RESEARCH PAPER

Severe Liver Steatosis and Cardiovascular Disease in Patients with Metabolic Dysfunction-Associated Fatty Liver Disease

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ABSTRACT

Background: Metabolic dysfunction–associated fatty liver disease (MAFLD) is widely acknowledged as a multisystem condition linked to increased cardiovascular morbidity and mortality. However, data remain scarce on whether liver steatosis severity correlates with specific cardiovascular manifestations. This study examines the association between liver steatosis extent and CVD, including CAD, heart failure, and arrhythmia in MAFLD.
Methods and findings: This observational cross-sectional study included 152 patients with MAFLD at a tertiary care center between July 2025 and March 2026. Liver steatosis was assessed by transient elastography (FibroScan) with Controlled Attenuation Parameter (CAP) and classified as mild–moderate (<289 dB/m) or severe (≥290 dB/m). Cardiovascular outcomes were CAD, heart failure, and arrhythmia. Severe steatosis occurred in 57.2% and cardiovascular disease in 36.2%; CAD was most common (23.0%). Severe steatosis was associated with CAD (p=0.020; OR=2.65, 95% CI: 1.14–6.15) and CVD (adjusted OR=3.29; 95% CI: 1.51–7.16), but not heart failure or arrhythmia. No significant associations were observed for either outcome.
Conclusion: Severe liver steatosis is associated with increased cardiovascular risk in patients with MAFLD, particularly coronary artery disease, and may support cardiovascular risk stratification in clinical practice.

Keywords: MAFLD; liver steatosis; cardiovascular disease; coronary artery disease; transient elastography

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INTRODUCTION

Metabolic dysfunction–associated fatty liver disease (MAFLD) is a chronic liver disease characterized by hepatic steatosis in the presence of metabolic dysfunction, including obesity, type 2 diabetes mellitus (T2DM), or metabolic syndrome.[1–3] Hepatic steatosis is defined as excessive lipid accumulation exceeding 5% of liver parenchyma and currently represents the most common cause of chronic liver disease worldwide.[4,5] The global prevalence of MAFLD has been estimated to exceed one-third of the general population and continues to increase in parallel with the growing burden of obesity and T2DM, particularly in Asia.[3,5]

The pathophysiology of MAFLD involves metabolic dysregulation leading to hepatic lipid accumulation, oxidative stress, mitochondrial dysfunction, and chronic inflammation.[5,6] These processes extend beyond hepatic injury and contribute to systemic vascular damage and cardiometabolic complications.[3,6,7] Consequently, MAFLD is increasingly recognized as a multisystem disease with substantial cardiovascular implications.[5]

Cardiovascular disease (CVD), including coronary artery disease, heart failure, arrhythmia, and cerebrovascular disease, remains the leading cause of mortality among patients with MAFLD, exceeding liver-related mortality.[8,9] Several studies have demonstrated that hepatic steatosis

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independently predicts cardiovascular outcomes through mechanisms involving insulin resistance, endothelial dysfunction, atherogenic dyslipidemia, and systemic inflammation.[5,10,11] A large cohort study in Korea reported that severe hepatic steatosis assessed using the Fatty Liver Index was associated with increased risk of heart failure, while evidence from North America demonstrated a higher incidence of coronary artery disease among patients with hepatic steatosis.[11,12]

Although MAFLD has been consistently associated with an increased risk of cardiovascular disease (CVD), including coronary artery disease, heart failure, and arrhythmia, evidence regarding the relationship between the severity of hepatic steatosis and specific cardiovascular manifestations remains limited, particularly in Indonesia and other Southeast Asian populations.[5,10,11] Since CVD represents the leading cause of mortality among patients with MAFLD, identifying the cardiovascular burden across different stages of hepatic steatosis may improve risk stratification and support earlier preventive strategies.[5,9] Therefore, this study aimed to analyze the relationship between mild, moderate, and severe liver steatosis and the occurrence of coronary artery disease, heart failure, and arrhythmia among patients with MAFLD. In addition, this study evaluated the distribution of cardiovascular disease across each stage of hepatic steatosis in MAFLD patients.

