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Prevalence and Risk Factors of Metabolic Associated Fatty Liver Disease in Benha University Hospitals Employees, Egypt: A Cross-Sectional Study

Marwa S. Mohamady, Mohamed S. El Sayed, Ayman M. El Badawy, Amira M. El Sayed, Walaa M. Ibrahem, Rasha O. Abdelmoniem

Internal Medicine Department, Faculty of Medicine Benha University, Egypt.

Corresponding to:
Dr. Marwa S. Mohamady.
Internal Medicine Department, Faculty of Medicine Benha University, Egypt.
Email: marwasallama0@gmail.com

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Abstract:

Background: Background: Metabolic Associated Fatty Liver Disease (MAFLD) is a common liver disorder linked to metabolic dysfunction and poses a growing public health challenge. This study aims to determine the prevalence and associated risk factors of MAFLD among employees at Benha University Hospitals, Egypt. **Methods:** A cross-sectional study was conducted involving 200 adult hospital employees over the period from July 2022 to July 2023. **Results:** MAFLD was identified in 18.6% of participants. The MAFLD group had significantly higher age (47 ± 9 vs. 37 ± 7 years, $P < 0.001$), BMI (28.7 ± 4.5 vs. 24.3 ± 3.7 kg/m², $P < 0.001$), Fatty Liver Index (FLI) (74 ± 17 vs. 51 ± 17, $P < 0.001$), and serum ferritin (276 vs. 149 ng/mL, $P < 0.001$). ROC analysis showed good predictive performance for serum ferritin (AUC = 0.78) and BMI (AUC = 0.75). Logistic regression identified FLI (OR = 1.06, P = 0.002) and serum ferritin (OR = 1.01, P = 0.027) as independent predictors of MAFLD, while BMI and Triglycerides (TGs) showed borderline significance. **Conclusion:** MAFLD is highly prevalent among hospital employees and is strongly related to metabolic risk factors. FLI and serum ferritin are reliable non-invasive predictors. Targeted screening and metabolic control strategies are essential to address this growing burden.

Keywords: Fatty Liver Index, Metabolic Associated Fatty Liver Disease, Serum Ferritin, Metabolic Syndrome, Hepatic Steatosis.

Introduction

MAFLD, previously[y designated as NAFLD, constitutes the most prevalent form of chronic hepatic pathology globally, with an estimated prevalence of approximately 24% among the adult population [1], It is characterized by hepatic fat accumulation in individuals with metabolic dysfunction and is recognized as the hepatic component of a multisystem metabolic disorder [2, 3]; Key risk factors encompass obesity, T2DM, physical inactivity, and suboptimal dietary patterns [4, 5].

Historically, MAFLD was diagnosed by excluding other liver diseases and significant alcohol intake [1]. The updated diagnostic criteria, however, adopt a positive diagnostic orientation, mandating the demonstration of hepatic steatosis alongside the presence of one or more defined metabolic risk determinants: obesity or overweight, evidence of metabolic dysregulation, or T2DM presence [2]. The latter includes metabolic abnormalities like increased waist circumference, hypertension, dyslipidemia, and insulin resistance. Diagnostic modalities frequently employed for the detection of hepatic steatosis include ultrasound, FibroScan with CAP, CT, MRI-PDFF, as well as serum-based biomarkers such as the FLI [6, 7].

MAFLD is not limited to overweight individuals; lean individuals with metabolic risk factors are also susceptible. Up to 20% of MAFLD cases may have normal BMI but still exhibit liver damage [8, 9]. This highlights the independent contribution of metabolic dysfunction, beyond BMI alone, in the pathogenesis of MAFLD and its complications, including cardiovascular and renal diseases [10, 11].

Given its rising prevalence and significant morbidity, MAFLD poses a major public health challenge. In Egypt, understanding its burden among specific populations such as hospital employees can inform targeted interventions. Factors such as age, sex, residence, menopausal status, and comorbidities like diabetes and hypertension are important to explore in association with MAFLD risk [5, 12].

This study aims to determine the prevalence and associated risk factors of MAFLD among employees of Benha University Hospitals, with particular attention to lean individuals, metabolic syndrome, sociodemographic characteristics, menopausal status, comorbidities (especially cardiovascular and renal diseases), liver function, and the role of metabolic syndrome control.

