, Balikesir University Medical School, Altieylül, 10145 Balikesir, Turkey. [3]The Global NASH Council, Washington, DC, USA. [4]Department of Internal Medicine, Bozyaka Training and Research Hospital, Karabağlar, 35170 İzmir, Turkey. [5]Department of Internal Medicine, Kayseri City Hospital, Kocasinan, 38080 Kayseri, Turkey. [6]Department of Internal Medicine, Faculty of Medicine, Firat University, 23119 Elazığ, Turkey. [7]Department of Internal Medicine, Marmara University Pendik Training and Research Hospital, Pendik, 34899 İstanbul, Turkey. [8]Department of Internal Medicine, Antalya City Hospital, Kepez, 07080 Antalya, Turkey. [9]Department of Internal Medicine, Muğla Sıtkı Koçman University, Faculty of Medicine, 48000 Muğla, Turkey. [10]Department of Internal Medicine, Giresun University, Faculty of Medicine, 28200 Giresun, Turkey. [11]Department of Internal Medicine, Bolu Abant Izzet Baysal University Hospital, Golkoy, 14280 Bolu, Turkey. [12]Department of Internal Medicine, Eskisehir Osmangazi University, Faculty of Medicine, Odunpazarı, 26040 Eskisehir, Turkey. [13]Department of Internal Medicine, University of Health Sciences, Ankara Etlik City Training & Research Hospital, Yenimahalle, 06170 Ankara, Turkey. [14]Department of Internal Medicine, Ankara Bilkent City Hospital, 06800Çankaya, Ankara, Turkey. [15]Department of Internal Medicine, Aydın Adnan Menderes University Faculty of Medicine, 09100 Efeler, Aydın, Turkey. [16]Department of Internal Medicine, Antalya Training and Research Hospital, 07100Muratpaşa, Antalya, Turkey. [17]Department of Internal Medicine, University of Health Sciences - Adana Health Practice and Research Center, Yüreğir, 01230 Adana, Turkey. [18]Department of Internal Medicine, Bursa City Hospital, 16250 Bursa, Turkey. [19]Department of Internal Medicine, Kütahya Health Sciences University, 43020 Kütahya, Turkey. [20]Department of Internal Medicine, Izmir City Hospital, Bayraklı, 35540 İzmir, Turkey. [21]Department of Internal Medicine, Çorlu Vatan Hospital, Çorlu, 59850 Tekirdag, Turkey. [22]Department of Internal Medicine, Dicle University Medical School, Sur, 21010 Diyarbakir, Turkey. [23]Department of Internal Medicine, Seyhan Hospital, Seyhan, 01150 Adana, Turkey. [24]Department of Internal Medicine, Manisa City Hospital, 45040 Manisa, Turkey. [25]Department of Internal Medicine, Umraniye Training and Research Hospital, Umraniye, 34764 Istanbul, Turkey. [26]Department of Internal Medicine, Istanbul Medipol University Faculty of Medicine, Bagcilar, 34214 Istanbul, Turkey. [27]Department of Internal Medicine, Health Sciences University, Erzurum Faculty of Medicine, Yakutiye, 25240 Erzurum, Turkey. [28]Department of Internal Medicine, Recep Tayyip Erdogan University Faculty of Medicine, 53020 Rize, Turkey. [29]Department of Internal Medicine, University of Health Sciences, Bursa Yüksek İhtisas Training and Research Hospital, Yildirim, 16310 Bursa, Turkey. [30]Department of Internal Medicine, Faculty of Medicine, Sakarya University, Adapazarı, 54290 Sakarya, Turkey. [31]Department of Internal Medicine, Samsun University Medical School, Canik, 55080 Samsun, Turkey. [32]Department of Internal Medicine, Ahi Evran University Medical School, 40200 Kirsehir, Turkey. [33]Department of Internal Medicine, Van Yüzüncü Yıl University, Faculty of Medicine, Tuşba, 65090 Van, Turkey. [34]Department of Internal Medicine, University of Health Sciences, Fatih Sultan Mehmet Education and Research Hospital, , Ataşehir, 34752 Istanbul, Turkey. [35]Department of Internal Medicine, 5 Ocak State Hospital, Seyhan, 01210 Adana, Turkey. [36]Department of Internal Medicine, Çanakkale Onsekiz Mart University School of Medicine, 17020 Canakkale, Turkey. [37]Department of Internal Medicine, Biruni University Vocational School, Topkapı, 34020 İstanbul, Turkey. [38]Department of Internal Medicine, Siirt University Medical School, 56100 Siirt, Turkey. [39]Department of Internal Medicine, Faculty of Medicine, Biruni University, Gültepe, 34295 İstanbul, Turkey. [40]Department of Internal Medicine, Ordu University Training and Research Hospital, Altinordu, 52200 Ordu, Turkey. [41]Department of Internal Medicine, Malatya Training and Research Hospital, Yesilyurt, 44300 Malatya, Turkey. [42]Department of Internal Medicine, Kocaeli City Hospital, İzmit, 41100 Kocaeli, Turkey. [43] Department of Internal Medicine, Faculty of Medicine, Bursa Uludag University, Nilufer, 16059 Bursa, Turkey. [44]Department of Internal Medicine, EBYU Medical School, 24100 Erzincan, Turkey. [45]Department of Internal Medicine, University of Health Sciences, Bagcilar Training and Research Hospital, 34200Bagcılar, İstanbul, Turkey. [46]Department of Internal Medicine, Akdeniz University Medical School, Konyaaltı, 07100 Antalya, Turkey. [47]Department of Internal Medicine, Gaziosmanpasa University Faculty of Medicine, Merkez, 60100 Tokat, Turkey. [48]Department of Internal Medicine, Dokuz Eylul University Faculty of Medicine, Balçova, 35330 İzmir, Turkey. [49]Department of Internal Medicine, Gaziantep University Medical School, Şehitkamil, 27850 Gaziantep, Turkey. ✉email: ihsan2157@gmail.com; kamil.konur@erdogan.edu.tr