MATERIALS AND METHODS

Study design, setting, and participants

This observational analytical cross-sectional study used secondary data obtained from medical records. The study was conducted at the outpatient and inpatient units of a tertiary care hospital. Data collection was performed from July 2025 to March 2026 until the required sample size was achieved.

The study population consisted of all adult patients diagnosed with MAFLD who attended outpatient or inpatient services during the study period. Eligible participants were selected using purposive sampling. The minimum sample size was estimated using a comparative analytical formula based on the expected standard deviation (SD = 1.90), estimated mean difference (0.85), α level of 0.05, and statistical power of 80%, resulting in a minimum required sample of 77 patients.

Eligibility criteria

Patients were included if they were aged ≥18 years, fulfilled the diagnostic criteria for MAFLD, and had complete medical records including laboratory examination and at least one imaging modality for hepatic steatosis assessment (abdominal ultrasonography, computed tomography, transient elastography/FibroScan, or liver biopsy if available), as well as cardiovascular evaluation data including electrocardiography, echocardiography, or coronary imaging. Patients with incomplete clinical records or unavailable hepatic steatosis assessment were excluded from analysis.

Liver steatosis and cardiovascular disease assessment

The primary independent variable was liver steatosis severity, while the primary outcome variable was CVD. Hepatic steatosis was assessed using transient elastography (FibroScan®) with CAP, expressed in decibels per meter (dB/m). CAP values were categorized into mild–moderate steatosis (S1–S2: <289 dB/m) and severe steatosis (S3: ≥290 dB/m), based on validated thresholds against histological findings.[13]

Cardiovascular disease was defined as the presence of at least one of the following conditions: coronary artery disease, heart failure, or atrial fibrillation. Coronary artery disease was diagnosed based on clinical presentation and supporting investigations including electrocardiography, echocardiography, previous percutaneous coronary intervention history, or coronary angiography findings. Heart failure was identified based on clinical symptoms with echocardiographic confirmation, while arrhythmia was defined as atrial fibrillation documented on electrocardiography. Patients were categorized as having CVD if one or more cardiovascular conditions were identified.

Covariates

Demographic and clinical variables included age, sex, body mass index (BMI), and waist circumference. Age was categorized into <60 years and ≥60 years. BMI classification followed the Asia–Pacific criteria: underweight (<18.5 kg/m²), normal (18.5–22.9 kg/m²), overweight (23.0–24.9 kg/m²), and obesity (≥25.0 kg/m²). Waist circumference was obtained from anthropometric records and used as an indicator of visceral adiposity.

Data collection procedure

Medical records of eligible patients were reviewed systematically using a standardized extraction form. Information collected included demographic characteristics, metabolic parameters, FibroScan results, cardiovascular evaluations, and supporting laboratory findings. Data completeness and consistency were verified before statistical analysis.

Statistical analysis

Data were analyzed using IBM SPSS Statistics version 30. Continuous variables were summarized as mean ± standard deviation or median with interquartile range depending on distribution, while categorical variables were presented as frequency and percentage. The association between liver steatosis severity and cardiovascular disease was evaluated using Chi-square or Fisher's exact test where appropriate. Statistical significance was defined as a two-tailed p-value <0.05. Variables associated with cardiovascular disease in the bivariate analysis were further examined using multivariable binary logistic regression, adjusting for age, sex, hypertension, and type 2 diabetes mellitus; results were expressed as adjusted odds ratios (aOR) with 95% confidence intervals. Coronary artery disease was modelled separately, adjusting for age and sex only owing to the limited number of events.

