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Non-Invasive Assessment of MAFLD: The Role of Adiponectin and Metabolic Biomarkers in Liver Fibrosis Detection

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Abstract: Background: *Metabolically associated fatty liver disease (MAFLD) is a growing concern in Egypt, replacing viral hepatitis as the dominant liver disease, linked to obesity, diabetes, and metabolic dysfunction.*
Aim: *To assess the prevalence of MAFLD and investigate the role of serum adiponectin as a noninvasive liver fibrosis marker.*
Patients and Methods: *A cross-sectional case-control study was conducted on 101 participants, divided into two groups: the MAFLD (90 patients) and non-MAFLD (11 participants). All participants were undergoing complete history taking and full clinical examination, laboratory investigations (CBC, liver/renal profile, lipid profile, fasting blood glucose, HbA1c, fasting insulin, and serum adiponectin), imaging studies (ultrasound and FibroScan), and fibrosis evaluation using non-invasive scores (APRI, FIB-4, NFS, HSI, and FLI).*
Results: *MAFLD group patients had significantly higher BMI, waist circumference, FBS, HbA1c, and non-HDL-C. HDL and adiponectin (13.50 vs. 29.10 μg/L, p = 0.029) levels were considerably lower in MAFLD patients. Fibrosis and steatosis scores (LSM and CAP) were higher in the MAFLD group (p = 0.018, p < 0.001, respectively). MAFLD patients had more advanced liver involvement with higher liver stiffness stages (46.7%), higher CAP scores (53.3%), higher BARD scores (36.7%), and higher HSI (40.52 ± 9.34 vs. 34.02 ± 5.17). BMI was positively correlated with Hepatic Steatosis Index (HIS), METS-IR, triglycerides, VLDL-C, HbA1c, total cholesterol, LDL, non-HDL-C, TG/glucose ratio, BARD score, CAP, and LSM, while negatively correlated with LDL/VLDL ratio and adiponectin. The non-HDL cholesterol, total cholesterol, HbA1c, weight, BMI, and obesity are independent risk factors for the development of MAFLD, while adiponectin and HDL levels may serve as protective factors. The ROC curve analysis showed significant diagnostic value for diagnosing liver fibrosis in MAFLD patients. HbA1c had the highest diagnostic performance; HbA1c had an AUC of 0.839, HSI had an AUC of 0.767, BMI had an AUC of 0.762, and METS-IR had an AUC of 0.740, with high sensitivity and specificity. Adiponectin was found to be inversely associated with MAFLD.*
Conclusion: *Serum adiponectin level is a useful noninvasive marker for the detection and risk assessment of MAFLD fibrosis. Combining biochemical and anthropometric data enhances early diagnosis, especially in settings that lack imaging facilities.*
Keywords: *MAFLD, BMI, metabolic syndrome, liver fibrosis scores, hepatic steatosis index.*

INTRODUCTION

Egypt's chronic liver disease has seen a significant shift, with a decrease in viral hepatitis and a rise in metabolic-associated fatty liver disease (MAFLD), previously referred to as non-alcoholic fatty liver disease (NAFLD) **(Bazeed et al., 2022).** MAFLD has reached an alarming prevalence, posing serious health and economic burdens. Liver steatosis identified through histology, imaging, or non-invasive biomarkers, along with at least one of the following: overweight or obesity, type 2 diabetes mellitus, or clear clinical evidence of metabolic dysfunction **(Fouad et al., 2022).**Chronic kidney disease (CKD) and MAFLD often coexist in individuals with diabetes, hypertension, obesity, or dyslipidemia. Both diseases share overlapping metabolic risk factors and potentially common pathophysiological mechanisms **(Ahmed et al. 2025; Bazeed et al. 2025).**

Non-obese or lean NAFLD can be categorized into two forms. The first includes non-obese patients who may still have increased visceral adiposity or waist circumference **(Ghweil et al., 2023; Sayed et al., 2024).** The second includes truly lean individuals with no excess fat, where secondary causes such as high fructose intake, protein malnutrition, drug-induced steatosis, or genetic predisposition may play a role **(Ahadi et al., 2021)**.
This study aimed to identify the prevalence of MAFLD in the local population, explore its interaction with other organ dysfunctions, and assess its response to various risk factors. We looked at how liver fibrosis severity relates to serum adiponectin levels in MAFLD patients to see if adiponectin could be a useful noninvasive marker for fibrosis and how it connects to metabolic risk factors.

Patients and methods

This was a cross-sectional case-control study conducted from April 2022 to April 2023 at the Tropical Medicine and Gastroenterology Department, Qena University Hospital, Egypt.
The participants were divided into two groups: the MAFLD group (90 patients) and the non-MAFLD group (11 participants), based on clinical evaluation, anthropometric measurements, laboratory tests, ultrasonography, and FibroScan findings.
Patients with active malignancy, decompensated liver cirrhosis, a history of long-term use of hepatotoxic or weight-loss medications, positive HBsAg or HCV antibodies, genetic conditions such as hemochromatosis and Wilson disease, and excessive alcohol consumption (≥ 20 g/day for women or ≥ 30 g/day for men) were excluded from the study.
All participants were subjected to comprehensive history-taking, including age, sex, smoking, alcohol habits, and any relevant medical history, such as diabetes mellitus or hypertension, and full clinical examinations. MAFLD diagnosed according to European Association for the Study of the Liver (EASL) guideline **(EASL et al., 2024)**

Laboratory investigation:

Sampling of 10 ml of fasting venous blood under aseptic conditions, divided into EDTA tubes for complete blood count (CBC), citrate tubes for prothrombin time and INR, and plain tubes for liver and renal functions. Estimated glomerular filtration rate (eGFR) was calculated using the CKD-EPI equation, fasting lipid profile, fasting blood glucose, HbA1c, fasting serum insulin, and serum adiponectin. Adiponectin was measured using an ELISA Kit (Diagnostics Biochem (DBC) Canada Inc.), REF: CAN-APN-5000.