Metabolic dysfunction–associated steatotic liver disease (MASLD) is the whole set of pathologies that occur in the absence of significant alcohol consumption and in the presence of metabolic disorders and can progress from simple steatosis in the liver parenchyma to fibrosis and cirrhosis[1–3]. MASLD has emerged as one of the leading causes of liver-related morbidity and mortality on a global scale[4,5]. The pathological process that begins with increased lipid accumulation in hepatocytes at the initial stage may progress to liver cirrhosis and

hepatocellular carcinoma (HCC), with ballooning and fibrosis occurring over time[6,7]. The presence of MASLD is closely associated with type 2 diabetes mellitus (T2DM), obesity, and other cardiometabolic risk factors[8,9]. The prevalence and incidence of MASLD are increasing in parallel with the increasing burdens of obesity, T2DM, and metabolic syndrome (MetS)[10].

The monocyte-to-HDL cholesterol ratio (MHR) is an emerging inflammatory biomarker that has gained importance in recent years, reflecting systemic inflammation and oxidative stress levels[11]. An elevated MHR has been associated with various metabolic disorders, primarily cardiovascular diseases, indicating the prominence of inflammatory activity and oxidative stress[12]. Recent studies have reported that the MHR is also significantly associated with conditions such as insulin resistance, obesity, type 2 diabetes, and MASLD[13]. Therefore, the MHR is considered a potential biomarker that could be used in the diagnosis and risk assessment of inflammatory and metabolic-based diseases such as MASLD.

The visceral adiposity index (VAI) is a parameter calculated via sex-specific formulas that indirectly reflects visceral fat distribution and the metabolic activity of this adipose tissue in individuals[14]. Studies have reported that the VAI is strongly correlated with insulin resistance, type 2 diabetes, metabolic syndrome, cardiovascular diseases, and particularly MASLD[15]. Developed by Amato et al.[16], this index provides a practical clinical tool for assessing metabolically active visceral fat accumulation, which is difficult to define via classical anthropometric measurements. Owing to these features, the VAI is considered a useful biomarker for determining both metabolic risk and the likelihood of liver steatosis in MASLD screening.

The atherogenic index of plasma (AIP) is one of the lipid indices defined in recent years and is a parameter that provides information about the atherogenic lipoprotein profile of individuals. PAI is considered an indirect indicator of small and dense LDL particles in plasma and is closely associated with increased cardiovascular risk[17]. Since the PAI reflects types of dyslipidemia that contribute to atherosclerotic processes, it is suggested that it may be associated not only with cardiovascular diseases but also with metabolic disorders such as MASLD. In the literature, the PAI has been reported to correlate with pathophysiological processes closely associated with MASLD, such as insulin resistance, obesity, and type 2 diabetes[18,19]. Therefore, the PAI can be considered a potential biomarker in the diagnostic evaluation process of MASLD.

The cardiometabolic index (CMI) is a composite parameter developed to assess cardiometabolic risk due to visceral adiposity and insulin resistance in individuals[20]. This index considers both abdominal obesity and dyslipidemia simultaneously and has been shown to be strongly associated with various metabolic disorders, including metabolic syndrome, type 2 diabetes, hypertension, and cardiovascular diseases[21]. Recent studies have demonstrated that CMI is also significantly associated with hepatic steatosis and MASLD. In particular, CMI may be a practical and effective biomarker in MASLD screening since it simultaneously assesses visceral fat accumulation and lipid irregularities directly associated with liver steatosis[22].

To the best of our knowledge, no clinical study in Turkey has investigated the relationships between lipid indices and hepatosteatosis and fibrosis in MASLD patients on a nationwide scale. The aim of this study was to investigate the roles of the MHR, VAI, PAI, and CMI indices in determining hepatic inflammation and fibrosis in MASLD patients on a nationwide scale given their noninvasive nature, ease of use, and cost-effectiveness.

Materials and methods

Study design and population

This nationwide study is a retrospective, cross-sectional analysis conducted by reviewing a cohort of individuals with at least one cardiometabolic risk factor across 44 internal medicine outpatient clinics in 31 provinces representing all the statistical regions of Turkey. Clinical centers were included according to the 12 Nomenclature of Territorial Units for.

Statistics (NUTS) for regions of the country[23]. The study was approved by the Local Ethics Committee of the University of Health Sciences Gazi Yaşargil Training and Research Hospital (approval date: 13/06/2025, approval number: 511) and conducted in accordance with the Declaration of Helsinki.

Individuals aged 18 years and older with at least one cardiometabolic risk factor were consecutively enrolled in the study. The exclusion criteria included pregnancy, acute inflammation, malignancy, a history of bariatric surgery, and specific conditions that may cause chronic liver disease (such as high alcohol intake, viral hepatitis, and storage diseases).

Hepatosteatosis was assessed via liver ultrasonography and defined as increased echogenicity in the liver parenchyma compared with the renal cortex. Ultrasonographic examinations were performed by experienced radiologists at each participating center according to routine clinical protocols.

Anthropometric measurements

A medical history was obtained from all participants, and all of them underwent physical examination. Body mass index (BMI) was calculated via the weight/height^2 formula. Waist circumference was measured at the midpoint between the anterior superior iliac crest and the lowest rib. Blood pressure was measured three times while the participants were in the seated position, and the median value was recorded.