Patients and methods:

Patients:

This cross-sectional, hospital-based study was conducted on 85 adult employees working at Benha University Hospitals, Egypt, over a 12-month period from July 2022 to July 2023.

Written informed consent was secured from all participants after a clear explanation of the study's purpose. Each participant was assigned a confidential identification code to ensure privacy. Approval for conducting the study was secured from the Research Ethics Committee, Faculty of Medicine, Benha University (Approval code: MD 2-6-2023).

Participants included in this study were employees of both sexes aged 18 years or older, working at Benha University Hospitals during the study period from July 2022 to July 2023. Individuals who were not employed at Benha University Hospitals or who declined to participate were excluded from the study.

MAFLD diagnosis was determined based on the detection of hepatic steatosis—ascertained either through ultrasonographic evaluation or a FLI exceeding 60—in association fulfilling at least one of the subsequent parameters: T2DM, overweight or obesity (BMI $\geq$ 25 kg/m^2), or evidence of metabolic dysregulation. T2DM was defined by documented history, use of antidiabetic medications, or abnormal glucose profiles.

In lean, non-diabetic participants, metabolic dysregulation was diagnosed if at least two metabolic risk features were present, including central obesity, hypertriglyceridemia, low HDL, HTN, prediabetes, insulin resistance, or elevated hs-CRP [13-15].

Clinical, biochemical, and radiological evaluation of study participants

All participants underwent detailed clinical evaluation, including medical history, physical examination, and anthropometric measurements. Collected data encompassed demographics, metabolic risk factors, menopausal status, drug history, comorbidities, and cardiovascular or renal disease history. Laboratory investigations included glycemic markers (HbA1C, FBS, 2-hPP), lipid profile (TGs, cholesterol, LDL), hepatic function tests (GGT, AST, ALT, bilirubin), renal function (creatinine), CBC, serum uric acid, ferritin, and autoimmune liver markers (anti-LKM-1, ASMA). Hepatic steatosis was assessed via abdominal ultrasound using standard sonographic criteria [16].

FLI calculation

FLI was computed utilizing the validated formula:

$$FLI = (e^{0.953\times\log e(\text{triglycerides})+0.139\times BMI+0.718\times\log e(GGT)+0.053\times\text{waistcircumference}-15.745}) / (1+e^{0.953\times\log e(\text{triglycerides})+0.139\times BMI+0.718\times\log e(GGT)+0.053\times\text{waistcircumference}-15.745}) \times 100$$

A score >60 was considered indicative of hepatic steatosis [17].

Statistical analysis

Statistical analyses were performed using SPSS software version 27 (IBM Corp., Armonk, NY, USA). The distribution of quantitative data was evaluated through the Shapiro–Wilk test, supplemented by visual assessment techniques. Descriptive statistics were reported as mean ± SD for normally distributed continuous variables, and as median with range for non-normally distributed ones. Categorical data were presented as absolute frequencies and corresponding percentages. Comparative analyses between groups were performed using the Independent t-test or Mann–Whitney U test for continuous variables, and Chi-square test or Fisher's exact test for categorical variables, as appropriate. ROC analysis was performed for variables such as age, BMI, GGT, FLI, and ferritin to predict MAFLD, with an AUC, cutoff points, and diagnostic indices reported. Correlations were assessed using Pearson's or Spearman's methods. Logistic regression analyses (univariate and multivariate) identified predictors of MAFLD, reporting OR with 95% CI. Statistical significance was defined as p-value < 0.05.

Results:

Subjects were stratified into two groups based on ultrasound-confirmed diagnosis of fatty liver: those with MAFLD (n = 61) and those without MAFLD (n = 24). Participants with MAFLD were markedly older (42 ± 9 vs. 37 ± 8 years, $P = 0.032$) and had elevated BMI (35.19 ± 5.38 vs. 30.28 ± 6.28 kg/m², $P < 0.001$) relative to those without MAFLD. FLI was also substantially elevated in MAFLD group (82 ± 13 vs. 56 ± 33, $P < 0.001$). Anemia was more prevalent among MAFLD cases (26.2% vs. 4.2%, $P = 0.022$). No substantial variations were observed regarding sex, diabetes, hypertension, hyperuricemia, metabolic syndrome, other comorbidities, or menopausal status.