Anthropometric and non-invasive indices for evaluating inflammation, hepatic fibrosis, and steatosis:

- Body mass index (BMI) was calculated using a standard formula (weight in kilograms divided by height in meters squared).
- HOMA-IR (using fasting glucose and insulin)**.**
- Inflammatory markers such as NLR, PLR, and CRP/albumin ratio were also assessed.
- The HAIR score included hypertension, ALT, and insulin resistance. APRI was calculated using AST, its upper normal limit, and platelet count **(Browning et al., 2004)**.
- FIB-4 was computed using age, platelet count, AST, and ALT, with scores > 3.25 indicating significant fibrosis and < 1.45 excluding it **(Sterling et al., 2006)**.
- The BARD score used BMI ≥ 28 kg/m^2, AST/ALT ratio ≥ 0.8, and presence of diabetes **(Harrison et al., 2008).**
- The NAFLD fibrosis score (NFS) included age, hyperglycemia, BMI, platelet count, albumin, and AST/ALT ratio **(Angulo et al., 2007).**
- Fatty Liver Index (FLI) was based on waist circumference, BMI, triglycerides, and GGT, with scores < 30 excluding and ≥ 60 confirming fatty liver **(Bedogni et al., 2006)**.
- The Hepatic Steatosis Index (HSI) incorporated the ALT/AST ratio, BMI, sex, and diabetes status, where values < 30 were excluded and > 36 confirmed NAFLD **(Lee et al., 2010).**

Imaging study:

Abdominal ultrasonography using SONOACE R7 (Samsung Medison Co., Ltd., Korea) was used to diagnose NAFLD based on liver-kidney echogenicity contrast, portal vein wall visualization, and diaphragm clarity by a FibroScan® device (FibroScan, Echosens, Paris, France) by an expert FibroScan operator at the Internal Medicine Department, who was blinded about the patient's data**.** The assessment was conducted after fasting for at least 8 hours to measure liver stiffness (LSM) and controlled attenuation parameter (CAP); ten valid measurements were required for both LSM and CAP. The M probe was used initially, and if the M probe failed, an XL probe was used. Results from sessions with at least ten successful acquisitions were included in the final analysis.

Ethical approval

The study was approved by the Institutional Review Board of the Qena Faculty of Medicine, Egypt (code number SVU-**MED-GIT023-2-22-5-402);** following Helsinki guidelines, all participants gave informed consent to join the study.

Statistical analysis

Data were gathered, coded, and analyzed using IBM SPSS version 27. Categorical variables are expressed as numbers and percentages, while numerical variables are presented as means and standard deviations. Normality was tested using the Kolmogorov–Smirnov and Shapiro–Wilk tests. Chi-square, independent t-test, and Mann-Whitney tests were applied for group comparisons based on variable type and distribution. Pearson and Spearman correlations assessed associations between BMI and both continuous and ordinal variables. Binary logistic regression was used to identify predictors of MAFLD, and ROC curve analysis was used to evaluate diagnostic accuracy. A statistical significance level was set at a p-value < 0.05.

RESULTS

This cross-sectional case-control study included 101 patients with a mean age of 46.10 ± 16.60 years, classified into two groups, MAFLD and non-MAFLD, based on clinical, anthropometric, laboratory, radiological, and non-invasive markers.

Table 1: Demographic parameters in relation to MAFLD

Parameters		**MAFLD (n=90)**		**No MAFLD (n=11)**		**P-value**
		Number	%	Number	%	
Gender N (%)	• Male	16	17.8%	0	0%	0.127(1)
	• Female	74	82.2%	11	100%	
Diabetes N (%)		22	24.4%	2	18.2%	0.645(1)
Hypertension N (%)		33	36.7%	2	18.2%	0.224(1)
Smoking N (%)		5	5.6%	0	0%	0.423(1)
Anthropometric Parameters						
BMI groups x	• Lean (BMI < 25 kg/m^2)	22 (24.4%)		6 (54.5%)		0.035*(1)
	• Obese (BMI ≥ 25 kg/m^2)	68 (75.6%)		5 (45.5%)		
Abdominal obesity		63 (70%)		6 (54.50%)		0.298(1)
Age (years) Median (IQR)		45 (35.75)		33 (27-53)		0.138(2)
Mid-upper arm circumference (cm)		31.95 ± 5.61		32.54 ± 10.71		0.859(3)
Weight (kg)		75.81 ± 18.31		60.32 ± 13.96		0.008*(3)
Height (cm)		159.06 ± 9.92		155.91 ± 9.05		0.318(3)
BMI (kg/m^2)		29.91 ± 6.38		24.68 ± 4.85		0.010*(3)
Waist circumference (cm)		98.28 ± 17.04		86.54 ± 16.30		0.033*(3)

*Significant; (1): Chi-square test, (2): Mann-Whitney test, (3): t-test.

