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Metabolic-associated fatty liver disease in patients undergoing hemodialysis: A cross-sectional study of prevalence and diagnostic accuracy of the triglyceride–glucose index

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Abstract. *Metabolic dysfunction-associated fatty liver disease (MAFLD) is increasingly recognized in patients with metabolic and renal disorders; however, data in hemodialysis populations remain limited. This study assessed the prevalence of MAFLD in patients receiving maintenance HD and identified factors associated with its presence.*
Methods. A total of 95 patients with end-stage kidney disease (ESKD) receiving maintenance HD were included in this cross-sectional study conducted at Ain Shams University Hospital from January to September 2024. Clinical assessment, abdominal ultrasonography, and laboratory investigations were performed. The triglyceride-glucose (TyG) index, non-alcoholic fatty liver disease (NAFLD) fibrosis score, and fibrosis-4 (FIB-4) score were calculated.
Results. MAFLD was identified in 21 of 95 patients (22.1%). Compared with patients without MAFLD, those with MAFLD had significantly higher body mass index (35.14 ± 3.09 vs. 25.51 ± 4.74 kg/m^2), WC (116.95 ± 6.74 vs. 100.24 ± 9.56 cm), CRP (20.38 ± 5.24 vs. 4.59 ± 2.49 mg/L), ALT, AST, TC, LDL-C, triglycerides, TyG index (9.20 ± 0.34 vs. 8.63 ± 0.27), NAFLD fibrosis score, and FIB-4 score (all $P < 0.05$). Hemoglobin and albumin were substantially lower in the MAFLD group. The TyG index showed high discrimination for MAFLD, with an AUC of 0.906 (95% CI, 0.815–0.998); at a cutoff >8.88, sensitivity was 95.2% and specificity was 82.4%.
Conclusions. MAFLD is common among patients receiving hemodialysis and is associated with metabolic and inflammatory disturbances. The TyG index showed promising discriminative performance for identifying MAFLD in this population. However, the findings are exploratory and require validation in larger, multicenter studies.

Keywords: *fatty liver, triglyceride-glucose index, insulin resistance, end-stage kidney disease, hemodialysis, liver fibrosis.*

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Метаболічно-асоційована жирова хвороба печінки у пацієнтів, які лікуються гемодіалізом: поширеність та дискримінаційна здатність індексу тригліцериди–глюкоза

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Резюме. *Метаболічно-асоційована жирова хвороба печінки (МАЖХП), часто поєднується з ожирінням, інсулінорезистентністю та іншими метаболічними порушеннями. Водночас дані щодо її поширеності серед пацієнтів, які лікуються гемодіалізом (ГД), залишаються обмеженими. Метою дослідження було оцінити поширеність МАЖХП у ГД пацієнтів та визначити чинники, пов'язані з її наявністю.*

Методи. До поперечного дослідження, проведеного в Університетській лікарні Айн-Шамс із січня до вересня 2024 року, включено 95 пацієнтів з термінальною стадією хронічної хвороби нирок, які лікувались ГД. Усім пацієнтам проведено клінічне обстеження, ультразвукове дослідження органів черевної порожнини та лабораторні дослідження. Розраховано індекс тригліцериди–глюкоза (TyG), шкалу фіброзу неалкогольної жирової хвороби печінки (NFS) та індекс FIB-4.

Результати. МАЖХП виявлено у 21 із 95 пацієнтів (22,1%). Порівняно з пацієнтами без МАЖХП, пацієнти з МАЖХП мали вищий індекс маси тіла (35,14 ± 3,09 проти 25,51 ± 4,74 кг/м^2), більшу окружність талії (116,95 ± 6,74 проти 100,24 ± 9,56 см), вищі рівні С-реактивного білка (20,38 ± 5,24 проти 4,59 ± 2,49 мг/л), аланінамінотрансферази, аспартатамінотрансферази, загального холестерину, холестерину ліпопротеїнів низької щільності та тригліцеридів. Значення індексу TyG (9,20 ± 0,34 проти 8,63 ± 0,27), шкали NFS та індексу FIB-4 також були вищими у пацієнтів із МАЖХП (усі $p < 0,05$). Рівні гемоглобіну й альбуміну були суттєво нижчими у групі МАЖХП. Індекс TyG продемонстрував високу дискримінаційну здатність щодо наявності МАЖХП: площа під ROC-кривою становила 0,906 (95% ДІ 0,815–0,998). За порогового значення >8,88 чутливість становила 95,2%, специфічність – 82,4%.