Table 1

Participants with MAFLD had markedly higher TG levels (124 mg/dL vs. 80 mg/dL, P = 0.02), GGT levels (23.4 U/L vs. 16.8 U/L, P = 0.002), WBC count (111 ± 17 ×10³/μL vs. 100 ± 17 ×10³/μL, P = 0.007), and serum ferritin levels (315 vs. 195, P < 0.001) relative to those without MAFLD. However, no substantial variations were observed between the two groups regarding FBS, 2hpp, HbA1C, uric acid, cholesterol, LDL, creatinine, total bilirubin, direct bilirubin, AST, ALT, negative ASMA ab, or Anti-LKM-1 levels.

Table 2

Table 1: Demographics and clinical characteristics between the studied groups

	Total	MAFLD		
		Yes (n = 61)	No (n = 24)	P-value
Age (years)	40 ±9	42 ±9	37 ±8	**0.032***
Sex				
Males	3 (3.5)	1 (1.6)	2 (8.3)	0.191
Females	82 (96.5)	60 (98.4)	22 (91.7)	
BMI (kg/m^2)	33.8 ±6.03	35.19 ±5.38	30.28 ±6.28	**<0.001***
Diabetes mellitus	18 (21.2)	16 (26.2)	2 (8.3)	0.069
Hypertension	13 (15.3)	11 (18)	2 (8.3)	0.263
Menoupase state	19 (11.8)	8 (13.1)	2 (8.3)	0.538
Hyperuricemia	15 (17.6)	12 (19.7)	3 (12.5)	0.435
Anemia	17 (20)	16 (26.2)	1 (4.2)	**0.022***
Metabolic syndrome	31 (36.5)	25 (41)	6 (25)	0.168
Other comorbidities	11 (12.9)	9 (14.8)	2 (8.3)	0.427
Fatty liver index	75 ±24	82 ±13	56 ±33	**<0.001***

Data were presented as mean ±SD or n (%), n: number: BMI: Body mass index, SD: Standard deviation, *: Significant P-value.

Table 2: Laboratory findings between the studied groups

	Total	MAFLD		
		Yes (n = 61)	No (n = 24)	P-value
FBS (mg/dL)	92 (68 - 295)	93 (68 - 295)	92 (70 - 257)	0.88
2hpp (mg/dL)	110 (88 - 500)	110 (89 - 500)	110 (88 - 333)	0.802
HbA1C (%)	5.8 ±1.5	5.9 ±1.7	5.4 ±1.1	0.187
Uric acid (mg/dL)	4.9 ±1	4.9 ±1	4.7 ±0.9	0.381
Cholesterol (mg/dL)	213 ±44	213 ±41	215 ±53	0.892
TG (mg/dL)	110 (45 - 360)	124 (45 - 360)	80 (63 - 210)	**0.02***
LDL (mg/dL)	136.7 ±40.8	133.9 ±38.9	144.1 ±45.5	0.302
Creatinine (mg/dL)	0.7 ±0.1	0.7 ±0.1	0.7 ±0.1	0.111
Total bilirubin (mg/dL)	0.6 ±0.1	0.6 ±0.1	0.6 ±0.1	0.722
Direct bilirubin (mg/dL)	0.1 ±0.1	0.1 ±0.1	0.2 ±0.1	0.953
AST (U/L)	18 (12 - 44)	18 (12 - 44)	18 (12 - 25)	0.503
ALT (U/L)	18 ±5	18 ±5	17 ±4	0.109
GGT (U/L)	22.2 (9.5 - 98)	23.4 (11.2 - 98)	16.8 (9.5 - 80.6)	**0.002***
WBC (×10³/µL)	108 ±18	111 ±17	100 ±17	**0.007***
Negative ASMA ab	85 (100)	61 (100)	24 (100)	1
Anti LKM -1	10 (5 - 21)	11 (6 - 21)	9 (5 - 16)	0.057
Serum ferritin	312 (18 - 420)	315 (308 - 420)	195 (18 - 331)	**<0.001***

Data were presented as mean ±SD, median (range) or n (%),n: number; FBS: Fasting blood sugar; 2hpp: Two-hour postprandial blood sugar; HbA1C: Hemoglobin A1C; TG: Triglycerides; LDL: Low-density lipoprotein; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; GGT: Gamma-glutamyl transferase; WBC: White blood cell count; ASMA: Anti-smooth muscle antibody; Anti-LKM-1: Anti-liver kidney microsomal antibody type 1; SD: Standard deviation; *: Significant P-value.