Ethics Approval and Consent to Participate

This study was approved by the Biomedical Research Ethics Committee, Faculty of Medicine, Universitas Hasanuddin, Indonesia (Approval No. 577A/UN4.6.4.5.31/PP36/2025). The study was conducted in accordance with the ethical principles of the Declaration of Helsinki. As this study used retrospective medical record data, patient confidentiality and anonymity were maintained throughout the study.

RESULTS

Characteristics of study participants

A total of 152 patients with metabolic dysfunction–associated fatty liver disease (MAFLD) met the eligibility criteria and were included in the analysis. Baseline characteristics are summarized in Table 1. The study population showed a relatively balanced sex distribution, comprising 77 males (50.7%) and 75 females (49.3%). Most participants were aged <60 years (77.6%). Based on body mass index (BMI), overweight was the predominant category (57.2%), followed by obesity (33.6%) and normal BMI (9.2%). Central obesity based on waist circumference was observed in 91.4% of participants. Most participants were non-smokers (69.1%) and had no family history of cardiovascular disease (70.4%). Hypertension and type 2 diabetes mellitus were each identified in 46.1% of participants. Transient elastography (FibroScan) showed that severe liver steatosis was more frequent than mild–moderate steatosis (57.2% vs. 42.8%). Cardiovascular disease (CVD) was identified in 36.2% of participants, with coronary artery disease being the most common manifestation (23.0%), followed by heart failure (10.5%) and arrhythmia (2.6%).

Coronary artery disease across liver steatosis severity in patients with MAFLD

Table 2 presents the distribution of coronary artery disease (CAD) according to liver steatosis severity among patients with metabolic dysfunction–associated fatty liver disease (MAFLD). Among patients without CAD (n=117), 56 (47.9%) had mild–moderate steatosis and 61 (52.1%) had severe steatosis. In contrast, most patients with CAD had severe steatosis (n=26, 74.3%), whereas only 9 patients (25.7%) had mild–moderate steatosis. Chi-square analysis demonstrated a significant association between liver steatosis severity and CAD occurrence (p=0.020). Patients with severe steatosis had a 2.65-fold higher odds of developing CAD compared with those with mild–moderate steatosis (OR=2.65; 95% CI: 1.14–6.15). After adjustment for age and sex, severe steatosis remained independently associated with CAD (adjusted OR=2.58; 95% CI: 1.11–6.02; p=0.028).

Heart failure across liver steatosis severity in patients with MAFLD

Table 3 presents the distribution of heart failure according to liver steatosis severity among patients with metabolic dysfunction–associated fatty liver disease (MAFLD). Among patients without heart failure (n=136), 61 (44.9%) had mild–moderate steatosis and 75 (55.1%) had severe steatosis. Among patients with heart failure, most had severe steatosis (n=12, 75.0%), while 4 patients (25.0%) had mild–moderate steatosis. Although heart failure was more frequently observed in patients with severe steatosis, Chi-square analysis showed no statistically significant association between liver steatosis severity and heart failure occurrence (p=0.129).

Arrhythmia across liver steatosis severity in patients with MAFLD

Table 4 presents the distribution of arrhythmia according to liver steatosis severity among patients with metabolic dysfunction–associated fatty liver disease (MAFLD). Among patients without arrhythmia (n=148), 64 (43.2%) had mild–moderate steatosis and 84 (56.8%) had severe steatosis. Among patients with arrhythmia, most had severe steatosis (n=3, 75.0%), whereas only 1 patient (25.0%) had mild–moderate steatosis. Despite the higher proportion of arrhythmia observed in patients with severe steatosis, Fisher's exact test demonstrated no statistically significant association between liver steatosis severity and arrhythmia occurrence (p=0.636).