Висновки. МАЖХП виявлено більш ніж у п'ятої частини ГД пацієнтів, а її наявність асоціювалась з метаболічними та запальними порушеннями. Індекс TyG продемонстрував перспективну дискримінаційну здатність для виявлення МАЖХП у цій популяції. Отримані результати мають описовий характер і потребують підтвердження у більших багатоцентрових дослідженнях.

Ключові слова: *жирова хвороба печінки, індекс тригліцериди–глюкоза, інсулінорезистентність, термінальна стадія хронічної хвороби нирок, гемодіаліз, фіброз печінки.*

Introduction. Steatotic liver disease is one of the most common chronic liver disorders worldwide and is closely linked to obesity, insulin resistance, and other cardiometabolic abnormalities [1]. Chronic kidney disease (CKD) shares multiple pathogenic mechanisms with steatotic liver disease, including insulin resistance, chronic low-grade inflammation, oxidative stress, endothelial dysfunction, and metabolic dysregulation [2]. Insulin resistance promotes hepatic lipid accumulation and systemic metabolic imbalance, while chronic inflammation and oxidative stress contribute to both hepatic injury and kidney dysfunction [3]. Emerging evidence also highlights the gut–liver–kidney axis, in which alterations in gut microbiota and increased intestinal permeability may promote systemic inflammation and metabolic disturbances affecting both the liver and kidneys [4].

Hepatic steatosis has also been associated with CKD progression [2]. However, patients with end-stage kidney disease (ESKD) receiving maintenance hemodialysis (HD) remain underrepresented in the literature. This population has complex metabolic alterations, including protein-energy wasting, chronic inflammation, uremia-related metabolic disturbances, and fluid shifts, which may influence hepatic fat accumulation and complicate the assessment of hepatic steatosis and fibrosis [5].

Therefore, this study aimed to assess the prevalence of metabolic-associated fatty liver disease (MAFLD) in patients receiving maintenance HD, identify factors associated with its presence, and evaluate the discriminative performance of the triglyceride–glucose (TyG) index for MAFLD.

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Patients and methods. *Study design and setting*. This cross-sectional investigation was performed on 95 cases with ESKD at the HD unit of Ain Shams University Hospital from January 2024 to September 2024. The study was approved by the Ethical Committee of Ain Shams University Hospital hemodialysis unit (approval code: FMASU MS 379/2023). Before enrolment, written informed consent was secured from all cases or their relatives.

Eligibility criteria. Eligible cases were aged 18 to 60 years, of both sexes, and were on regular maintenance HD for more than 6 months, receiving three sessions per week, each lasting 4 hours. Cases were excluded if they had marked alcohol intake (≥20 g/day in women and ≥30 g/day in men), hepatitis C or B virus positivity, other chronic liver diseases, concurrent active infection, malignancy, or other decompensated organ failure.

Clinical, laboratory, and imaging assessment. All included cases underwent detailed history taking and clinical examination. Clinical assessment included measurement of weight, height for calculation of BMI, WC, and blood pressure, in addition to documentation of CKD etiology and duration. Abdominal ultrasonography was performed as part of the imaging assessment using standardized criteria for hepatic steatosis, including increased hepatic echogenicity relative to the renal cortex, vascular blurring, and deep attenuation, as assessed by an experienced radiologist. Laboratory investigations included CBC, CRP, serum creatinine, blood urea, urea reduction ratio, ALT, AST, serum albumin, FBS, and full lipid profile. Calculated parameters included the TyG index and the NAFLD fibrosis score.

Diagnostic criteria. The current multisociety nomenclature uses metabolic dysfunction-associated steatotic liver disease (MASLD) [1]. However, the present study was designed and analyzed according to the metabolic dysfunction-associated fatty liver disease (MAFLD) criteria proposed in 2020 [6]; therefore, MAFLD terminology is retained throughout the manuscript. MAFLD was defined as ultrasonography-confirmed hepatic steatosis together with overweight/obesity, type 2 diabetes mellitus, or metabolic dysregulation [6]. Metabolic dysregulation was defined by the presence of at least two additional abnormalities, including elevated WC, hypertension, hypertriglyceridemia, impaired fasting glucose, low HDL cholesterol, insulin resistance, or subclinical inflammation [6].