Clinical and biochemical analyses

Blood samples were collected from the participants after at least 8 h of fasting, and complete blood count and biochemical analyses were performed via standardized devices. Fasting plasma glucose (FPG), creatinine, alanine aminotransferase (ALT), aspartate aminotransferase (AST), total cholesterol (TC), triglyceride (TG) and high-density lipoprotein cholesterol (HDL-C) values were recorded. Low-density lipoprotein cholesterol (LDL-C) was calculated via the Friedewald formula: LDL-C = TC - (HDL-C + TG/5)[24].

MHR=Monocyte count (10^3/μL)/HDL cholesterol (mg/dL).

VAI (Men) = (Waist circumference (cm)/(39.68 + (1.88 × BMI)) × (Triglyceride (mmol/L)/1.03) × (1.31/HDL (mmol/L)).

VAI (Women) = (Waist circumference (cm)/(36.58 + (1.89 × BMI)) × (Triglyceride (mmol/L)/0.81) × (1.52/HDL (mmol/L)).

PAI = log x (Triglyceride (mg/dL)/HDL cholesterol (mg/dL)).

CMI = waist circumference/height ratio × triglyceride/HDL-C (WHtR × TG/HDL-C).

The FIB-4 score was calculated using the formula: FIB-4 = (Age × AST) / (Platelet count × √ALT).

Identification of metabolic disorders

MASLD was defined as steatotic liver disease (SLD) in the presence of at least one cardiometabolic risk factor, according to the EASL-EASD-EASO clinical practice guidelines[9].

BMI ≥ 25 kg/m² or waist circumference ≥ 94 cm (men), ≥ 80 cm (women).

FPG ≥ 100 mg/dL or 2-hour OGTT ≥ 140 mg/dL or HbA1c ≥ 5.7% or receiving diabetes treatment.

Blood pressure ≥ 130/85 mmHg or use of antihypertensive medication.

TG ≥ 150 mg/dL or lipid-lowering therapy.

HDL-C ≤ 40 mg/dL (men), ≤ 50 mg/dL (women) or lipid-lowering therapy[9].

Type 2 diabetes was defined as FPG ≥ 126 mg/dL, 2-hour OGTT ≥ 200 mg/dL, HbA1c ≥ 6.5%, or the use of antidiabetic medication[25].

Hypertension was defined as a home blood pressure > 135/85 mmHg, hospital blood pressure > 140/90 mmHg, or the use of antihypertensive drug medication[26].

Dyslipidemia was defined as TG > 150 mg/dL and/or LDL-C > 100 mg/dL and/or low HDL-C (< 40 mg/dL in men, < 50 mg/dL in women) or the use of lipid-lowering therapy[27].

Metabolic syndrome (MetS) was as the presence of at least three of the following five criteria (according to the NCEP ATP III criteria):

1. Abdominal obesity (waist circumference): >102 cm in men, > 88 cm in women.
2. Elevated TG: ≥150 mg/dL.
3. Low HDL-C: <40 mg/dL in men, < 50 mg/dL in women.
4. Elevated blood pressure: ≥130/85 mmHg or use of antihypertensive medication.
5. Elevated fasting glucose: ≥100 mg/dL or the use of antidiabetic medication[28].

Cardiovascular disease (CVD) was defined as the presence of a history of angina, myocardial infarction, heart failure, or peripheral vascular disease[29].

Assessment of advanced hepatic fibrosis risk

Advanced hepatic fibrosis risk was evaluated using the fibrosis-4 (FIB-4) score, which is calculated based on age, aspartate aminotransferase (AST), alanine aminotransferase (ALT), and platelet count. Patients were categorized as low risk (FIB-4 < 1.3; <2.0 if > 65 years) and high risk (FIB-4 ≥ 1.3; ≥2.0 if > 65 years), according to the EASL-EASD-EASO clinical practice guidelines and previously validated cut-off values (FIB-4 < 1.3; <2.0 if > 65 years; ≥1.3; ≥2.0 if > 65 years)[9].

Statistics

The data were analyzed using IBM SPSS Statistics for Windows, version 25.0 (IBM Corp., Armonk, NY, USA). Numerical data are expressed as medians (interquartile range, 25th–75th percentile), and categorical variables are expressed as numbers (n) and percentages (%). The normality of the distribution was assessed via the Kolmogorov–Smirnov test. Differences between groups were compared via the chi-square test for categorical variables and the Mann–Whitney U test for continuous variables. Spearman's rho correlation test was applied between measurements. A two-tailed p value of ≤ 0.05 was considered statistically significant. Comparative analyses were performed between MASLD (+) and MASLD (–) groups, as well as between low- and high-risk advanced fibrosis groups, with respect to demographic characteristics and lipid indices (MHR, VAI, PAI, and CMI). Multivariable logistic regression analyses were conducted using the enter method. Collinearity diagnostics were evaluated prior to model estimation.

Results

A total of 14,322 patients from 44 centers were included in our study. The overall study flow and fibrosis stratification process are presented in Fig. 1.

Among all patients, 5,527 (38.59%) were men, and 8,795 (61.4%) were women. Diabetes mellitus (DM) occurred in 6,163 patients, and hypertension (HT) occurred in 6,089 patients. While 1,486 patients had a BMI < 25, 12,836 patients had a BMI > 25.

Among them, 10,836 (75.7%) were classified as MASLD (+) and 3,486 (24.3%) as MASLD (–). Compared with individuals without MASLD, those with MASLD had significantly higher VAI, PAI, and CMI values ($p < 0.001$ for all), whereas MHR and age did not differ significantly between the groups ($p = 0.453$ and $p > 0.05$, respectively). In addition, significant differences were observed in sex, diabetes mellitus, hypertension, height, weight, and BMI between the groups. Detailed demographic, clinical, and index-related data are presented in Table 1.