MAFLD cases had significantly higher weight (75.81 ± 18.31 kg vs. 60.32 ± 13.96 kg, p = 0.008), BMI (29.91 ± 6.38 vs. 24.68 ± 4.85 kg/m², p = 0.010), obesity prevalence (75.6% vs. 45.5%, p = 0.035), and waist circumference (98.28 ± 17.04 cm vs. 86.54 ± 16.30 cm, p = 0.033) compared with non-MAFLD participants. **(Table 1).**

Table 2: Laboratory data of our included patients

	MAFLD (n=90)	**No MAFLD (n=11)**	**P-value**
	Median (IQR)	Median (IQR)	
Total serum bilirubin (mg/dl)	0.40 (0.30-0.70)	0.35 (0.30-0.40)	0.395(2)
Direct bilirubin (mg/dl)	0.10 (0.10-0.20)	0.10 (0.099-0.13)	0.091(2)
AST (U/L)	19 (16-27)	17 (16-19)	0.428(2)
ALT (U/L)	18 (16-25)	16 (13-18)	0.112(2)
ALP (U/L)	143 (78.25-198.25)	160 (110-199)	0.362(2)
Serum Albumin (g/dl)	3.80 (3.50-4.125)	3.80 (3.60-4.0)	0.878(2)
GGT (U/L) MWU	59.00 (25.75-88)	25.00 (17-45)	0.024*(2)
Triglyceride (mg/dL)	180 (130.5-250)	110 (90-135)	0.005*(2)
Triglyceride/HDL	10.53 (5.25-17.4)	2.75 (2.40-13.46)	0.025*(2)
Triglyceride/glucose	1.32 (0.83-1.86)	1.06 (0.55-1.33)	0.152(2)
VLDL-C (mg/dL)	36 (26.10-50)	22 (18-27)	0.005*(2)
Non-HDL-C (mg/dL)	151.5 (103.8-193.5)	74 (60-104)	<0.001*(2)
LDL/VLDL	3.39 (2.1064-4.73)	4.45 (1.77-5.93)	0.150(2)
Non-HDL/HDL	7.62 (4.39-15.13)	2.75 (1.44-3.00)	0.003*(2)
Total cholesterol/HDL ratio	8.62 (5.39-16.13)	3.75 (2.44-4)	0.003*(2)
	Mean ± SD	**Mean ± SD**	
Total cholesterol (mg/dL)	178.54 ± 58.76	117.36 ± 43.79	0.001*(3)
HDL (mg/dL)	22.39 ± 14.34	33 ± 19.14	0.028*(3)
LDL (mg/dL)	118.17 ± 44.69	98.64 ± 41.73	0.172(3)
Triglyceride/BMI	6.82 ± 3.43	4.92 ± 1.91	0.012*(3)
Fasting blood glucose (mg/dl)	158.96 ± 73.10	121.77 ± 36.56	0.011*(3)
HbA1c (%)	6.17 ± 1.16	5.08 ± 0.67	< 0.001*(3)
Hemoglobin (g/dl)	12.34 ± 2.11	12.06 ± 1.61	0.676
Platelet count (×10^9/L)	284.68 ± 99.76	279.27 ± 78.07	0.863
MPV (FL)	9.98 ± 1.29	10.14 ± 1.25	0.692
Lymphocyte count (×10^9/L)	2.070 ± 1.005	2.019 ± 0.608	0.873
Neutrophils (%)	60.26 ± 16.52	55.26 ± 14.31	0.340
	Median (IQR)	**Median (IQR)**	
Insulin (mIU/L)	6 (3.475-11.40)	5.40 (2.80-6.0)	0.308(2)
HOMA-IR	11.38 (2.51-40.42)	14.57 (1.24-50.72)	0.970(2)
METS-IR	64.36 (50.29-76.9)	41.69 (32.3-71.87)	0.009*(2)
WBCs (×10^9/L) MWU	7.21 (6.155-9.197)	5.900 (5.05-7.59)	0.045*
Neutrophil count (×10^9/L) MWU	4.83 (2.877-6.18)	3.70 (2.41-4.52)	0.072
Lymphocytes (%) MWU	26.7 (11.95-37.03)	28.80 (24.5-40.7)	0.380

*Significant; (2): Mann-Whitney test, (3): independent t-test. P < 0.05, AST: aspartate transferase; ALT: alanine transaminase; ALP: alkaline phosphatase; GGT: gamma-glutamyl transferase; HDL: high-density lipoprotein; LDL: low-density lipoprotein; VLDL-C: very low-density lipoprotein cholesterol; HOMA-IR: homeostatic model assessment for insulin resistance; METS-IR: metabolic score for insulin resistance; MPV: mean platelet volume.

MAFLD cases had significantly higher GGT levels (59 vs. 25, p = 0.024), total cholesterol (178.54 vs. 117.36 mg/dL, p = 0.001), triglycerides (180 vs. 110 mg/dL, p = 0.005), VLDL-C (36 vs. 22 mg/dL, p = 0.005), non-HDL cholesterol (151.5 vs. 74 mg/dL, p < 0.001), triglyceride/BMI ratio (6.82 vs. 4.92, p = 0.012), METS-IR (64.36 vs. 41.69, p = 0.009), fasting blood sugar (158.96 vs. 121.77 mg/dL, p = 0.011), and HbA1c (6.17% vs. 5.08%, p < 0.001), and WBCs (7.21 vs.5.900 p = 0.045). HDL levels were significantly lower in MAFLD cases (22.39 vs. 33 mg/dL, p = 0.028) (**Table 2**).