Sample size calculation. The required sample size was calculated using a single-proportion formula based on an expected MAFLD prevalence of 43.4% as reported by Wong et al. [7], with a confidence level of 95% and a margin of error of 10%. Accordingly, a minimum sample size of 95 cases was required to achieve adequate statistical precision.

Statistical methods. Data were analyzed using SPSS version 26 (IBM Corp., Armonk, NY, USA). Quantitative variables were reported as mean ± SD when normally distributed and as median (range) when not normally distributed, while qualitative variables were summarized as frequency and percentage. Between-group comparisons were performed using the unpaired Student's t-test for quantitative variables and the Chi-square test or Fisher's exact test for qualitative variables, as appropriate. ROC curve analysis was used to assess the discriminative performance of the studied markers, with the area under the curve (AUC) used to quantify discrimination. Univariable logistic regression and separate age- and sex-adjusted logistic regression models were used to assess associations with MAFLD. Each adjusted model included one studied predictor together with age and sex. Odds ratios (ORs) with 95% confidence intervals (CIs) were calculated. A two-tailed P value <0.05 was considered statistically significant.

Results. Patients with MAFLD had significantly higher weight, BMI, and WC than those without MAFLD. They also had lower hemoglobin and albumin levels and higher CRP, ALT, AST, TC, LDL-C, TG, and TyG index values (Table 1).

Fibrosis-related indices, including the NAFLD fibrosis score and FIB-4 score, were also higher in patients with MAFLD (Table 1).

Table 1

Demographic, clinical, anthropometric, and laboratory characteristics of the studied patients according to MAFLD status

Variable	Data presentation	Total (n = 95)	MAFLD		P-value
			Yes (n = 21)	No (n = 74)	
Age (years)	Mean ±SD	42.71 ±14.78	46.76 ±13.44	41.55 ±15.03	0.16
Sex					
Males	n (%)	47 (49.5%)	11 (52.4%)	36 (48.6%)	0.76
Females	n (%)	48 (50.5%)	10 (47.6%)	38 (51.4%)	0.76
Height (cm)	Mean ±SD	166.69 ±7.88	167.14 ±5.03	166.57 ±8.54	0.7
Weight (kg)	Mean ±SD	76.92 ±16.93	97.95 ±10.01	70.95 ±13.35	<0.001*
BMI (kg/m^2)	Mean ±SD	27.64 ±5.97	35.14 ±3.09	25.51 ±4.74	<0.001*
WC (cm)	Mean ±SD	103.94 ±11.37	116.95 ±6.74	100.24 ±9.56	<0.001*

Continuation of Table 1

Variable	Data presentation	Total (n = 95)	MAFLD		P-value
			Yes (n = 21)	No (n = 74)	
SBP (mmHg)	Mean ±SD	135.16 ±26.00	126.67 ±28.17	137.57 ±25.02	0.09
DBP (mmHg)	Mean ±SD	86.11 ±13.15	82.86 ±11.89	87.03 ±13.42	0.2
Family history of CKD	n (%)	22 (23.2%)	5 (23.8%)	17 (23.0%)	1
HTN	n (%)	8 (8.4%)	2 (9.5%)	6 (8.1%)	1
DM	n (%)	12 (12.6%)	3 (14.3%)	9 (12.2%)	0.72
CAD	n (%)	5 (5.3%)	0 (0.0%)	5 (6.8%)	0.58
HGB (g/dL)	Mean ±SD	10.23 ±0.88	9.26 ±0.41	10.51 ±0.78	<0.001*
WBCs ($\times 10^3/\mu L$)	Mean ±SD	6.69 ±1.56	6.86 ±1.49	6.65 ±1.58	0.59
Platelets ($\times 10^3/\mu L$)	Mean ±SD	265.37 ±60.86	264.29 ±61.28	265.68 ±61.16	0.93
CRP (mg/L)	Mean ±SD	8.08 ±7.35	20.38 ±5.24	4.59 ±2.49	<0.001*
Creatinine (mg/dL)	Mean ±SD	8.50 ±0.93	8.74 ±0.75	8.43 ±0.96	0.17
Urea (mg/dL)	Mean ±SD	105.61 ±16.40	104.10 ±14.01	106.04 ±17.08	0.63
ALT (U/L)	Mean ±SD	29.80 ±7.86	33.19 ±9.66	28.84 ±7.05	0.02*
AST (U/L)	Mean ±SD	38.24 ±15.74	62.90 ±9.96	31.24 ±8.21	<0.001*
TC (mg/dL)	Mean ±SD	210.73 ±35.62	251.90 ±30.60	199.04 ±27.41	<0.001*
LDL (mg/dL)	Mean ±SD	111.68 ±33.82	151.90 ±30.60	100.27 ±24.88	<0.001*
HDL (mg/dL)	Mean ±SD	84.40 ±2.98	84.38 ±3.61	84.41 ±2.81	0.97
TG (mg/dL)	Mean ±SD	144.25 ±53.60	219.43 ±50.11	122.92 ±30.41	<0.001*
FBS (mg/dL)	Mean ±SD	95.36 ±19.96	94.71 ±19.65	95.54 ±20.17	0.87
TyG index	Mean ±SD	8.76 ±0.37	9.20 ±0.34	8.63 ±0.27	<0.001*
Albumin (g/dL)	Mean ±SD	3.67 ±0.31	3.43 ±0.31	3.74 ±0.28	<0.001*
NAFLD fibrosis score	Mean ±SD	−1.97 ±1.62	−0.24 ±1.25	−2.46 ±1.36	<0.001*
Liver fibrosis	n (%)	21 (22.1%)	-	-	-
MAFLD	n (%)	21 (22.1%)	-	-	-
FIB-4 score	Mean ±SD	1.21 ±0.72	2.05 ±0.81	0.97 ±0.47	<0.001*
NAFLD fibrosis score	Median (range)	-	-0.32 (-1.20 - 0.60)	-2.60 (-3.49 - -1.59)	<0.001*