Cardiovascular disease across liver steatosis severity in patients with MAFLD

Table 5 presents the association between liver steatosis severity and overall cardiovascular disease (CVD) among 152 patients with metabolic dysfunction–associated fatty liver disease (MAFLD). Among patients without CVD, 51 (52.6%) had mild–moderate steatosis and 46 (47.4%) had severe steatosis. In contrast, among patients with CVD, severe steatosis was more predominant, affecting 41 patients (74.5%), whereas 14 patients (25.5%) had mild–moderate steatosis. Chi-square analysis demonstrated a significant association between liver steatosis severity and CVD occurrence (p=0.001). Patients with severe steatosis had 3.25-fold higher odds of developing cardiovascular disease compared with those with mild–moderate steatosis (OR=3.25; 95% CI: 1.57–6.71). In the multivariable logistic regression model adjusting for age, sex, hypertension, and type 2 diabetes mellitus, severe liver steatosis remained independently associated with cardiovascular disease (adjusted OR=3.29; 95% CI: 1.51–7.16; p=0.003), while type 2 diabetes mellitus was also an independent predictor (adjusted OR=4.51; 95% CI: 1.99–10.19; p<0.001) (Table 6).

DISCUSSION

This study demonstrated a significant association between liver steatosis severity and CVD among patients with MAFLD. Patients with severe steatosis had 3.25-fold higher odds of CVD and 2.65-fold higher odds of CAD compared with those with mild–moderate steatosis. In contrast, no statistically significant associations were observed for heart failure and arrhythmia. The baseline characteristics reflected a metabolically high-risk population, characterized by high proportions of overweight/obesity, central obesity, hypertension, and type 2 diabetes mellitus. These findings align with previous evidence showing that obesity and insulin resistance are central drivers of MAFLD progression and major

contributors to cardiovascular risk through chronic inflammation, endothelial dysfunction, and oxidative stress.[2,14,15] The predominance of severe steatosis in this cohort further supports the concept that advanced hepatic fat accumulation represents a marker of systemic metabolic burden.

The strongest association in this study was observed for CAD. This finding is consistent with studies showing that hepatic steatosis assessed using Controlled Attenuation Parameter (CAP) correlates with coronary disease burden and cardiovascular risk. Sardjan et al. (2024) demonstrated a positive association between CAP values and SYNTAX score, suggesting that greater steatosis severity parallels increasing coronary lesion complexity.[16] Similarly, Li et al. (2025), using NHANES data, reported that liver steatosis measured by transient elastography was independently associated with coronary heart disease after adjustment for conventional cardiovascular risk factors.[17] Our findings also support observations by de Sousa Magalhaes et al. (2021), who identified severe steatosis as an independent predictor of cardiovascular events.[18]

Several biological mechanisms may explain this relationship. Excessive hepatic lipid accumulation promotes lipotoxicity, insulin resistance, and release of pro-inflammatory mediators, resulting in endothelial dysfunction and accelerated atherosclerosis.[14,19] In addition, MAFLD contributes to an atherogenic lipid profile characterized by increased very low-density lipoprotein and reduced HDL cholesterol, further increasing coronary plaque formation.[15] Although heart failure and arrhythmia were more frequently observed in patients with severe steatosis, the associations did not reach statistical significance. Previous studies suggest that MAFLD contributes to myocardial remodeling, fibrosis, and electrical conduction abnormalities that may predispose to heart failure and arrhythmia.[20,21] However, these outcomes may involve more complex and longer disease pathways than coronary atherosclerosis, potentially reducing detectability in a cross-sectional design.

These findings suggest that transient elastography with CAP may have utility beyond liver assessment and could support cardiovascular risk stratification in patients with MAFLD. Early identification of severe steatosis may help identify individuals requiring further cardiovascular evaluation. This study has several limitations. First, the cross-sectional design precludes causal inference. Second, cardiovascular manifestations may coexist in the same individual, introducing potential outcome overlap and classification bias. Third, liver steatosis was assessed using FibroScan without histopathological confirmation.

