

RESEARCH ARTICLE

Evaluation of Subclinical and Clinical Manifestations of Atherosclerosis in Patients with Metabolic Dysfunction-Associated Steatotic Liver Disease

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Abstract

Fatty liver disease (FLD) associated with metabolic dysfunction is one of the most common metabolic pathologies worldwide, and in recent years its close association with cardiovascular disease has been demonstrated. FLD is not only a liver disease, but also a multisystem pathology associated with systemic inflammation, insulin resistance, endothelial dysfunction, and accelerated atherosclerosis.

To evaluate the clinical features of early (increased carotid intima-media thickness, endothelial dysfunction) and late (regional myocardial hypokinesia, coronary artery stenosis) signs of atherosclerosis in patients with CKD.

The study included 63 patients examined at the Shirvan Central City Hospital. Patients were divided into two groups based on carotid intima-media thickness (CIMT): normal CIMT (<0.9 mm; $n=30$) and elevated CIMT (≥ 0.9 mm; $n=33$). The evaluation included anthropometric parameters, lipid profile, echocardiographic parameters, epicardial fat thickness, and the degree of hepatosteatosis based on abdominal ultrasound.

Age was higher in the group with elevated TIMCA, serving as an independent predictor of elevated TIMCA ($OR \approx 1.08$; 95% CI 1.02–1.15; $p < 0.01$). LDL and triglyceride levels were relatively higher in the group with elevated TIMCA. A positive correlation was found between the degree of hepatosteatosis and epicardial fat thickness. Epicardial fat thickness was higher in patients with regional myocardial hypokinesia.

FLD is significantly associated with both early and late symptoms of atherosclerosis. A combined assessment of TIMCA, epicardial fat thickness, and hepatic steatosis may be useful for the early identification of high-risk patients.

Keywords: Fatty liver disease, Atherosclerosis, Carotid intima-media thickness, Epicardial fat, Hepatosteatosis.

Introduction

Metabolic-associated steatotic liver disease (MASLD) is the most common chronic liver disease, closely associated with carbohydrate and lipid metabolism disorders, obesity, insulin resistance, and other components of metabolic syndrome [1–3]. According to modern epidemiological studies, the prevalence of MASLD in the adult population reaches 25–30%, and in certain high-cardiometabolic risk groups, it exceeds 40% [2–5]. In 2023, international expert communities proposed a new nomenclature for the disease, replacing the term “non-alcoholic fatty liver disease (NAFLD)” with “metabolic-associated steatotic liver disease (MASLD).” This change reflects the leading role of metabolic dysfunction in the pathogenesis of the disease and allows for more

accurate identification of patients with high cardiometabolic risk [1,4,6]. Currently, MASLD is considered not only a liver disease but also a systemic pathology accompanied by chronic low-grade inflammation, endothelial dysfunction, oxidative stress, dyslipidemia, and insulin resistance. The combination of these disorders contributes to the accelerated development of atherosclerosis and increases the likelihood of cardiovascular complications [5–9]. Numerous clinical studies and meta-analyses have shown that patients with MASLD are significantly more likely to have subclinical atherosclerosis, coronary artery calcification, increased carotid intima-media thickness, and coronary artery disease. Moreover, cardiovascular disease remains the leading cause of mortality in this category of

patients, significantly outpacing the development of liver complications [7–11]. It should be noted that in recent years, MASLD has increasingly been considered an independent predictor of cardiovascular risk, independent of the presence of traditional risk factors. According to current concepts, pathological processes occurring in the liver have a systemic impact on the vascular wall through chronic inflammation, impaired adipokine secretion, activation of oxidative stress, and altered production of proinflammatory cytokines. This results in chronic endothelial dysfunction, which contributes to the development of subclinical atherosclerosis long before the onset of clinical symptoms of cardiovascular disease [9–12]. In addition to inflammatory mechanisms, insulin resistance plays a significant role, being a central link in the pathogenesis of MASLD. Increased concentrations of free fatty acids, impaired tissue sensitivity to insulin, and increased synthesis of atherogenic lipoproteins contribute to the accumulation of lipids in both the liver and the vascular wall. For this reason, MASLD is considered a systemic disease affecting various organs and systems of the body [13–16]. One of the most informative and accessible methods for the early detection of subclinical atherosclerosis is ultrasound measurement of the carotid intima-media thickness (CIMT). Elevated CIMT is recognized as an independent predictor of myocardial infarction, stroke, and other cardiovascular events [12–15]. In recent years, increasing attention has been paid to epicardial adipose tissue as an active visceral fat depot located directly around the myocardium and coronary arteries. Epicardial adipose tissue synthesizes a wide range of proinflammatory cytokines, adipokines, and oxidative stress mediators, contributing to the development of endothelial dysfunction, vascular inflammation, and the progression of atherosclerosis [16–20]. Despite the significant number of publications devoted to CIMT, a comprehensive assessment of the relationship between the degree of liver steatosis, carotid intima-media thickness, epicardial adipose tissue thickness, and regional myocardial contractility disorders has been insufficiently studied. Clarification of these relationships is important for improving early diagnosis and cardiovascular risk stratification in patients with MASLD [18–22].