Abbreviations: WC: waist circumference, MAFLD: metabolic-associated fatty liver disease, TC: total cholesterol, CKD: chronic kidney disease, CAD: coronary artery disease, HGB: hemoglobin, AST: aspartate aminotransferase, LDL: low-density lipoprotein, HDL: high-density lipoprotein, TG: triglycerides, ALT: alanine aminotransferase, FBS: fasting blood sugar, TyG: triglyceride-glucose, NAFLD: non-alcoholic fatty liver disease, FIB-4: fibrosis-4, *: Significant P-value.

The TyG index showed high discrimination for MAFLD, with an AUC of 0.906 (95% CI, 0.815–0.998). At a cutoff value of >8.88, sensitivity was 95.2%, specificity was 82.4%, the positive predictive value was 60.5%, and the negative predictive value was 98.4% (Fig. 1).

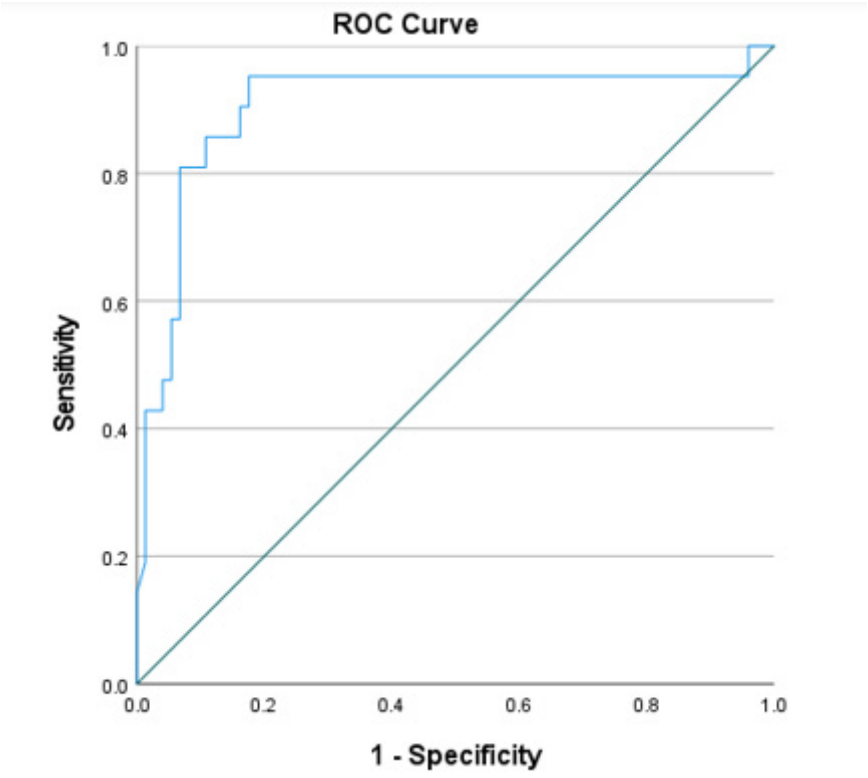

Fig. 1. ROC curve analysis of the discriminative performance of the TyG index for MAFLD.