Table 3: Inflammatory markers of our included patients

Inflammatory markers and indices	**MAFLD (n=90)**	**No MAFLD (n=11)**	**P-value**
	Median (IQR)	**Median (IQR)**	
Age/platelet index	0.16 (0.12-0.22)	0.11 (0.09-0.24)	0.168(2)
MPV/platelet count	0.035 (0.03-0.05)	0.038 (0.03-0.043)	0.896(2)
C-reactive protein (mg/L)	15.50 (7-30.75)	13 (6.30-23)	0.608(2)
Lymphocyte/monocyte ratio	3.5 (2.03-5.24)	4 (2.98-4.79)	0.366(2)
C-reactive protein/albumin ratio	3.95 (1.69-7.86)	3.513 (1.80-5.75)	0.708(2)
Platelets × neutrophils/lymphocytes	686.95 (354.1-1032.7)	561.69 (298.9-665.63)	0.202(2)
Neutrophil/lymphocyte ratio (NLR)	2.049 (1.194-4.27)	2.232 (0.788-2.64)	0.563(2)
Platelet/lymphocyte ratio (PLR)	11.64 (7.26-20.59)	9.58 (5.71-12.24)	0.377(2)
Platelet count/(monocyte* neutrophil fraction)	6034.3 (4615.3-8573.3)	6164.3 (4716.4-9029.3)	0.939(2)
Adiponectin (µg/L)	13.50 (7.90-22.45)	29.10 (13.45-55.8)	0.029*(2)
	Mean ± SD	**Mean ± SD**	
WBC/MPV ratio	0.83 ± 0.37	0.63 ± 0.20	0.010*(3)
Platelet/albumin ratio	75.41 ± 30.75	73.06 ± 24.49	0.807(3)

Neutrophil and platelet score	**Number**	**%**	**Number**	**%**	
Score 0: Neutrophil count ≤ 7.5 × 10^9/L and platelet count ≤ 400 × 10^9/L	71	78.9%	10	90.9%	0.749(1)
Score 1: Neutrophil count > 7.5 × 10^9/ L or platelet count > 400 × 10^9/L	17	18.9%	1	9.1%	
Score 2: Neutrophil count > 7.5 × 10^9/L and platelet count > 400 × 10^9/L	2	2.2%	0	0%	
Inflammation-based predictive score (IPS) CRP-WBC score					
Score 0: CRP ≤ 10 mg/L and WBC ≤ 11 × 10^9/L	30	33.33%	4	36.4%	0.508(1)
Score 1: CRP ≤ 10 mg/L and WBC > 11 × 10^9/L or CRP > 10 mg/L and WBC ≤ 11 × 10^9/L	48	53.33%	7	63.6%	
Score 2: CRP > 10 mg/L and WBCs > 11 × 10^9/L	12	13.33%	0	0%	
Echocardiography					
Normal	50	55.6%	9	81.8%	0.095(1)
Early diastolic dysfunction	40	44.4%	2	18.2%	

*Significant; (1): Chi-square test, (2): Mann-Whitney test, (3): independent t-test, MPV: mean platelet volume; NLR: neutrophil/lymphocyte ratio; PLR: platelet/lymphocyte ratio; WBC: white blood cells.

MAFLD cases had a significantly higher WBC/MPV ratio than controls (0.83 vs. 0.63, p = 0.010), whereas serum adiponectin was significantly lower (13.50 vs. 29.10, p = 0.029). Other inflammatory markers, including MPV/platelet ratio, LMR, NLR, PLR, CRP, and related indices, showed no significant differences

(p > 0.05). CRP-WBC and neutrophil-platelet scores and echocardiography showed insignificant differences between MAFLD and non-MAFLD groups (p > 0.05) (**Table 3**).

Table 4: FibroScan and non-invasive indices of our included patients

		MAFLD (n=90)		No MAFLD (n=11)		P-value
		Number	%	Number	%	
Liver stiffness measurement (LSM)	F0	32	35.6%	7	63.6%	0.018*(1)
	FI	42	46.7%	1	9.1%	
	F2	12	13.3%	2	18.2%	
	F3	4	4.4%	0	0%	
	F4	0	0%	1	9.1%	
Controlled attenuation parameter (CAP)	S0	0	0%	11	100%	< 0.001*(1)
	S1	13	14.4%	0	0%	
	S2	48	53.3%	0	0%	
	S3	29	32.2%	0	0%	
BARD score	0	6	6.7%	0	0%	0.039*(1)
	1	5	5.6%	0	0%	
	2	29	32.2%	9	81.8%	
	3	33	36.7%	1	9.1%	
	4	17	18.9%	1	9.1%	
Liver fibrosis index		Median (IQR)		Median (IQR)		
FIB-4		0.70 (0.50-1.20)		0.50 (0.50-1.30)		0.547(2)
		Mean ± SD		Mean ± SD		
Hepatic steatosis index [t]		40.52 ± 9.34		34.018 ± 5.17		0.026*(3)

*Significant; (1) chi-square test; (2) Mann-Whitney test; (3) independent t-test; BARD score: BMI, AST to ALT ratio; and type 2 diabetes mellitus; FIB-4: Fibrosis-4.

In this study, patients with MAFLD (n = 90) showed significantly more advanced liver involvement than patients without MAFLD (n = 11). Liver stiffness stages were significantly higher in MAFLD (P = 0.018), with FI being the most common stage (46.7%) and F4 absent (0%), whereas in non-MAFLD, F0 was predominant (63.6%) and F3 was absent (0%). CAP scores were significantly elevated in MAFLD patients (P < 0.001), with S2 being the most frequent (53.3%) and S0 absent (0%), whereas all non-MAFLD patients had S0 (100%). The BARD score was significantly higher in MAFLD patients (P = 0.039), with a score of 3 most prevalent (36.7%) and 0 least common (6.7%); in contrast, non-MAFLD patients mostly scored 2 (81.8%) with no scores of 0 or 1. The Hepatic Steatosis Index was significantly elevated in MAFLD patients (40.52 ± 9.34 vs. 34.02 ± 5.17, P = 0.026), while no significant difference was observed in FIB-4 values (P = 0.547) (**Table 4**).