ROC curve analysis was done for age to predict MAFLD. It revealed a significant AUC of 0.648 with a 95% CI from 0.526 to 0.771, suggesting poor to fair ability to predict MAFLD. The best cutoff was >45 years, at which sensitivity, specificity, PPV, and NPV were 42.62%, 91.67%, 92.9%, and 38.6%, respectively. **Figure 1A**

ROC curve analysis was done for BMI to predict MAFLD. It revealed a significant AUC of 0.745 with a 95% CI from 0.620 to 0.870, suggesting good ability to predict MAFLD. The best cutoff was >30.48 kg/m², at which sensitivity, specificity, PPV, and NPV were 85.25%, 62.50%, 85.2%, and 62.5%, respectively. **Figure 1B**

ROC curve analysis was done for GGT to predict MAFLD. It revealed a significant AUC of 0.714 with a 95% CI from 0.572 to 0.856, suggesting fair to good ability to predict MAFLD. The best cutoff was >17.8 U/L, at which sensitivity, specificity, PPV, and NPV were 83.61%, 70.83%, 87.9%, and 63%, respectively. **Figure 1C**

ROC curve analysis was performed for TG to predict the MAFLD. It demonstrated a significant AUC of 0.663, with a 95% CI from 0.563 to 0.791, indicating a fair predictive ability. The optimal cutoff value was >80, achieving a specificity of 54.17%, sensitivity of 78.69%, PPV of 81.4%, and NPV of 50% (P = 0.02). **Figure 1D**

ROC curve analysis was done for FLI to predict MAFLD. It revealed a significant AUC of 0.707 with a 95% CI from 0.558 to 0.855, suggesting fair to good ability to predict MAFLD. The best cutoff was >62, at which sensitivity, specificity, PPV, and NPV were 91.8%, 54.17%, 83.6%, and 72.2%, respectively. **Figure 1E**

ROC curve analysis was done for serum ferritin to predict MAFLD. It revealed a significant AUC of 0.760 with a 95% CI from 0.615 to 0.905, indicating good ability to predict MAFLD. The best cutoff value was >307 ng/mL, achieving a sensitivity of 100%, specificity of 62.5%, PPV of 85.9%, and NPV of 100% (P < 0.001). **Figure 1F**

FLI revealed substantial positive correlations with BMI (r = 0.276, P = 0.011), WBC (r = 0.54, P < 0.001), and GGT (r = 0.224, P = 0.039). In contrast, it did not reveal significant correlations with age (P = 0.976), FBS (P = 0.887), 2hpp (P = 0.648), HbA1C (P = 0.461), uric acid (P = 0.689), cholesterol (P = 0.351), TG (P = 0.298), LDL (P = 0.384), creatinine (P = 0.507), total bilirubin (P = 0.439), direct bilirubin (P = 0.204), ALT (P = 0.918), AST (P = 0.123), anti LKM -1 (0.615), or serum ferritin (0.887). **Figure 2**

In the multivariate stepwise analysis, serum ferritin (OR = 1.116, 95% CI = 1.03–1.209, P = 0.007) and FLI (OR = 1.102, 95% CI = 1.036–1.171, P = 0.002) emerged as independent predictors. BMI (OR = 1.34, 95% CI = 1.00–1.79, P = 0.05) and TGs (OR = 1.031, 95% CI = 0.99–1.06, P = 0.057) were associated with MAFLD with borderline significance. GGT levels were not substantially correlated with MAFLD (P = 0.136). **Table 3**