In the light of the above mentioned the purpose of the present study was to evaluate the relationship between subclinical and clinical manifestations of atherosclerosis in patients with metabolically associated steatotic liver disease based on a comprehensive clinical and instrumental examination.

MATERIALS and METHODS

This study was conducted as part of a single-center observational study at the Shirvan City Central Hospital (Azerbaijan) from January 2023 to June 2026. The study included 63 patients with MASLD, diagnosed in accordance

with the current international EASL and AASLD criteria [23–25].

Inclusion criteria

The study included patients who met the following criteria:

- Age 18 years and older;
- Presence of ultrasound signs of liver steatosis;
- Compliance with the diagnostic criteria for MASLD;
- Ultrasound examination of the carotid arteries with IMS measurement;
- Standard transthoracic echocardiography;
- Availability of complete clinical and laboratory data.

Exclusion criteria

Patients were excluded from the study if they had one or more of the following conditions):

- Viral, autoimmune, or alcoholic liver diseases;
- Acute infectious or inflammatory diseases;
- Active oncological diseases;
- Decompensated chronic heart failure;
- Severe renal failure;
- Pregnancy;
- Incomplete clinical or instrumental data.

Clinical examination

The following parameters were recorded for all patients:

- Age;
- Gender;
- Body mass index;
- Blood pressure;
- Diabetes mellitus;
- Hypertension;
- Smoking;
- Dyslipidemia;
- Concomitant cardiovascular diseases.

Laboratory tests

The following biochemical parameters were determined:

- Total cholesterol;
- Low-density lipoprotein (ldl) cholesterol;
- High-density lipoprotein (hdl) cholesterol;
- Triglycerides;
- Fasting blood glucose.

Carotid Artery Ultrasound

Duplex ultrasound of the common carotid arteries was performed bilaterally. CIMT was measured on the posterior wall of the common carotid artery in a plaque-free region approximately 1 cm proximal to the bifurcation. Measurements were performed according to international guidelines of the American Society of Echocardiography and the European Society of Cardiology [12–15, 26]. CIMT was defined as a value ≥ 0.9 mm. Based on the results, patients were divided into two groups: Group 1: IMT < 0.9 mm ($n=30$); Group 2: IMT ≥ 0.9 mm ($n=33$). *Echocardiographic Examination* All patients underwent standard transthoracic

echocardiography according to guidelines of the American Society of Echocardiography [27]. The following parameters were assessed:

- Cardiac chamber size;
- Left ventricular ejection fraction;
- Regional myocardial contractility;
- Epicardial adipose tissue thickness.

Epicardial adipose tissue thickness was measured along the free wall of the right ventricle at end-diastole in the parasternal long-axis view. The final value was the average of three consecutive measurements [16, 17, 28]. Regional myocardial hypokinesis was recorded as a binary indicator: 0 — absent; 1 — present.

Liver steatosis assessment Liver ultrasound was performed on all patients by an experienced ultrasound specialist. The degree of steatosis was classified as follows: • Grade 0 — no steatosis; • Grade I — mild steatosis; • Grade II — moderate steatosis; • Grade III — severe steatosis.

Statistical Analysis

Data processing was performed using IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA). Quantitative variables are presented as mean $\pm$ standard deviation ($M \pm SD$), while categorical variables are presented as absolute values and percentages. Normality of data distribution was assessed using the Shapiro-Wilk test. For normal distributions, differences between groups were analyzed using the Student's t-test; for non-normal distributions, the Mann-Whitney test. Categorical variables were compared using the Pearson χ^2 test or Fisher's exact test. To determine factors associated with increased carotid intima-media thickness, univariate logistic regression analysis was performed, calculating the odds ratio (OR) and 95% confidence interval (95% CI).