CONCLUSIONS

Among 152 patients with MAFLD, severe liver steatosis assessed using transient elastography (FibroScan) was more prevalent than mild–moderate steatosis. Cardiovascular disease was identified in more than one-third of patients, with CAD being the most common manifestation. This study demonstrated a significant association between liver steatosis severity and cardiovascular disease. Patients with severe steatosis had 3.25-fold higher odds of cardiovascular disease and 2.65-fold higher odds of coronary artery disease compared with patients with mild–moderate steatosis. No significant associations were observed for heart failure and arrhythmia. These findings suggest that severe liver steatosis in MAFLD may serve as a marker of increased cardiovascular risk, supporting the potential role of transient elastography not only for liver assessment but also for cardiovascular risk stratification and early clinical evaluation. This association remained significant after adjustment for age, sex, hypertension, and type 2 diabetes mellitus, indicating that severe steatosis is independently associated with cardiovascular disease rather than merely reflecting shared metabolic risk factors.

DECLARATIONS

Data Availability

Data cannot be shared publicly because the dataset contains potentially identifiable patient information. Data are available from the corresponding author upon reasonable request and with approval from the Biomedical Research Ethics Committee, Faculty of Medicine, Hasanuddin University.

Competing Interests

The authors have declared that no competing interests exist.

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REFERENCES

1. Gofton C, Upendran Y, Zheng MH, George J. MAFLD: How is it different from NAFLD? Clin Mol Hepatol. 2023;29(Suppl):S17-S31.

doi:10.3350/CMH.2022.0367.