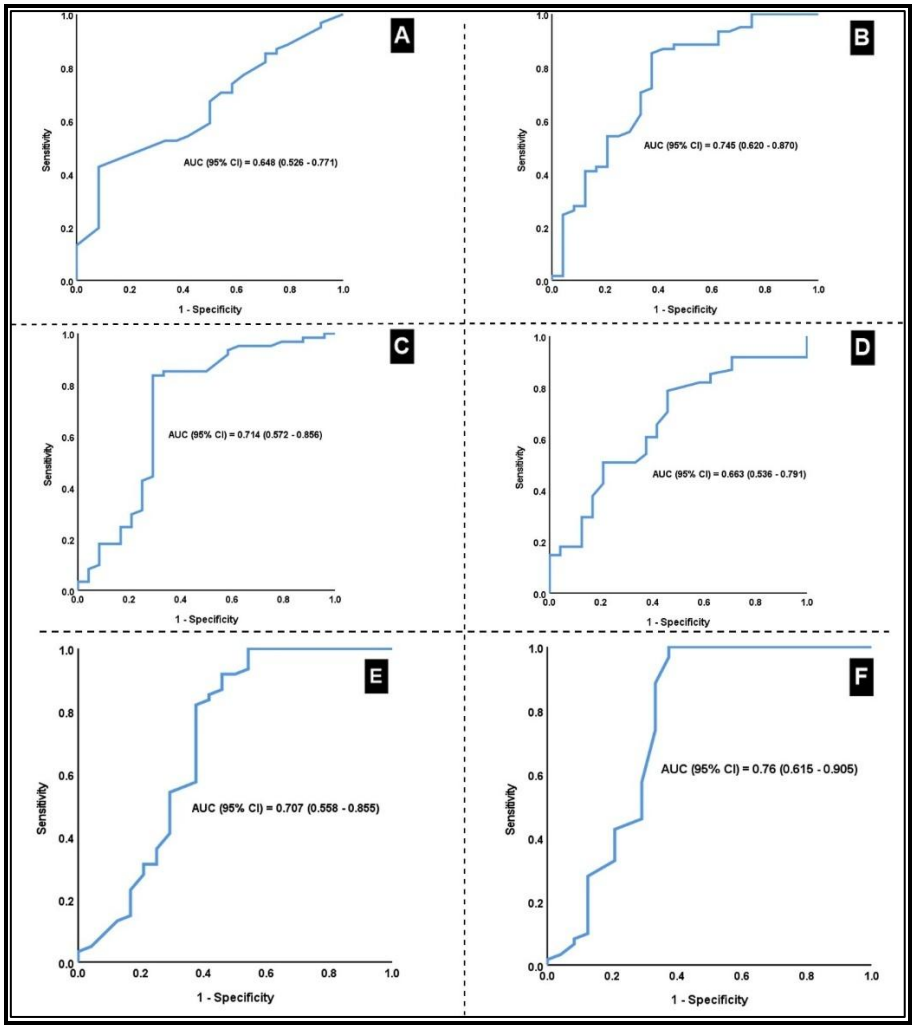

Figure 1: ROC analysis of A) age, B) BMI, C) gamma-glutamyl transferase, D) TG, E) fatty liver index, and F) serum ferritin to predict MAFLD

Table 3: Univariate and multivariate logistic regression analysis to predict MAFLD

	Univariate		Multivariate	
	OR (95% CI)	**P-value**	**OR (95% CI)**	**P-value**
Age (years)	1.061 (1.004 - 1.122)	**0.036***	-	-
BMI (kg/m^2)	1.199 (1.07 - 1.343)	**0.002***	1.34 (1.00 - 1.79)	0.05
Anemia	8.178 (1.02 - 65.577)	**0.048***	-	-
TG (mg/dL)	1.011 (1.001 - 1.022)	**0.033***	1.031 (0.99 – 1.06)	0.057
GGT (U/L)	1.03 (0.991 - 1.072)	0.136	-	-
WBC (×10³/µL)	1.043 (1.01 - 1.077)	**0.011***	-	-
Ferritin (ng/mL)	1.037 (1.007 – 1.069)	**0.017***	1.116 (1.03 - 1.209)	**0.007***
Fatty liver index	1.052 (1.025 - 1.08)	**<0.001***	1.102 (1.036 - 1.171)	**0.002***

OR: Odds ratio, CI: Confidence interval, BMI: Body mass index, TG: Triglycerides, GGT: Gamma-glutamyl transferase, WBC: White blood cell, *: Significant P-value.