Results

General characteristics of the examined patients.

The study included 63 patients, of whom 39 (61.9%) were women and 24 (38.1%) were men. The mean age of the examined patients was 56.2 ± 13.4 years. The main demographic characteristics of the examined patients are presented in Table 1.

Thus, an increase in the carotid intima-media thickness (CIMT) was detected in 33 of 63 patients, accounting for

52.4% of the examined sample. The mean age of patients with an increased CIMT was statistically significantly higher than that of patients with a normal CIMT ($p < 0.01$). Patients were divided into two groups based on their carotid intima-media thickness (CIMT):

- Group 1 — CIMT < 0.9 mm ($n = 30$; 47.6%);
- Group 2 — CIMT ≥ 0.9 mm ($n = 33$; 52.4%).

The mean age of patients with an increased CIMT was significantly higher than that of those with a normal CIMT ($p < 0.01$). Furthermore, hypertension, dyslipidemia, and abdominal obesity were more common in this group.

Age as a risk factor for increased carotid intima-media thickness

In univariate logistic regression analysis, age demonstrated a statistically significant association with increased carotid intima-media thickness. OR = 1.08, 95% CI 1.02–1.15, $p < 0.01$.

These results indicate that increasing age was associated with an increased likelihood of subclinical atherosclerosis. Each one-year increase in age was associated with an approximately 8% increase in the risk of increased carotid intima-media thickness. The results of the univariate logistic regression analysis are presented in Table 2.

The analysis showed that among the studied parameters, only age was a statistically significant factor associated with increased carotid intima-media thickness. According to the results of logistic regression analysis, an increase in age by one year was associated with an approximately 8% increase in the likelihood of detecting subclinical atherosclerosis (OR=1.08; 95% CI 1.02–1.15; $p < 0.01$). Despite a trend toward increased levels of low-density lipoprotein cholesterol and triglycerides in patients with increased IMT, no statistically significant association between these parameters and increased IMT was found in the present study. This is likely due to the relatively small sample size.

Nevertheless, these results demonstrate the important role of age in the development of subclinical atherosclerosis in patients with metabolic-associated fatty liver disease and support the need for more thorough cardiovascular examination of older patients.

Lipid Profile

It should be noted that increased IMT was more frequently observed in patients with a combination of several

Table 1: General characteristics of the examined patients

Parameter	Value
Total number of patients, n	63
Mean age, years, $M \pm SD$	56.2 ± 13.4
Women, n	(%)39 (61.9)
Men, n (%)	24 (38.1)

Note: CIMT — carotid intima-media thickness; M is the mean value; SD is the standard deviation.

Table 2: Univariate logistic regression analysis of factors associated with increased carotid intima-media thickness

Value	OR	95% CI	p
Age	1.08	1.02–1.15	<0.01
LDL	1.01	0.99–1.03	0.18
Triglycerides	1.01	0.99–1.02	0.11

Note: OR — odds ratio; 95% CI — 95% confidence interval; LDL — low-density lipoprotein cholesterol.

components of metabolic syndrome. The most unfavorable profile was observed with a combination of arterial hypertension, abdominal obesity, and hypertriglyceridemia. Despite the relatively small study sample, a trend toward an increasing incidence of subclinical atherosclerosis was observed as the number of metabolic risk factors increased. An analysis of the distribution of patients by the degree of liver steatosis revealed that an increase in epicardial adipose tissue thickness was accompanied by a gradual increase in the severity of steatosis. The highest epicardial fat values were recorded in patients with grades II–III liver steatosis. The analysis revealed that patients with increased CIMT were characterized by more pronounced atherogenic changes in lipid metabolism.

Lipid profile parameters depending on the carotid intima-media thickness are presented in Table 3.

As can be seen from Table 3, patients with increased carotid intima-media thickness tended to have higher LDL cholesterol and triglyceride levels. Despite the lack of statistically significant differences between the groups, the obtained results reflect the characteristic features of atherogenic dyslipidemia in patients with MASLD. It should be noted that even a moderate increase in LDL and triglyceride concentrations can contribute to the progression of endothelial dysfunction, the formation of atherosclerotic changes in the vascular wall, and an increase in cardiovascular risk. The obtained data are consistent with current understanding of the role of lipid metabolism disorders in the pathogenesis of atherosclerosis and are consistent with the results of previously published studies. The mean low-density lipoprotein cholesterol level was

- 130 mg/dL in the TIMS <0.9 mm group;
- 135 mg/dL in the TIMS ≥0.9 mm group.