To assess whether these associations were independent, multivariable logistic regression analyses were performed. After adjustment for age, sex, diabetes mellitus, hypertension, and BMI (where appropriate), VAI (OR 1.025; 95% CI 1.018–1.033; $p < 0.001$), PAI (OR 2.38; 95% CI 2.06–2.75; $p < 0.001$), and CMI (OR 1.14; 95% CI 1.11–1.17; $p < 0.001$) remained independently associated with MASLD. When MHR was rescaled (multiplied

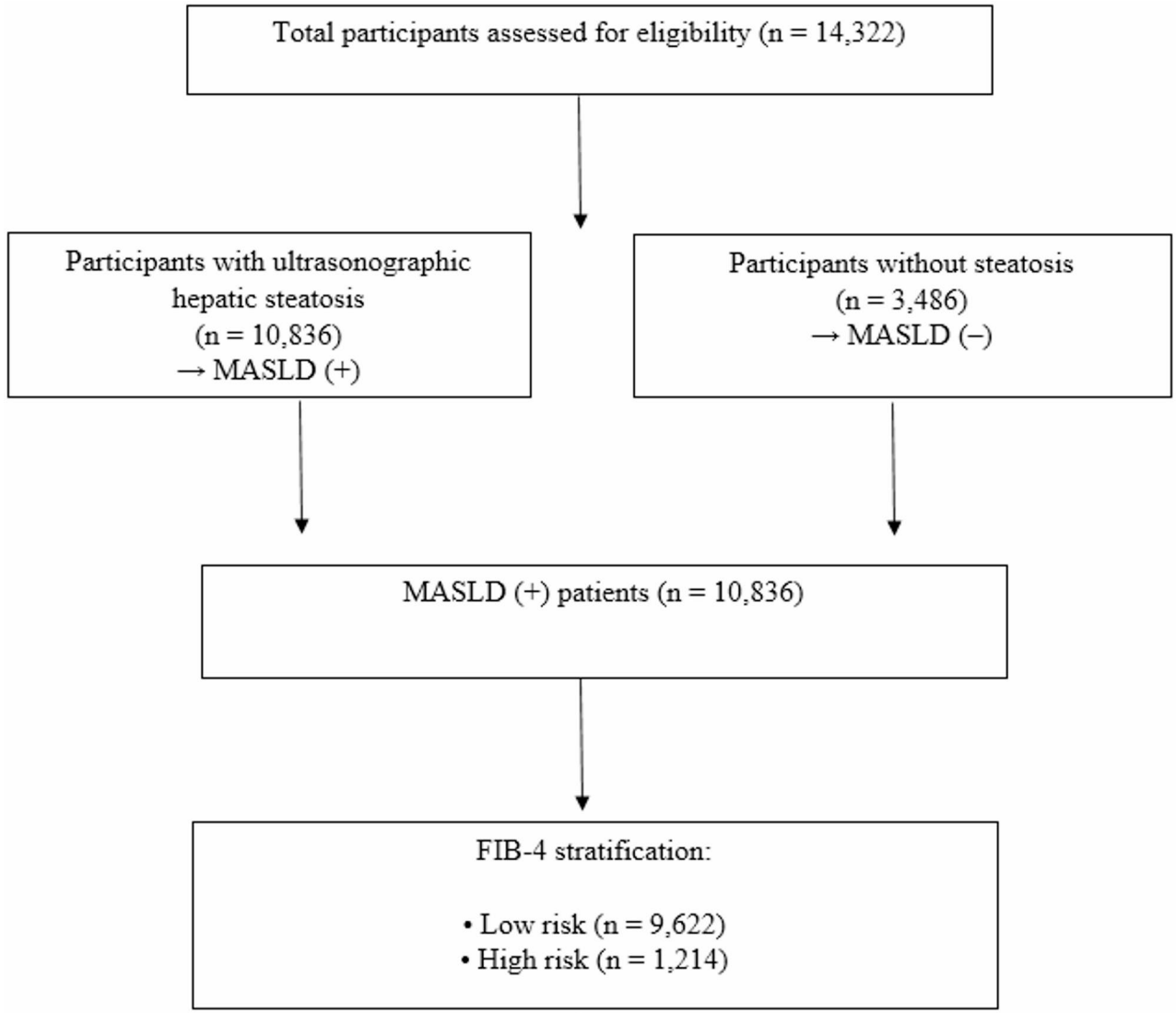

Fig. 1. Flow diagram of study participants, MASLD classification based on ultrasonography, and FIB-4–based fibrosis risk stratification.

by 100) for interpretability, it showed a modest inverse association with MASLD (OR 0.95; 95% CI 0.93–0.97; $p < 0.001$). Detailed regression results are provided in Supplementary Table 1.

Comparison of indices according to risk of advanced hepatic fibrosis (FIB-4 Based)

Among patients with MASLD, 1,214 (11.2%) were classified as high risk of advanced hepatic fibrosis based on FIB-4, while 9,622 (88.8%) were classified as low risk. Significant differences were observed between the groups in terms of age, sex, diabetes mellitus, hypertension, and BMI. However, MHR, VAI, PAI, and CMI values did not differ significantly between low- and high-risk groups. Detailed findings are presented in Table 2. These findings suggest that lipid-derived indices differentiate hepatic steatosis status rather than fibrosis severity.