Table 5: Correlation between BMI with METS-IR, triglycerides, total cholesterol, LDL, and triglyceride/glucose ratio

Parameters	BMI (kg/m^2)	
Pearson correlation	r[1]	P-value
Hepatic steatosis index	0.755	<0.001
METS-IR	0.547	<0.001
HbA1c (%)	0.393	<0.001
Triglyceride (mg/dl)	0.352	<0.001
VLDL-C (mg/dl)	0.352	<0.001
Total cholesterol (mg/dl)	0.261	0.008
Non-HDL cholesterol (mg/dl)	0.257	0.009

LDL/VLDL ratio	-0.224	0.025
Adiponectin (µg/L)	-0.242	0.028
Triglyceride/glucose ratio	0.212	0.033
LDL (mg/dl)	0.209	0.036
Spearman correlation	**r^2**	**P-value**
BARD score	0.637	<0.001
Controlled attenuation parameter (CAP)	0.386	<0.001
Liver Stiffness Measurement (LSM)	0.280	0.005

r^1: Pearson correlation coefficient, r^2: Spearman correlation coefficient; METS-IR: metabolic score for insulin resistance; HbA1c: hemoglobin A1c; VLDL-C: very low-density lipoprotein cholesterol; HDL: high-density lipoprotein**;** LDL: low-density lipoprotein; BARD score (BMI, AST/ALT ratio, Diabetes mellitus).

BMI was significantly positively correlated with HSI (r = 0.755, p < 0.001), METS-IR (r = 0.547, p < 0.001), triglycerides, VLDL-C (r = 0.352, p < 0.001), HbA1c (r = 0.393, p < 0.001), total cholesterol (r = 0.261, p = 0.008), LDL (r = 0.209, p = 0.036), non-HDL-C (r = 0.257, p = 0.009), and TG/glucose ratio (r = 0.212, p = 0.033).
It also correlated positively with BARD score (r = 0.637, p < 0.001), CAP (r = 0.386, p < 0.001), and LSM (r = 0.280, p = 0.005).
The BMI negatively correlated with the LDL/VLDL ratio (r = -0.224, p = 0.025) and adiponectin (r = -0.242, p = 0.028) **(Table 5)**.

Table 6: Results of univariable and multivariable logistic regression analysis of factors affecting MAFLD

Predictor variable	**Univariable analysis**		**Multivariable analysis**	
	Odds ratio (95% CI)	**P-value**	**Odds ratio (95% CI)**	**P-value**
Non-HDL-C (mg/dL)	1.031 (1.011-1.051)	0.003*	1.029 (1.008-1.050)	0.006*
Total cholesterol (mg/dl)	1.027 (1.009-1.046)	0.004*	1.026 (1.007-1.045)	0.008*
HbA1c (%)	5.154 (1.599-16.614)	0.006*	6.579 (1.203-35.98)	0.030*
Weight (Kg)	1.064 (1.015-1.116)	0.009*	1.045 (0.988-1.104)	0.124
BMI (Kg/m^2)	1.187 (1.032-1.364)	0.016*	1.027 (0.882-1.197)	0.730
Adiponectin (µg/L)	0.964 (0.935-0.993)	0.017*	0.979 (0.947-1.012)	0.215
Hepatic steatosis index	1.156 (1.026-1.303)	0.018*	1.162 (1.008-1.341)	0.039*
VLDL-C mg/dL)	1.063 (1.007-1.121)	0.027*	1.048 (0.994-1.105)	0.083
Triglycerides (mg/dL)	1.012 (1.001-1.023)	0.027*	1.007 (0.997-1.018)	0.168
GGT	1.030 (1.003-1.058)	0.030*	1.034 (1.004-1.064)	0.026*
HDL (mg/dL)	0.959 (0.922-0.997)	0.035*	0.987 (0.943-1.032)	0.558
Waist circumference (cm)	1.044 (1.003-1.088)	0.036*	1.030 (0.985-1.076)	0.193
NFS	2.403 (1.033-5.591)	0.042*	2.405 (1.001-5.775)	0.050
F1	9.188 (1.075-78.493)	0.043*	7.545 (0.855-66.615)	0.069
Overweight or obesity	3.709 (1.031-13.347)	0.045*	2.222 (0.533-9.269)	0.273
METS-IR	1.039 (1.00-1.080)	0.049*	1.008 (0.971-1.048)	0.671
WBCs/MPV ratio	16.886 (0.93-306.626)	0.056	8.824 (0.357-217.826)	0.183

*: significant; BMI: body mass index; HDL: high-density lipoprotein; VLDL-C: very low-density lipoprotein cholesterol; NFS: NAFLD fibrosis score; HOMA-IR: Homeostatic Model Assessment for Insulin Resistance; GGT: gamma-glutamyl transferase; LSM: liver stiffness measurement.

Univariate logistic regression analysis showed that non-HDL cholesterol, total cholesterol, HbA1c, weight, BMI, hepatic steatosis index, VLDL-cholesterol, triglyceride, GGT, waist circumference, NFS, and F1 as

measured by liver stiffness FibroScan, obesity, and METS-IR may be independent risk factors for the occurrence of MAFLD. Serum adiponectin and HDL levels may be protective factors against MAFLD. Multivariable logistic regression analysis showed that non-HDL-C, total cholesterol, GGT, and HbA1c may be risk factors for MAFLD **(Table 6)**.

Table 7: ROC curve analysis for the diagnostic accuracy of laboratory parameters, ratios, and liver fibrosis indices in diagnosing of liver fibrosis in MAFLD

Parameter	AUC	Cutoff	Sensitivity	Specificity	PPV	NPV	P-value	95% CI		Accuracy
								Lower	Upper	
HbA1c	0.839	5.05 (%)	84.44%	81.82%	97.44%	39.13%	<0.001*	0.709	0.970	84.16%
HSI	0.767	32.31	87.78%	63.64%	95.18%	38.89%	0.004*	0.621	0.912	85.15%
BMI	0.762	22.89 kg/m^2	83.33%	54.55%	93.75%	28.57%	0.005*	0.552	0.928	80.20%
METS-IR	0.740	44.299	85.56%	72.73%	96.25%	38.10%	0.009*	0.552	0.928	84.16%
Adiponectin	0.724	28.75 µg/l	86.49%	55.56%	94.12%	33.33%	0.029*	0.551	0.898	83.13%

*: significant; AUC: area under curve; CI: Confidence interval; PPV: positive predictive value; NPV: negative predictive value; BMI: body mass index; HDL: high-density lipoprotein; VLDL-C: very low-density lipoprotein-cholesterol; METS-IR: metabolic score for insulin resistance; HSI: hepatic steatosis index; NFS: NAFLD fibrosis score; GGT: gamma-glutamyl transferase.