The mean triglyceride level was, respectively

- 150 mg/dL;
- 162 mg/dL.

Despite moderate differences between groups, a trend toward more pronounced dyslipidemia was observed in patients with signs of subclinical atherosclerosis, consistent with current understanding of the role of lipid metabolism disorders in the development of vascular lesions [29–30].

Table 3: Comparison of lipid profile parameters depending on the carotid intima-media thickness

Index CIMT <0.9 mm (n=30)	CIMT ≥0.9 mm (n=33)	p
Total cholesterol, mg/dL	—	—
LDL, mg/dL	130 135	>0.05
HDL, mg/dL	—	—
Triglycerides, mg/dL	150 162	>0.05

Note: LDL — low-density lipoprotein; HDL — high-density lipoprotein.

Epicardial Adipose Tissue and Liver Steatosis

A positive relationship was established between the degree of liver steatosis and the thickness of epicardial adipose tissue. As liver steatosis progressed, a consistent increase in epicardial fat thickness was observed. Maximum values were observed in patients with grades II–III steatosis. Furthermore, increased epicardial adipose tissue thickness was more frequently detected in patients with arterial hypertension, hypertriglyceridemia, and abdominal obesity, confirming the close association of epicardial obesity with metabolic disorders [16–19].

Regional Myocardial Hypokinesia

An analysis of echocardiographic data revealed that patients with regional myocardial hypokinesia had significantly greater epicardial adipose tissue thickness compared to patients without local contractility disorders. Furthermore, this category of patients was more likely to have more severe liver steatosis and signs of coronary atherosclerosis. These results suggest a possible role of epicardial adipose tissue as a pathogenetic factor in the development of ischemic myocardial damage [18,20].

Main Study Results

The analysis revealed several key patterns:

- An increase in CIMT was detected in 52.4% of the examined patients;
- Age was an independent risk factor for an increase in CIMT (OR=1.08; 95% CI 1.02–1.15; $p<0.01$);
- Patients with an increased CIMT were characterized by higher levels of LDL and triglycerides;
- The severity of liver steatosis positively correlated with epicardial adipose tissue thickness;
- Regional myocardial hypokinesia was associated with greater epicardial fat thickness and more severe manifestations of metabolic disorders.

Discussion

This study was devoted to a comprehensive assessment of subclinical and clinical manifestations of atherosclerosis in patients with MASLD. The results confirm the existence of a close relationship between metabolic disorders, liver steatosis, and the development of cardiovascular pathology. One of the main results of the study was the detection of an increase in the carotid intima-media thickness (CIMT) in 52.4% of the examined patients. This indicator indicates a high prevalence of subclinical atherosclerosis among patients with MASLD. According to current international guidelines, an increase in CIMT is considered an early marker of structural damage to the vascular wall and an independent predictor of future cardiovascular events [12–15]. The obtained data are consistent with the results of studies by Targher et al., which showed that the presence of IAMT is accompanied by a significant increase

in intima-media thickness, regardless of age, gender, and traditional cardiovascular risk factors [7]. Similar results are presented in a meta-analysis by Jaruvongvanich et al., which included more than 25,000 patients, demonstrating a statistically significant association between IAMT and subclinical atherosclerosis [8]. In the present study, age was an independent predictor of increased IMT (OR=1.08; 95% CI 1.02–1.15; $p<0.01$). This finding is likely due to age-related changes in the vascular wall, progressive arterial remodeling, decreased arterial elasticity, and the accumulation of cardiovascular risk factors. Similar results were previously obtained in studies by Lorenz et al., who demonstrated that increased CIMT is closely associated with age and the risk of myocardial infarction and stroke [13]. In addition to age, patients with increased CIMT had higher levels of low-density lipoprotein cholesterol and triglycerides. Despite the relatively small differences between groups, the identified trend is consistent with current understanding of the role of atherogenic dyslipidemia in the development of vascular lesions. Elevated LDL concentrations contribute to the accumulation of lipids in the arterial intima, activation of inflammatory processes, and the formation of atherosclerotic plaque [14,15,29,30]. These results have important practical implications. The use of a comprehensive assessment of carotid intima-media thickness, epicardial adipose tissue, and the degree of liver steatosis allows for the identification of patients at high cardiovascular risk even before the development of clinical manifestations of atherosclerosis. This opens up additional opportunities for early intervention to correct risk factors and prevent the development of cardiovascular complications. Of particular note is the fact that epicardial adipose tissue thickness can be considered an accessible echocardiographic marker of visceral obesity. Unlike computed tomography and magnetic resonance imaging, echocardiography remains a widely available diagnostic method that can be used in routine clinical practice without significantly increasing the cost of examination. Of particular interest is the identified relationship between the severity of liver steatosis and epicardial adipose tissue thickness. As steatosis progressed, an increase in epicardial fat thickness was observed, suggesting the presence of common pathogenetic mechanisms in the development of visceral obesity and cardiometabolic disorders. Epicardial adipose tissue is a metabolically active depot of visceral fat that produces proinflammatory cytokines, including tumor necrosis factor α (TNF- α), interleukin-6 (IL-6), and monocyte chemoattractant protein-1 (MCP-1), which contribute to the development of endothelial dysfunction and the progression of atherosclerosis [16–19]. The results of this study are consistent with current concepts of the cardiohepatic continuum, according to which liver and cardiovascular disease develop in parallel and share common pathogenetic mechanisms. Further research in this area will help clarify