To further evaluate whether lipid-derived indices were independently associated with advanced hepatic fibrosis risk, multivariable logistic regression analysis was performed among MASLD-positive individuals. The model was adjusted for sex, body mass index (BMI), diabetes mellitus, and hypertension. Age was not included in the model because it constitutes a direct component of the FIB-4 formula.

None of the investigated lipid-derived indices (MHR, VAI, PAI, or CMI) were independently associated with high FIB-4 risk after adjustment for these covariates. However, diabetes mellitus and hypertension remained significantly associated with high fibrosis risk. Detailed regression results are presented in Supplementary Table 2.

A correlation test was performed with the FIB-4, MHR, VAI, PAI, and CMI indices measured in MASLD + patients. Spearman correlation analysis revealed a statistically significant but clinically negligible weak negative correlation between FIB-4 score and MHR ($r = -0.030$, $p = 0.002$). Given the extremely low correlation coefficient, this association is unlikely to have meaningful clinical relevance (Table 3).

Variables	MASLD (+) $n = 10,836(75.66\%)$	MASLD (-) $n = 3486\ (24.34\%)$	Total population $n = 14,322$	*p* values
Demographic parameters				
Age (year)*	52(42–61)	52(39–64)	52 (42–62)	0.418
Sex (women)**	6482(59.81%)	2313(66.35%)	8795(61.4%)	< 0.001
BMI > 25 (kg/m^2) *	10,065(92.88%)	2771(79.48%)	12,836(89.6%)	< 0.001
Comorbid diseases				
T2DM**	4907(45.4%)	1256(36.02%)	6163(43.03%)	< 0.001
Hypertension**	4756(43.89%)	1333(38.23)	6089(42.51%)	< 0.001
Laboratory and indexes				
MHR	0.01 (0.01–0.02)	0.01 (0.01–0.02)	0.01 (0.01–0.02)	0.453
VAI	5.61 (3.62–9.07)	4.77 (3.05–7.71)	5.42 (3.45–8.76)	< 0.001
PAI	0.50 (0.32–0.70)	0.40 (0.23–0.60)	0.48 (0.29–0.68)	< 0.001
CMI	1.99 (1.27–3.16)	1.47 (0.95–2.38)	1.86 (1.18–2.98)	< 0.001

Table 1. Demographic and metabolic characteristics of the study population. Continuous variables are presented as median (interquartile range, 25th–75th percentile), and categorical variables as n (%). BMI, body mass index; T2DM, type 2 diabetes mellitus; MASLD, metabolic dysfunction-associated steatotic liver disease; MHR, monocyte/HDL cholesterol ratio; VAI, visceral adiposity index; PAI, plasma atherogenic index; CMI, cardiometabolic index. Statistical comparisons were performed using the chi-square test for categorical variables and the Mann–Whitney U test for continuous variables.

Variables	Low FIB-4 score $n = 9622\ (88.8\%)$	High FIB-4 score $n = 1214\ (11.2\%)$	Total population $n = 10,836$	*p* values
Demographic parameters				
Age (year)*	51(41–60)	59(53–64)	52(42–61)	< **0.001**
Sex (women)**	5806 (60.34%)	676 (55.68%)	6482 (59.81%)	**0.001**
BMI > 25 (kg/m^2) *	8956 (93.07%)	1109 (91.35%)	10,065 (92.88%)	**0.020**
Comorbid diseases				
T2DM**	4280 (44.48%)	624 (51.4%)	4907 (45.28%)	< **0.001**
Hypertension**	4108 (42.69%)	647 (53.29%)	4755 (43.88%)	< **0.001**
Laboratory and Indexes				
MHR	0.01 (0.01–0.02)	0.01 (0.01–0.02)	0.01 (0.01–0.02)	0.705
VAI	5.61 (3.63–9.07)	5.60 (3.55–9.00)	5.61 (3.62–9.07)	**0.530**
PAI	0.50 (0.32–0.70)	0.51 (0.31–0.70)	0.50 (0.32–0.70)	**0.963**
CMI	1.99 (1.28–3.16)	1.97 (1.24–3.19)	1.99 (1.27–3.16)	0.719
FIB-4 score	0.75 (0.55–0.99)	1.69 (1.44–2.28)	0.81 (0.58–1.12)	< **0.001**

Table 2. Demographic and metabolic parameters of the study population according to FIB-4 score. Continuous variables are presented as median (interquartile range, 25th–75th percentile), and categorical data as n (%). BMI, body mass index; T2DM, type 2 diabetes mellitus; MHR, monocyte/HDL cholesterol ratio; VAI, visceral adiposity index; PAI, plasma atherogenic index; CMI, cardiometabolic index; FIB-4, fibrosis-4; Statistical comparisons were performed using the chi-square test for categorical variables and the Mann–Whitney U test for continuous variables.

Variable	*r*	*p*-value
FIB-4	1.000	–
MHR	− 0 0.030	0.002
VAI	− 0.008	0.405
PAI	− 0.008	0.431
CMI	− 0.009	0.365

Table 3. Correlation of indices with the FIB-4 score.

MHR, monocyte/HDL cholesterol ratio; VAI, visceral adiposity index; PAI, plasma atherogenic index; CMI, cardiometabolic index; FIB-4, fibrosis-4; **. The correlation is significant at the 0.01 level (2-tailed). Correlation analysis was performed using Spearman's rho test.