Univariable and age- and sex-adjusted logistic regression analyses were performed to assess factors associated with MAFLD. Each adjusted model included one studied predictor together with age and sex. In the adjusted models, each one-unit increase in BMI was associated with 66.4% higher odds of MAFLD (OR = 1.664, 95% CI: 1.314–2.108, P < 0.001). Each one-unit increase in ALT was associated with 7.0% higher odds (OR = 1.070, 95% CI: 1.005–1.138, P = 0.033), and each one-unit increase in CRP was associated with 62.3% higher odds (OR = 1.623, 95% CI: 1.271–2.073, P < 0.001). Each one-unit increase in the TyG index was associated with higher odds of MAFLD (OR = 3.380, 95% CI: 1.985–5.757, P < 0.001). Conversely, higher albumin was associated with lower odds of MAFLD (OR = 0.028 per 1 g/dL increase, 95% CI: 0.004–0.200, P < 0.001) (Table 2).

Table 2

Univariable and age- and sex-adjusted logistic regression analyses of factors associated with MAFLD

	Univariate		Multivariate	
	OR (95% CI)	P-value	OR (95% CI)†	P-value
BMI (kg/m^2)	1.582 (1.295 - 1.933)	<0.001*	1.664 (1.314 - 2.108)	<0.001*
Albumin (g/dL)	0.031 (0.005 - 0.192)	<0.001*	0.028 (0.004 - 0.200)	<0.001*
ALT (U/L)	1.070 (1.007 - 1.138)	0.030*	1.070 (1.005 - 1.138)	0.033*
CRP (mg/L)	1.545 (1.288 - 1.854)	<0.001*	1.623 (1.271 - 2.073)	<0.001*
TyG index	3.300 (1.968 - 5.532)	<0.001*	3.380 (1.985 - 5.757)	<0.001*

*†Adjusted for age and sex. OR: odds ratio; CI: confidence interval; BMI: body mass index; ALT: alanine aminotransferase; CRP: C-reactive protein; TyG: triglyceride-glucose; MAFLD: metabolic dysfunction-associated fatty liver disease; *: statistically significant P value.*

Discussion. Several mechanisms may explain the association between hepatic steatosis and CKD. Fatty liver disease and CKD share major cardiometabolic risk factors and pathogenic pathways, including hypertension, obesity, insulin resistance, and dyslipidemia [7]. Previous studies have also reported associations between MAFLD and CKD risk [8].

The observed MAFLD prevalence of 22.1% highlights the metabolic burden in patients receiving HD. This finding was comparable to the 22.2% prevalence reported by Deng et al. [8] among participants with CKD in the National Health and Nutrition Examination Survey. Differences between studies may reflect ethnicity, metabolic profiles, kidney disease severity, and diagnostic methods.

In contrast, Wong et al. [7] reported a higher MAFLD prevalence of 43.4% in a multiethnic Malaysian cohort of 447 patients receiving HD. In a Japanese cohort of 13,159 participants followed for 10 years, Tanaka et al. [9] reported a prevalence of 32.3% and found that MAFLD was independently associated with incident CKD. These differences suggest that MAFLD prevalence varies according to population characteristics and study design.

In the present study, patients with MAFLD had significantly higher body weight, BMI, and WC than those without MAFLD, supporting the close relationship between MAFLD and abdominal obesity. Most patients had no history of hypertension or diabetes mellitus.

Previous studies have reported similar metabolic differences between patients with and without MAFLD. Deng et al. [8] found that MAFLD was associated with higher rates of hypertension, diabetes mellitus, and hyperuricemia. Tanaka et al. [9] also reported higher WC, systolic blood pressure, and BMI in patients with MAFLD. In the present study, the low prevalence of hypertension and diabetes mellitus may partly reflect the relatively young mean age of the cohort. Nevertheless, patients with MAFLD had greater WC and BMI than those without MAFLD.