Several laboratory parameters demonstrated significant diagnostic value in ROC curve analysis for diagnosing liver fibrosis in MAFLD patients ($P < 0.05$). HbA1c showed the highest diagnostic performance with an AUC of 0.839 at a cutoff of 5.05%, yielding 84.44% sensitivity, 81.82% specificity, 97.44% PPV, and 39.13% NPV ($P < 0.001$), with 84.16% accuracy. The Hepatic Steatosis Index (HSI) had an AUC of 0.767 at a cutoff of 32.31, sensitivity of 87.78%, and specificity of 63.64% ($P = 0.004$), with 85.15% accuracy. BMI had an AUC of 0.762 at a cutoff of 22.889 kg/m^2, sensitivity of 83.33%, specificity of 54.55%, and accuracy of 80.20% ($P = 0.005$). METS-IR also showed significance with an AUC of 0.740 at a cutoff of 44.299, sensitivity of 85.56%, specificity of 72.73%, and 84.16% accuracy ($P = 0.009$). Adiponectin was inversely associated with MAFLD, showing an AUC of 0.724 at a cutoff of 28.75 µg/L, with 86.49% sensitivity, 55.56% specificity, and 83.13% accuracy ($P = 0.029$) **(Table 7)**.

DISCUSSION

MAFLD, characterized by hepatic steatosis ≥ 5% and metabolic abnormalities, is a major health concern affecting both obese and lean individuals because of its association with systemic metabolic dysfunction. It has a stronger prognostic value than NAFLD, particularly in predicting liver/non-liver mortality and fibrosis **(Nguyen et al., 2021).** This study aimed to assess MAFLD prevalence and the role of serum adiponectin as a noninvasive fibrosis marker.In the current study, we found no significant difference in diabetes mellitus and hypertension incidence between MAFLD and non-MAFLD groups This suggests that, within our study population, MAFLD may not have been a strong independent predictor for these metabolic conditions, or that other confounding factors might have influenced the results, also non-MAFLD group in our study including two subjects with diabetes mellitus and two with hypertension. However, **Liang et al. (2022)** reported that fatty liver patients had a higher risk of developing diabetes than non-fatty liver patients did. They reported MAFLD was associated with higher risks of incident diabetes (risk ratio [RR] 2.08; 95% CI, 1.72-2.52). emphasizing the need for early management of metabolic disorders. This discrepancy may be due to differences in population characteristics, study design, follow-up duration, or diagnostic criteria used in the two studies.Our study revealed significantly higher BMI, waist circumference, and weight in MAFLD patients and non-MAFLD patients. The mid-upper arm circumference (MUAC) showed no significant difference, with nearly equal means in both groups. that agree with, **Hosseini et al. (2024)** reported significant