Discussion

This clinical study is a multicenter investigation conducted across all 12 statistical regions of Turkey, with high national representativeness, and the primary aim was to examine the role of both traditional and previously described lipid indices (MHR, VAI, PAI, CMI) in MASLD patients and their relationship with the FIB-4 score, a noninvasive marker of liver fibrosis. Within the scope of the study, it was observed that while the VAI, which has been investigated in the literature for many years, was associated with hepatosteatosis, it was not correlated with fibrosis. Similarly, in MASLD patients, the PAI and CMI were associated with hepatostetosis but not with fibrosis. On the other hand, the MHR was not associated with either hepatosteatosis or fibrosis. In addition, no direct link was identified between FIB-4 elevation and lipid indices in patients with MASLD.

MASLD and VAI

Today, various scores and biomarkers have been investigated to avoid liver biopsy, an invasive procedure that has become the gold standard for evaluating MASLD and liver fibrosis. Although the VAI is one of the leading lipid indices investigated in this sense, heterogeneity has been observed in clinical studies in the literature. In a clinical study by Roslyn Vongsuvanh et al., which evaluated 190 adults with biopsy-proven NAFLD and 129 control patients, no significant relationship was found between the VAI and liver histology[30]. On the other hand, in another clinical study by Mirac Vural Keskinler et al., which included 57 biopsy-proven NAFLD patients and 57 healthy controls, VAI levels were significantly greater in NAFLD patients[31]. In addition, in a meta-analysis evaluating 24 clinical studies investigating the role of the VAI in NAFLD patients, the VAI had predictive value in diagnosing NAFLD and NASH in adult NAFLD patients with significantly greater values in severe steatosis than in simple steatosis patients and controls[32]. In light of all this information, the VAI was significantly greater in the MASLD group than in the other groups in the present clinical study. These results indicate that the VAI could be a valuable biomarker for MASLD risk assessment. It should be acknowledged that MASLD was defined in the presence of at least one cardiometabolic risk factor, some of which overlap with the components of the investigated lipid-derived indices. Therefore, the observed associations may partly reflect shared metabolic characteristics between exposure and outcome rather than entirely independent pathophysiological mechanisms. Accordingly, these findings should be interpreted as associative rather than demonstrating independent predictive value. Abdominal fat, especially in women, has been reported to predict the risk of MASLD more strongly, which is also consistent with the greater proportion of women patients in our study. On the other hand, the VAI was not found to be associated with the degree of fibrosis, and new clinical studies are needed in this field.

MASLD and PAI

PAI is calculated by logarithmic transformation of the triglyceride/HDL ratio and provides information about the atherogenic lipoprotein profile. Clinical studies in the literature have emphasized that it may be associated with metabolic syndrome and cardiovascular diseases[33]. However, few studies have investigated the role of the PAI in individuals with MASLD. The role of PAI in MASLD pathogenesis in these studies is unclear. In a clinical study by Carlo De Matteis et al., a significant increase in PAI was observed in patients with liver steatosis compared with healthy controls[34]. On the other hand, in another study by Kirik et al., involving 129 individuals with biopsy-proven MAFLD, the PAI was found to be unrelated to steatohepatitis and fibrosis[35]. In our study, we found that the PAI was significantly greater in patients with MASLD. This result supports the close relationship between MASLD and dyslipidemia and suggests that the PAI may be an important marker not only for cardiovascular risk but also for hepatic steatosis. On the other hand, the PAI is not associated with the degree of fibrosis, and new studies are needed in this field.

MASLD and CMI

The CMI is a recently investigated parameter calculated by combining the waist/height ratio and triglyceride/HDL ratio to assess both obesity and dyslipidemia simultaneously. In the literature, CMI is a strong predictor of metabolic syndrome and type 2 diabetes and can be used in the diagnosis of NAFLD[21,22]. However, few studies have investigated the role of CMI in individuals with NAFLD. In a clinical study by Wen-feng Xi et al. involving 1,759 patients, CMI was associated with hepatosteatosis in individuals with MASLD[36]. In addition, another cohort study revealed that CMI was associated with MASLD incidence and progression to fibrosis, especially in individuals with high waist circumference and high triglyceride levels[22]. On the other hand, in our study, although the CMI was associated with hepatosteatosis, it was not associated with the degree of fibrosis. This result suggests that the CMI may be a noninvasive and practical tool in MASLD screening; however, it may not be effective in predicting progression to fibrosis in individuals with MASLD.

MASLD and MHR

The MHR is an inflammatory biomarker reflecting systemic inflammation and oxidative stress. Its association with cardiovascular diseases and metabolic syndrome has been strongly demonstrated in the literature[12,13]. A clinical study conducted by Jun-feng Zhang et al. involving 504 children with childhood obesity demonstrated that the MHR may be a current and useful indicator of NAFLD in these individuals[37]. In addition, in another study by Liring Wang et al., which included 1,703 NAFLD patients, the MHR was associated with hepatosteatosis but not with advanced liver fibrosis[38]. In light of all this information, no significant difference was found between MASLD and MHR in our study. The MHR was not associated with either hepatosteatosis or fibrosis, which contrasts with the current literature. This observation may reflect the possibility that MHR primarily captures

systemic inflammatory burden rather than lipid-driven metabolic dysfunction; however, this interpretation should be considered hypothesis-generating and requires confirmation in prospective studies. In addition, the fact that the MHR better reflects acute inflammatory responses may cause it to have limited sensitivity in MASLD, which progresses chronically and silently.