2. Gofton C, George J. Updates in fatty liver disease: pathophysiology, diagnosis and management. Aust J Gen Pract. 2021;50(10):702-707. doi:10.31128/AJGP-05-21-5974.
3. Eslam M, Fan JG, Yu ML, Wong VWS, Cua IH, Liu CJ, et al. The Asian Pacific Association for the Study of the Liver clinical practice guidelines for the diagnosis and management of metabolic dysfunction-associated fatty liver disease. Hepatol Int. 2025;19(2):261-301. doi:10.1007/s12072-024-10774-3.
4. Segura-Azuara N de los Á, Varela-Chinchilla CD, Trinidad-Calderón PA. MAFLD/NAFLD biopsy-free scoring systems for hepatic steatosis, NASH, and fibrosis diagnosis. Front Med (Lausanne). 2022;8:774079. doi:10.3389/fmed.2021.774079.
5. Pipitone RM, Ciccioli C, Infantino G, La Mantia C, Parisi S, Tulone A, et al. MAFLD: a multisystem disease. Ther Adv Endocrinol Metab. 2023;14:20420188221145549. doi:10.1177/20420188221145549.
6. Xu H, Wang L. The role of Notch signaling pathway in non-alcoholic fatty liver disease. Front Mol Biosci. 2021;8:792667. doi:10.3389/fmolb.2021.792667.
7. Li Y, Yang P, Ye J, Xu Q, Wu J, Wang Y. Updated mechanisms of MASLD pathogenesis. Lipids Health Dis. 2024;23(1):117. doi:10.1186/s12944-024-02108-x.
8. Muzurović E, Peng CCH, Belanger MJ, Sanoudou D, Mikhailidis DP, Mantzoros CS. Nonalcoholic fatty liver disease and cardiovascular disease: a review of shared cardiometabolic risk factors. Hypertension. 2022;79(7):1319-1326. doi:10.1161/Hypertensionaha.122.17982.
9. Badmus OO, Hinds TD, Stec DE. Mechanisms linking metabolic-associated fatty liver disease (MAFLD) to cardiovascular disease. Curr Hypertens Rep. 2023;25(8):151-162. doi:10.1007/s11906-023-01242-8.
10. Boccatonda A, Andreetto L, D'Ardes D, Cocco G, Rossi I, Vicari S, et al. From NAFLD to MAFLD: definition, pathophysiological basis and cardiovascular implications. Biomedicines. 2023;11(3):883. doi:10.3390/biomedicines11030883.
11. Meyersohn NM, Mayrhofer T, Corey KE, Bittner DO, Staziaki PV, Szilveszter B, et al. Association of hepatic steatosis with major adverse cardiovascular events, independent of coronary artery disease. Clin Gastroenterol Hepatol. 2021;19(7):1480-1488.e14. doi:10.1016/j.cgh.2020.07.030.
12. Park J, Kim G, Kim H, Lee J, Jin SM, Kim JH. The associations between changes in hepatic steatosis and heart failure and mortality: a nationwide cohort study. Cardiovasc Diabetol. 2022;21(1):250. doi:10.1186/s12933-022-01725-z.
13. Goyal P, Chopra R, Goyal O, Goyal P, Chopra R, Goyal O. Do hepatic fibrosis and steatosis measured by hepatic transient elastography (FibroScan) predict cardiovascular risk in patients with non-alcoholic fatty liver disease: an observational cross-sectional study. Indian J Cardiovasc Dis Women. 2024;9(2):66-73. doi:10.25259/IJCDW_2_2024.
14. Hassen G, Singh A, Belete G, Jain N, De la Hoz I, Camacho-Leon GP, et al. Nonalcoholic fatty liver disease: an emerging modern-day risk factor for cardiovascular disease. Cureus. 2022;14(5):e25495. doi:10.7759/cureus.25495.
15. Ratti C, Malaguti M, Emanuele DA, Bellasi A, Sanna G. Understanding MASLD—from molecular pathogenesis to cardiovascular risk: a concise review for the clinical cardiologist. Atherosclerosis. 2025;409:120495. doi:10.1016/j.atherosclerosis.2025.120495.
16. Sardjan J, Lesmana CRA, Rusdi L, Kurniawan J, Yunihastuti E, Susilo A, et al. Correlation between controlled attenuation parameter values with SYNTAX score in patients with significant coronary artery disease. Sci Rep. 2024;14(1):15382. doi:10.1038/s41598-024-63792-4.
17. Li S, Li X, Xiao H, Dou J. The relationship between liver stiffness, fat content measured by liver elastography, and coronary artery disease: a study based on the NHANES database. Sci Rep. 2025;15(1):30010. doi:10.1038/s41598-025-15709-y.
18. de Sousa Magalhães R, Xavier S, Magalhães J, Rosa B, Marinho C, Cotter J. Transient elastography through controlled attenuation parameter assisting the stratification of cardiovascular disease risk in NAFLD patients. Clin Res Hepatol Gastroenterol. 2021;45(5):101561. doi:10.1016/j.clinre.2020.11.010.
19. Abbaszadeh M, Hosseinpanah F, Tohidi M, Karimpour Reyhan S, Mahdavi M, Valizadeh M. Sex-specific impact of metabolic dysfunction-associated fatty liver disease on incident cardiovascular diseases and mortality. Endocrinol Diabetes Metab. 2025;8(3):e70035. doi:10.1002/edm2.70035.
20. Simon TG, Ebrahimi F, Roelstraete B, Hagström H, Sundström J, Ludvigsson JF. Incident cardiac arrhythmias associated with metabolic dysfunction-associated steatotic liver disease: a nationwide histology cohort study. Cardiovasc Diabetol. 2023;22(1):276. doi:10.1186/s12933-023-02070-5.
21. Szymala-Pędzik M, Piersiak M, Pachana M,

Lindner-Pawłowicz K, Szczepaniak W, Sobieszczańska M. MAFLD as a cardiovascular risk factor: an extended retrospective study with a control group. J Clin Med. 2025;14(12):4181. doi:10.3390/jcm14124181.