differences in anthropometric measures, including BMI (30.26 vs. 32.9 kg/m^2; P < 0.0001), weight (92 vs. 83 kg; P < 0.0001), height (174 vs. 159 cm; P < 0.0001), wrist (18.50 vs. 17.7 cm; P < 0.0001), waist (103 vs. 109 cm; P < 0.0001), and hip circumferences (105.9 vs. 111.0; P < 0.0001) in MAFLD patients .We found no significant differences in liver function test results between the MAFLD and non-MAFLD groups that align with **Forlano et al.(2021),** who found that Liver function tests might both underestimate and overestimate MASH-associated liver disease. In our study, MAFLD patients had significantly higher total cholesterol, median triglycerides, VLDL-C, non-HDL cholesterol, and triglyceride/BMI ratios, with significantly lower HDL levels. These results are consistent with **Wu et al. (2016),** who demonstrated more prominent dyslipidemia in MAFLD patients, with multiple abnormal lipid markers linked to MAFLD occurrence. The total cholesterol/HDL-C ratio was 3.7 ± 1.0 (mean: 4.0 ± 0.9; women: 3.2 ± 0.8). The triglyceride/HDL-C ratio was 2.6 ± 2.3 in all cases, while it was 3.0 ± 2.5 among men and 1.7 ± 1.4 among women. Similarly, previous studies, reported a 1.446-fold increased risk of MAFLD in patients with high total cholesterol **(Hamaguchi et al., 2007; Leite et al., 2009)**.In our study, MAFLD patients had significantly higher median METS-IR, mean fasting blood glucose, and HbA1c than non-MAFLD individuals. However, the median insulin was higher among MAFLD patients, but not significant. The median HOMA-IR score was lower in MAFLD patients, but the difference was not statistically significant. **Liao et al. (2021)** found a significant correlation between insulin metabolism and metabolic syndrome components, partially consistent with our findings for glucose parameters but not for HOMA-IR. Serum HOMA-IR level was higher in metabolically unhealthy non-obesity (β = 0.42, 95% CI: 0.21–0.64), metabolically healthy obesity (β = 0.68, 95% CI: 0.36–1.00), and metabolically unhealthy obesity (β = 0.69, 95% CI: 0.46–0.91) twins compared with their metabolically healthy non-obesity counterparts.Our findings showed that MAFLD patients had a significantly higher WBC/MPV ratio, suggesting systemic inflammation. However, other inflammatory and hematologic indices such as MPV/platelet, LMR, NLR, PLR, CRP, platelet × neutrophil/lymphocyte ratio, platelet/albumin ratio, RBC indices, prothrombin time, and INR were not significant. Similarly, the neutrophil-platelet score distribution showed no significant difference between MAFLD patients and controls (p = 0.749), despite the higher scores in MAFLD patients. Our data align with those of **Lee et al., 2010, who** found that higher WBCs were associated with NAFLD**.** The corresponding adjusted OR (95% CI) for the highest quartile of WBC count was 1.84 (1.35–2.51) in men. Among women, the prevalence risk of NAFLD for the highest quartile of WBC count was 2.74 (1.68–4.46) after adjustment for the same co-variables. **Choe and Kang (2024)** found WBC/MPV and PLR to be associated with MAFLD in non-obese individuals, but not in obese individuals. WBC/MPV was significantly higher in subjects with NAFLD, while PLR was significantly lower in subjects with NAFLD. In the analysis restricted to the non-obese (BMI < 25 kg/m^2) population without MS, both WBC/MPV and PLR were independently associated with NAFLD in the non-obese, metabolically healthy group; the adjusted odds ratios for WBC/MPV and PLR were 2.055 and 0.660, respectively. **Chung et al., 2016,** analyzed the relationship between the risk of developing NAFLD and the WBC count. The prevalence of NAFLD increased steadily with increasing WBC counts after adjustment for age and BMI [odds ratio (OR) 2.44, 95% confidence interval (CI) = 1.49–4.00 for women and OR 2.42, 95% CI = 1.61–3.63 for men].In contrast to our non-significant findings, **Xie et al. (2017–2018)** found that patients with higher high platelet/albumin ratios had higher prevalence of Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD). The study involved 3287 participants, of whom 873 (26.5%) were diagnosed with MASLD. The platelet/albumin ratio level in the MASLD group was significantly higher than in the non-MASLD group (5.92 ± 1.71 vs. 5.64 ± 1.74; P = 0.003). Multivariate logistic regression revealed that a high platelet/albumin ratio was identified as an independent risk factor for MASLD (OR = 2.58, 95% CI: 1.26–5.27, P = 0.03).**Chen et al., 2022,** investigated the role of inflammatory markers in the prediction of NAFLD. They found that cases with NAFLD were associated with significantly higher PLR (104.47 vs 129.92; P < 0.0001), lymphocyte count (2.180 vs 1.750; P < 0.0001), and WBC (6.830 vs 5.760; P = 0.037) than non-NAFLD cases.Our **study the first study to use** CRP-WBC scores as a predictor of inflammation that found no significant difference in CRP-WBC scores.Our study revealed that MAFLD patients had a

significantly lower median adiponectin level (13.5 μg/L) versus non-MAFLD individuals (29.1 μg/L) ($p = 0.029$). This finding aligns with **Pan et al. (2023),** who observed higher adiponectin levels in those without MAFLD (3.6 vs. 2.17; $P < 0.0001$) than in cases with MAFLD and a negative association with MAFLD.
Our study revealed that MAFLD patients had significantly higher non-invasive fibrosis and steatosis indices, including the NAFLD Fibrosis Score and Hepatic Steatosis Index (HSI). However, the mean Fatty Liver Index (FLI), APRI, and FIB-4 scores were higher but not significant. The median GGT level was significantly elevated in the MAFLD group, whereas the AST/ALT and ALT/AST ratios showed no significant differences. **Sviklāne et al. (2018)** found that FLI correlated with intrahepatic fat ($r = 0.510$, $P < 0.01$), while HSI did not ($r = 0.240$, $P > 0.05$).Our results are partially aligned with those of **Hemida et al. (2021),** who reported higher FIB-4 and NAFLD Fibrosis Scores in diabetics with NAFLD, with the mean FIB-4 being 2.22 ± 0.99 and the mean NAFLD Fibrosis Score being -0.49 ± 1.34. **Park et al. (2022)** recommended FIB-4 as the primary screening tool for advanced fibrosis in MAFLD. They found higher FIB-4 among MAFLD cases, with increased FIB-4 and severity of MAFLD. FIB-4 had a sensitivity of 66.6%, specificity of 77.6%, PPV of 67%, and NPV of 98.9% for predicting MAFLD.**Xuan et al. (2024)** identified a high ALT/AST ratio as a risk factor for MAFLD and liver fibrosis, contrasting our non-significant results. In their study, all three multivariable logistic regression models showed positive correlations between ALT/AST and NAFLD the risk; model 1 (OR = 9.201, 95% CI: 7.536, 11.234), model 2 (OR = 9.732, 95% CI: 7.853, 12.060), and model 3 (OR = 3.648, 95% CI: 2.827, 4.706).**Chen et al. (2021)** found that ALT and GGT levels were dose-dependently linked to NAFLD and metabolic syndrome. Both GGT and ALT values were positively associated with the degree of hepatic steatosis (GGT beta = 0.093, $p = 0.004$; ALT beta = 0.332, $p < 0.001$) or fat component (GGT beta = 0.103, $p < 0.001$; ALT beta = 0.504, $p < 0.001$) using the linear regression analysis. For hepatic fibrosis, the GGT value was positively associated with fibrosis degree according to the NAFLD fibrosis score by linear regression analysis (beta = 0.056, $p = 0.006$).Our findings are supported by **Priego-Parra et al. (2024),** who found that the HSI showed a diagnostic performance with an AUC of 0.80. A cutoff point of 39.9 was established for the HSI, with a sensitivity of 63%, specificity of 74%, PPV of 73%, and NPV of 64%. Also, similar to our study, **Vamja et al. (2024)** supported FLI as useful tools for identifying MAFLD. They reported that at a cut off value of ≥ 60, FLI had a sensitivity of 96% and specificity of 92.5% for identifying MAFLD.
Our study demonstrated a significant association between MAFLD and both liver fibrosis and steatosis grades assessed by LSM and CAP, respectively. This agrees with **Abo El Soud et al. (2021),** who found higher FibroScan values among cases with NAFLD, which was 11 ± 3 dB/m, compared to 7 ± 1 dB/m in patients with hepatic steatosis only and 4 ± 1 dB/m among controls ($P < 0.0001$).Our study findings revealed a strong link between obesity and metabolic dysfunction, with higher BMI associated with increased levels of HSI, METS-IR, triglycerides, VLDL-C, HbA1c, total cholesterol, LDL, non-HDL-C, and the triglyceride/glucose ratio. Conversely, BMI negatively correlates with the LDL/VLDL ratio and serum adiponectin levels, suggesting that these protective markers decrease with increased BMI. In agreement, **Shrestha et al. (2021)** found obese NAFLD patients (BMI ≥ 25 kg/m^2) were 3.7 times more likely to have severe steatosis than non-obese individuals.Our study also confirmed that HbA1c is an independent risk factor for MAFLD, consistent with **Masroor et al. (2021).** Similarly, other independent risk factors include, non-HDL cholesterol, total cholesterol, and GGT, with higher serum adiponectin and HDL-C levels appearing protective. These associations were supported by **Pan et al. (2023),** who found adiponectin levels inversely related to MAFLD, as lower levels were found in non-MAFLD cases compared to the MAFLD group.In our study, ROC curve analysis identified non-HDL cholesterol at > 77.5 mg/dL (sensitivity 91.11%, specificity 72.73%), non-HDL/HDL ratio > 3.01, and HbA1c > 5.05% as the most effective markers for MAFLD detection, with excellent sensitivity, specificity, and AUC. HSI > 32.31 and total cholesterol > 112.5 mg/dL also demonstrated good diagnostic value. Triglycerides, VLDL-C, adiponectin, BMI, and METS-IR showed moderate performance, while GGT and NAFLD fibrosis score had lower diagnostic accuracy. Composite markers like TG/HDL and WBC/MPV ratios had limited utility in ruling out MAFLD.