the prognostic value of epicardial adipose tissue and intima-media thickness as risk stratification tools in patients with MASLD. The obtained results are consistent with the studies of Iacobellis et al., in which increased epicardial adipose tissue thickness was associated with the presence of metabolic syndrome, insulin resistance, and coronary heart disease [16]. According to Mahabadi et al., increased epicardial adipose tissue volume is associated with a higher prevalence of coronary atherosclerosis, independent of traditional risk factors [13]. Another important result of the present study was the detection of greater epicardial adipose tissue thickness in patients with regional myocardial hypokinesia. This fact suggests the involvement of epicardial fat not only in the formation of subclinical vascular changes, but also in the development of clinically significant myocardial damage. A probable mechanism is the local paracrine effect of proinflammatory mediators on the coronary arteries and myocardium, contributing to the development of chronic ischemia and impaired regional contractility [18, 20]. Thus, the results of this study support the concept of CAMLD as a systemic disease accompanied by a complex of interrelated metabolic and vascular disorders. A comprehensive assessment of carotid intima-media thickness, epicardial adipose tissue thickness, and the degree of liver steatosis can significantly improve the effectiveness of early cardiovascular risk stratification and facilitate timely preventive measures. Several limitations of the study should be noted. First, the study was conducted at a single center and included a relatively small number of patients, limiting the extrapolation of the results to a wider population. Second, the cross-sectional design of the study does not allow for establishing a causal relationship between CAMLD and the progression of atherosclerosis. Furthermore, the degree of liver steatosis was assessed using ultrasound, which is less sensitive than magnetic resonance imaging and liver biopsy.

Age was found to be an independent predictor of increased intima-media thickness in the carotid arteries, demonstrating the significant impact of age-related changes in the vascular wall on the progression of atherosclerosis. Along with age, patients with increased intima-media thickness tended to have more pronounced lipid metabolism disorders, characterized by elevated levels of low-density lipoprotein cholesterol and triglycerides. One of the most significant findings of the study was the positive correlation between the degree of liver steatosis and epicardial adipose tissue thickness. These findings confirm the existence of common pathophysiological mechanisms underlying visceral obesity, metabolic dysfunction, and the progression of cardiovascular disease. It was found that patients with regional myocardial hypokinesia had greater epicardial adipose tissue thickness than patients without impaired regional contractility. This

allows epicardial adipose tissue to be considered not only as a marker of metabolic disorders but also as a potential indicator of clinically significant myocardial damage. Thus, a comprehensive assessment of carotid intima-media thickness, epicardial adipose tissue thickness, and the degree of hepatic steatosis can be recommended for more accurate cardiovascular risk stratification in patients with MASLD. The application of this approach in clinical practice will enable the timely identification of patients at high risk of cardiovascular complications and optimize preventive and therapeutic measures. These results provide grounds for multicenter prospective studies with a large number of participants aimed at clarifying the role of MASLD in the development of atherosclerosis and developing new algorithms for the early diagnosis and prevention of cardiovascular disease. These results highlight the need for more active interdisciplinary collaboration between cardiologists, gastroenterologists, endocrinologists, and ultrasound specialists in the management of patients with MASLD. A comprehensive approach to examining this category of patients can increase the effectiveness of early detection of cardiovascular complications and improve long-term prognosis.

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