Table 1. Demographic Characteristics of Study Subjects

Variable	Category	n (%)
Sex	Female	75 (49.3%)
	Male	77 (50.7%)
Age	< 60 years	118 (77.6%)
	≥ 60 years	34 (22.4%)
Body Mass Index (BMI)	Normal	14 (9.2%)
	Overweight	87 (57.2%)
	Obesity	51 (33.6%)
Waist Circumference	Normal	13 (8.6%)
	Central Obesity	139 (91.4%)
Hypertension	No	82 (53.9%)
	Yes	70 (46.1%)
Type 2 Diabetes Mellitus	No	82 (53.9%)
	Yes	70 (46.1%)
Smoking Status	No	105 (69.1%)
	Yes	47 (30.9%)
Family History of Cardiovascular Disease	No	107 (70.4%)
	Yes	45 (29.6%)
Degree of Hepatic Steatosis	Mild–Moderate	65 (42.8%)
	Severe	87 (57.2%)
Cardiovascular Disease	No	97 (63.8%)
	Yes	55 (36.2%)
Type of Cardiovascular Disease	Coronary Artery Disease (CAD)	35 (23.0%)
	Heart Failure	16 (10.5%)
	Arrhythmia	4 (2.6%)

Table 2. Coronary Artery Disease (CAD) Across Different Degrees of Hepatic Steatosis in Patients with MAFLD

Degree of Hepatic Steatosis	No CAD (n=117)	CAD (n=35)	p-value	OR (95% CI)
Mild–Moderate	56 (47.9%)	9 (25.7%)	0.020	2.65 (1.14–6.15)
Severe	61 (52.1%)	26 (74.3%)		

Chi-square test; OR = odds ratio; CI = confidence interval; significance at $p < 0.05$.

Table 3. Heart Failure Across Different Degrees of Hepatic Steatosis in Patients with MAFLD

Degree of Hepatic Steatosis	No Heart Failure (n=136)	Heart Failure (n=16)	p-value
Mild–Moderate	61 (44.9%)	4 (25.0%)	0.129
Severe	75 (55.1%)	12 (75.0%)	

Chi-square test; significance at $p < 0.05$.

Table 4. Arrhythmia Across Different Degrees of Hepatic Steatosis in Patients with MAFLD

Degree of Hepatic Steatosis	No Arrhythmia (n=148)	Arrhythmia (n=4)	p-value
Mild–Moderate	64 (43.2%)	1 (25.0%)	0.636
Severe	84 (56.8%)	3 (75.0%)	

Fisher's Exact test; significance at $p < 0.05$.

Table 5. Cardiovascular Disease (CVD) Across Different Degrees of Hepatic Steatosis in Patients with MAFLD

Degree of Hepatic Steatosis	No CVD (n=97)	CVD (n=55)	p-value	OR (95% CI)
Mild–Moderate	51 (52.6%)	14 (25.5%)	0.001	3.25 (1.57–6.71)
Severe	46 (47.4%)	41 (74.5%)		

Chi-square test; OR = odds ratio; CI = confidence interval; significance at $p < 0.05$.

Table 6. Multivariable Logistic Regression of Factors Associated with Cardiovascular Disease in Patients with MAFLD

Variable	Adjusted OR (95% CI)	p-value
Severe steatosis (vs mild–moderate)	3.29 (1.51–7.16)	0.003

Age (per year)	0.99 (0.95–1.03)	0.589
Male sex	1.23 (0.59–2.55)	0.581
Hypertension	1.18 (0.53–2.65)	0.682
Type 2 diabetes mellitus	4.51 (1.99–10.19)	<0.001

Multivariable binary logistic regression; outcome = cardiovascular disease; aOR = adjusted odds ratio; CI = confidence interval; reference category for steatosis = mild–moderate; significance at $p < 0.05$.

Table 7. Multivariable Logistic Regression of Factors Associated with Coronary Artery Disease in Patients with MAFLD

Variable	Adjusted OR (95% CI)	p-value
Severe steatosis (vs mild–moderate)	2.58 (1.11–6.02)	0.028
Age (per year)	0.99 (0.96–1.03)	0.741
Male sex	1.34 (0.62–2.92)	0.456

Multivariable binary logistic regression; outcome = coronary artery disease; adjusted for age and sex; aOR = adjusted odds ratio; CI = confidence interval; reference category for steatosis = mild–moderate; significance at $p < 0.05$.