Fibrosis-related risk was assessed using the NAFLD fibrosis score and the FIB-4 score. Patients with MAFLD had significantly higher values of both scores than those without MAFLD, indicating a higher estimated risk of liver fibrosis in the MAFLD group. Liver fibrosis is an important concern in patients with MAFLD. Wong et al. [7] reported advanced liver fibrosis in 25.5% of their HD cohort. Zou et al. [10] also found that higher fibrosis scores were associated with a greater risk of progression to end-stage kidney disease in patients with diabetic nephropathy and MAFLD.

Patients with MAFLD also showed a distinct inflammatory and biochemical profile. CRP, AST, ALT, and TC were significantly higher in the MAFLD group. The higher CRP level supports the inflammatory component of the disease. Similarly, Wong et al. [7] reported that elevated high-sensitivity CRP was independently associated with MAFLD in patients receiving HD. Deng et al. [8] and Tanaka et al. [9] also reported higher inflammatory and liver-related markers in patients with MAFLD.

Patients with MAFLD had significantly higher TG, TC, and LDL-C levels than those without MAFLD, supporting the close association between MAFLD and an adverse metabolic profile.

Lipid abnormalities are a well-recognized feature of MAFLD. In a HD cohort, Wong et al. [7] reported that low HDL-C, high FBS, and elevated TG were associated with MAFLD. Higher TG levels were also reported by Tanaka et al. [9] and Deng et al. [8]. In the present study, the TyG index, which combines TG and fasting glucose, was markedly higher in patients with MAFLD and may reflect the underlying metabolic dysfunction.

ROC analysis showed high discrimination of the TyG index for MAFLD in this cohort (AUC 0.906). At a cutoff >8.88, sensitivity was 95.2% and specificity was 82.4%. However, this cutoff was derived and evaluated in the same small cohort and should be considered exploratory.

Because the TyG index reflects triglyceride and fasting glucose levels, it may capture metabolic disturbances closely linked to MAFLD. However, these components also overlap with metabolic features used in the MAFLD definition, which may increase the apparent discriminative performance of the index.

The association between the TyG index and MAFLD is consistent with findings by Tanaka et al. [9], Xue et al. [11], Liu et al. [12], and Zhang et al. [13]. These studies also reported positive associations between higher TyG values and MAFLD or related metabolic risk.

Together, these findings support the substantial metabolic burden associated with MAFLD in patients with kidney disease. Previous studies have linked MAFLD to adverse metabolic profiles and CKD progression [7–9, 14]. In patients receiving HD, simple biochemical markers may help identify individuals who warrant further liver assessment, although prospective studies are needed to determine their clinical value.

Several limitations should be noted. The study was performed at a single center and included a relatively small sample of 95 patients, which may limit generalizability to the wider HD population. Its cross-sectional design does not allow causal or longitudinal inferences. Lifestyle factors such as dietary habits and physical activity were not assessed. Liver biopsy and advanced imaging modalities such as transient elastography (FibroScan) were not used, limiting fibrosis assessment. Abdominal ultrasonography is operator-dependent and may introduce variability in the detection of hepatic steatosis. In addition, the TyG index includes triglycerides and fasting glucose, which overlap with metabolic features used in the MAFLD definition. This overlap may increase its apparent discrimination for MAFLD. The ROC cutoff was derived and evaluated in the same small cohort and therefore requires external validation.

Conclusions. MAFLD was present in approximately one-fifth of patients receiving maintenance hemodialysis and was associated with metabolic and inflammatory abnormalities. The TyG index showed high discriminative performance for MAFLD in this cohort. However, these findings are exploratory and require validation in larger prospective, multicenter studies.

Ethics approval and consent to participate. The study was approved by the Ethical Committee of Ain Shams University Hospital (approval code: FMASU MS 379/2023). Written informed consent was obtained from all participants prior to inclusion in the study.

Competing interests. The authors declare no competing interests.

Findings. This work was done without funding support.

Availability of data and materials. The datasets used and/or analyzed during the current study are available from the author on reasonable request.

Author contributions.

Khaled Gouda Abdelwahab: conceptualized the study, supervised the research, and critically revised the manuscript.

Ahmed Esmat Saleh: contributed to study design, data collection, statistical analysis, and drafting of the manuscript.

Ashraf Amin Abdelaziz: participated in data collection, interpretation of results, and manuscript revision.

Mohammad Almohamady Khaskia: contributed to data acquisition, laboratory investigations, and manuscript editing.

All authors reviewed and approved the final version of the manuscript and agree to be accountable for all aspects of the work.

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