In our study, logistic regression identified non-HDL cholesterol, total cholesterol, HbA1c, weight, BMI, HSI, VLDL-C, triglycerides, GGT, waist circumference, NFS, liver stiffness (≥F1), obesity, and METS-IR as significant predictors of MAFLD. These findings are consistent with **Khamseh et al. (2021),** who found a strong link between triglycerides and MAFLD, which was higher among MAFLD cases (143.5 vs 106.5; $P < 0.0001$). Also, LDL cholesterol was significantly higher among MAFLD cases (105.4 vs. 95.6; $P = 0.008$), and **Liu et al. (2019)** found METS-IR significantly associated with hypertension in normal-weight individuals. The OR for hypertension in the normal BMI group in the highest quartile of METS-IR was 2.884 (95% CI: 2.468-3.369) in the total sample, 1.915 (95% CI: 1.614-2.271) in females, and 2.083 (95% CI: 1.717-2.527) in males.**Sun et al., 2016,** found that the FIB-4 index group, pooled sensitivity and specificity with 95% confidence interval (CI), and the area under the ROC (AUROC) were 0.844 (0.772–0.901), 0.685 (0.654–0.716), and 0.8496 ± 0.0680, respectively, at a cut-off of 1.30. At a threshold of 3.25, the same parameters were 0.38 (0.30–0.47), 0.96 (0.95–0.98), and 0.8445 ± 0.0981. At a cut-off of -1.455, values were 0.77 (0.69–0.84), 0.70 (0.67–0.73), and 0.8355 ± 0.0667, respectively. At a 0.676 cut-off, pooled sensitivity and specificity with 95% CI were 0.27 (0.19–0.35) and 0.98 (0.96–0.98), respectively, and the AUROC was 0.647 ± 0.2208. In our study, APRI was not significantly associated with steatosis severity. However, **Rigamonti et al. (2022)** found a significant association between APRI ($P < 0.0001$) and NAFLD, indicating that APRI can be used as a predictor for NAFLD. **Al Danaf et al. (2022)** suggested a strong correlation between APRI, FIB-4, AST/ALT ratio, and fibrosis stages, recommending their use alongside FibroScan to avoid biopsy. Similarly, **Amernia et al. (2021)** considered APRI the best index for predicting advanced fibrosis. The optimal cut-off of APRI was 0.702 for this purpose, with a sensitivity of 84.1%, specificity of 88.2%, PPV of 66.1%, NPV of 95.3%, and DA of 87.3%.Our study supports the role of non-invasive laboratory markers, particularly non-HDL cholesterol, HbA1c, and the non-HDL/HDL ratio, as simple, accessible tools for identifying MAFLD, especially when imaging is not available. These findings, together with multivariable analysis and ROC assessment, underscore the importance of integrating metabolic, anthropometric, and biochemical parameters for early MAFLD diagnosis and stratification.

This study has several limitations due to its small sample size, cross-sectional design, single-center nature, lack of histological confirmation, potential uncontrolled confounding factors, limited external validation, and possible variability in adiponectin measurements, which were based on a single ELISA test without repeat sampling.

CONCLUSION:

Non-HDL cholesterol, HbA1c, and the non-HDL/HDL ratio demonstrated the strongest diagnostic utility for MAFLD, and their integration into screening protocols could facilitate earlier interventions and potentially reduce MAFLD-related complications. Elevated levels of serum adiponectin and HDL-cholesterol were identified as protective factors.

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