FIB-4 and indices

The FIB-4 score, which is calculated from age, AST, ALT and platelet values, is a widely used noninvasive fibrosis marker in clinical practice. In recent studies, the sensitivity of FIB-4 in the detection of MASLD patients with advanced fibrosis has been limited, especially compared with that of imaging methods such as transient elastography (FibroScan) and magnetic resonance elastography (MRE); however, FIB-4 is considered valuable because of its usability for screening purposes and low cost [39]. It has also been reported that fibrosis is not only a liver-specific problem but also a barometer of systemic metabolic status[9]. In our study, the frequency of age, diabetes, and hypertension was significantly greater in MASLD patients with high FIB-4 scores. In contrast, no significant relationships were found between the VAI, the PAI, the CMI, or the MHR and FIB-4. In the literature, genetic factors as well as metabolic risk factors (e.g., PNPLA3 and TM6SF2 polymorphisms), the intestinal microbiota, and environmental factors have also been reported to be involved in fibrosis progression[7]. Therefore, it is expected that lipid indices may not directly reflect the degree of fibrosis. Furthermore, it should be emphasized that FIB-4 is strongly age-dependent, as age constitutes one of its core components. Therefore, differences observed between fibrosis risk categories may partially reflect age distribution rather than true biological differences in fibrosis severity, which should be carefully considered when interpreting these findings.

From a clinical perspective, VAI, PAI, and CMI may serve as practical, cost-effective, and easily accessible tools for identifying individuals at increased risk of MASLD, particularly in primary care or resource-limited settings. Given their reliance on routinely available anthropometric and biochemical parameters, these indices may support early risk stratification in patients with cardiometabolic risk factors. However, considering their lack of association with FIB-4–based advanced fibrosis risk in our study, these indices should not replace established noninvasive fibrosis assessment tools such as FIB-4, transient elastography, or ELF score. Future prospective studies and external validation across diverse populations are needed before integrating these indices into standardized clinical algorithms.

Although lipid-derived indices reflect metabolic burden and visceral adiposity, hepatic fibrosis progression involves complex and multifactorial mechanisms including genetic predisposition, inflammatory signaling pathways, mitochondrial dysfunction, oxidative stress, and gut–liver axis alterations. Therefore, the lack of association between lipid indices and FIB-4 in our cohort may indicate that fibrosis progression in MASLD is not solely driven by dyslipidemia but rather by a broader interplay of metabolic, inflammatory, and genetic factors.

The significant differences observed in sex distribution and BMI between MASLD-positive and MASLD-negative groups likely reflect the strong relationship between visceral adiposity and hepatic steatosis. Because some metabolic components overlap with the calculation of certain lipid-derived indices, partial interaction between exposure and outcome definitions should be acknowledged. Therefore, these associations should be interpreted within the broader metabolic context rather than as purely independent effects.

Strengths and limitations of the study

The strengths of our study include the inclusion of 44 centers from different regions of Turkey and more than 14,000 patients and enhancing the generalizability of the results. In addition, the most important contribution of this study to the current literature is that novel lipid-derived indices were significantly associated with the presence of MASLD. In particular, the VAI, PAI, and CMI offer advantages in large-scale population screenings because they are easily calculable and inexpensive in clinical practice. In countries with a high prevalence of obesity and metabolic syndrome, such as Turkey, integrating these indices into MASLD screening in primary healthcare could be a cost-effective approach.

However, there are also limitations. Owing to the retrospective design, causal relationships cannot be established. Hepatic steatosis was assessed via ultrasonography without histopathological confirmation. Lifestyle factors (diet, physical activity, alcohol consumption) and genetic variations were not included in the analysis. Additionally, lipid-lowering therapy may have influenced triglyceride and HDL-C levels, thereby directly affecting the calculated lipid-derived indices (VAI, PAI, and CMI). Although dyslipidemia and lipid-lowering therapy were recorded, specific adjustment for different classes of lipid-modifying agents was not performed. Therefore, residual confounding related to pharmacological treatment cannot be fully excluded. Furthermore, the FIB-4 score is a noninvasive parameter and may not reflect advanced fibrosis with full accuracy. Future prospective, biopsy-confirmed studies will provide more precise results on the role of lipid indices in MASLD diagnosis. In addition, combining these indices with other noninvasive tests (e.g., the NFS, ELF score, and transient elastography) could improve diagnostic accuracy. The integration of these indices into AI-based risk models could offer innovative approaches to MASLD screening and prognosis prediction.

The high prevalence of MASLD (75.7%) observed in our study should be interpreted in the context of the study design. Participants were consecutively recruited from internal medicine outpatient clinics and all had at least one cardiometabolic risk factor. Therefore, this cohort does not represent the general population but rather a metabolically high-risk clinical population. This selection framework likely explains the markedly higher MASLD prevalence compared with population-based epidemiological studies.

Moreover, although ultrasonography is widely used in routine clinical practice, its sensitivity decreases in mild steatosis and in individuals with high BMI, which may have influenced steatosis detection rates in our study.

Conclusion

This large nationwide multicenter study demonstrated that VAI, PAI, and CMI are significantly associated with the presence of MASLD in a metabolically high-risk Turkish population, whereas MHR showed no significant relationship. None of the investigated lipid indices were independently associated with FIB-4–based advanced fibrosis risk, and the minimal correlations observed are unlikely to be clinically meaningful. These results should be interpreted within the limitations of noninvasive fibrosis assessment. These findings suggest that lipid-derived indices primarily reflect metabolic steatosis burden rather than fibrosis severity; however, this interpretation should be considered cautiously given the age-dependent nature of FIB-4.

These findings support the potential role of easily accessible lipid-derived indices as practical screening tools for identifying individuals at risk of hepatic steatosis. However, they should not replace validated fibrosis assessment tools in clinical decision-making. Future prospective studies incorporating imaging-based fibrosis assessment and molecular markers are warranted to better define their role in MASLD risk stratification.

Given the high prevalence of obesity and metabolic syndrome in Turkey, this nationwide cohort provides a valuable population-specific perspective on the behavior of lipid-derived indices in MASLD. Genetic background, lifestyle patterns, and cardiometabolic risk clustering may differ from Western or East Asian populations, underscoring the importance of regional validation studies.

Data availability

The data supporting the findings of this study are available from the corresponding author upon reasonable request.Due to ethical and legal restrictions related to patient confidentiality, the dataset is not publicly available.

Received: 9 December 2025; Accepted: 23 March 2026
Published online: 19 April 2026

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Acknowledgements

The authors would like to thank all participating internal medicine clinics across 31 provinces in Turkey for their valuable contributions to data collection.

All authors reviewed and approved the final manuscript.

Author contributions

İhsan Solmaz (İ.S), Ali Kırık (A.K), İsmail Demir (İ.D), Oğuzhan Sıtkı Dizdar (O.S.D), Nevzat Gözel (N.G), Gökhan Tazegül (G.T), Bilgin Bahadır Başgöz (B.B.B), Cem Şahin (Ç.Ş), Kubilay İşsever (K.İ), Gülali Aktaş (G.A), Pınar Yıldız (P.Y), Emin Gemcioğlu (E.G), Enes Seyda Şahiner (E.Ş.Ş), Hilal Bektaş Uysal (H.B.U), Yasin Şahintürk (Y.Ş), Ömer Faruk Alakuş (Ö.F.A), Mustafa Akdeniz (M.A), Hilmi Erdem Sümbül (H.E.S), Nizameddin Koca (N.K), Türkan Paşalı Kilit (T.P.K), Sibel Demiral Sezer (S.D.S), Emine Binnetoğlu (E.B), Eşref Araç (E.A), Hacer Şen (H.Ş), Bahri Abaylı (B.A), Hale Akan (H.A), Canan Akkuş (C.A), Berrin Aksakal (B.Ak), Burçin Meryem Atak Tel (B.M.A.T), Ahmet Aydın (A.A), Sami Bahçebaşı (S.B), Orhan Balıkçı (O.B), Lale Saka Baraz (L.S.B), Muharrem Bayrak (M.B), Hatice Beyazal Polat (H.B.P), İfakat İrem Biçer (İ.İ.B), Rıfat Bozkuş (R.B), Fatih Coşkun (F.C), Cüneyt Çağatay (C.Ç), Feride Çağlar (F.Ç), Erkan Çakmak (E.Ç), Deniz Cekiç (D.C), Ülfet Değer (Ü.D), Ayşe Kevser Demir (A.K.D), Erkan Dulkadiroğlu (E.D), Nur Düzen Oflas (N.D.O), Betül Erişmiş (B.E), Ali Erol (A.E), Ayşegül Ertınmaz (A.Ert), Müzeyyen Eryılmaz (M.E), Ahmed Bilal Genç (A.B.G), Melis Gökgöz (M.G), Fatih İleri (F.İ), Uğur Can İzlimek (U.Ç.İ), Özge Kama Başcı (Ö.K.B), Aynur Kamburoğlu (A.Kam), Fatih Kamış (F.K), Sanem Kayhan (S.K), İsmail Kırlı (İ.K), Yusuf Kimyon (Y.K), Şeref Enes Kocaman (Ş.E.K), Kamil Konur (K.K), Özge Kurtkulağı (Ö.K), Celalettin Küçük (C.K), Mehmet Selim Mamiş (M.S.M), Hatice Metin (H.M), Necip Nas (N.N), Sibel Ocak Serin (S.O.S), Oktay Olmuşçelik (O.O), Alihan Oral (A.O), Muhammet Özbilen (M.Ö), Erkan Özdemir (E.Ö), Ensar Özmen (E.Öm), Hikmet Öztop (H.Ö), Hüseyin Ali Öztürk (H.A.Ö), Osman Özüdoğru (O.Ö), Emel Sağlam (E.S), Hatice Özge Serin (H.Ö.S), Hasan Sözel (H.S), Melisa Şahin Tekin (M.Ş.T), Ahmet Veli Şanibaş (A.V.Ş), Hakan Şıvgın (H.Şı), Abdullah Tanrıkulu (A.T), Tuba Taslamacıoğlu Duman (T.T.D), Elif Duygu Topan (E.D.T), Hasan Tunca (H.T), Seyit Uyar (S.U), Ece Ünal Çetin (E.Ü.Ç), Nazif Yalçın (N.Y), Demet Yalçın Kehribar (D.Y.K), Selçuk Yaylacı (S.Y), Hasan Esat Yıldırım (H.E.Y), Hüseyin Yıldız (H.Y), Hasan Esat Yücel (H.E.Yü), Oğuzhan Zengin (O.Z), Ali Zeynettin (A.Z), Fatih Atik (F.A), Selin Müge Aslan (S.M.A), Mert Akyıldız (M.Aky), Nurcan Aslan (N.A), Sare Babacan Çelikel (S.B.Ç), Suat Baran Bakan (S.B.B), Merve Durmuş (M.D), Mert Karacay (M.K), Çağatay Koçyiğitoğlu (Ç.K), Gökçe Paşa (G.P), Selvinaz Sivri (S.S), Tutku Naz Şahin (T.N.Ş), Yağmur Sena Tosun (Y.S.T), Zeynep Gizem Totik (Z.G.T), Rabia Ulutaş (R.U), Tuğçe Nur Yazıcı (T.N.Y), Emrah Yılmaz (E.Y), Hamit Yıldız (H.Yı).

Declarations

Competing interests

The authors declare no competing interests.

Informed consent

As anonymized, routinely collected clinical data were used, the requirement for written informed consent was waived.

Additional information

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1038/s41598-026-45869-4.

Correspondence and requests for materials should be addressed to İ.S. or K.K.