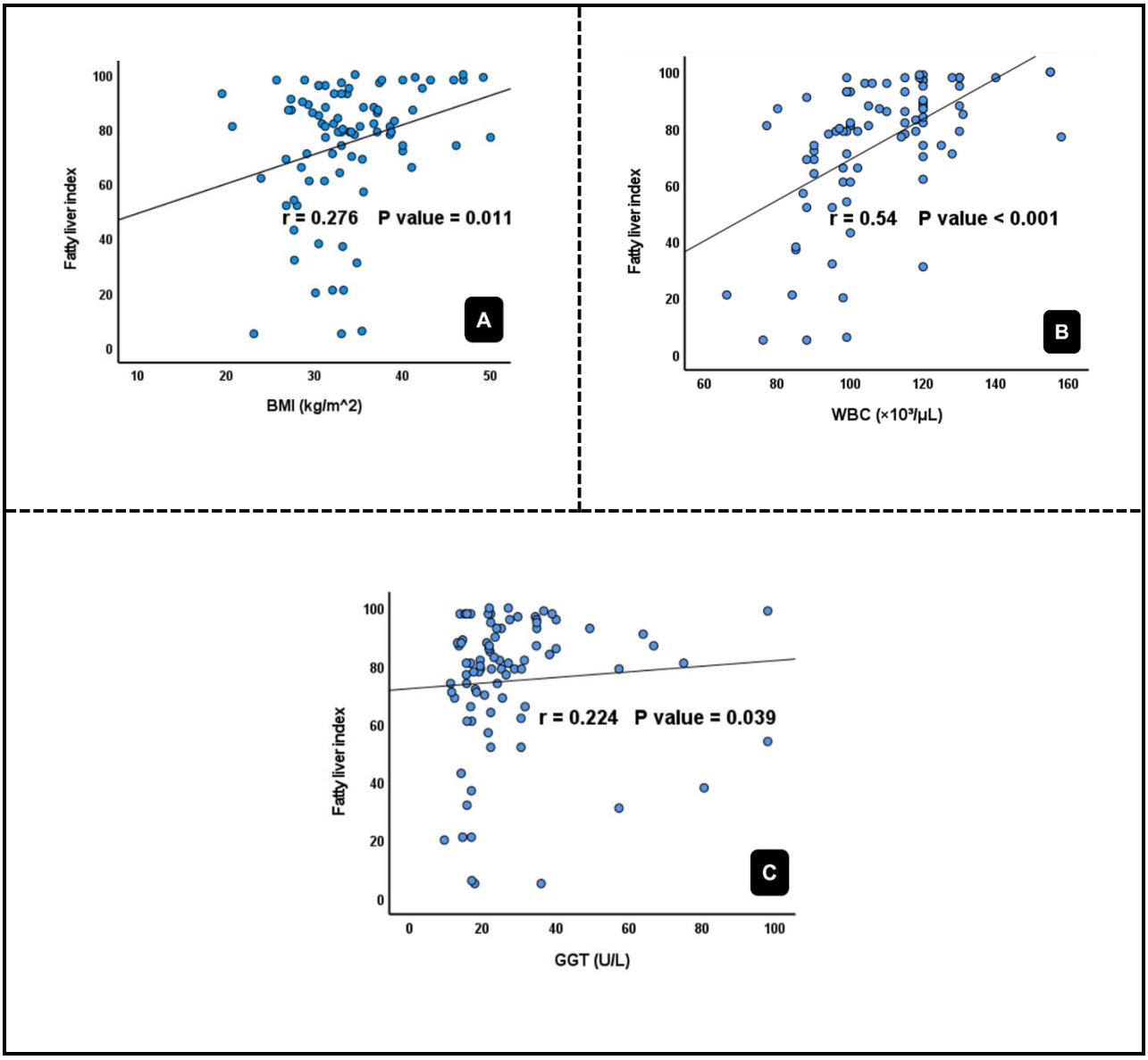

Figure 2: Correlation between fatty liver index and (A) BMI, (B) WBCs, and (C) GGT

Discussion:

MAFLD has become the most prevalent chronic liver disease worldwide, primarily driven by the increasing incidence of metabolic dysfunction. Its expanded diagnostic criteria underscore the pressing need to evaluate disease burden among high-risk populations, such as healthcare workers. So, a total of 85 adult hospital employees from Benha were recruited to assess MAFLD prevalence as well as to explore the contributing risk factors in this population.

In the current study, MAFLD was found in 71.8% of study participants, indicating a high prevalence. This exceeds rates reported by 2 studies revealing (56.4%) and (33.7%) [18, 19]. A meta-analysis [20] reported a pooled prevalence of 39.43% in the general population and 68.71% in diabetics in the MENA region, highlighting the growing burden of fatty liver disease.

FLI was markedly higher in MAFLD cases in our study, supporting its role as a diagnostic tool. This aligns with findings by a study reported FLI scores of

48.63 ± 25.5 in MAFLD versus 23.1 ± 20.75 in non-MAFLD (P < 0.0001) [21]. A study similarly observed a median FLI of 83.88 in MAFLD versus 26.20 (P < 0.001) [22]. Another study also noted higher FLI in fatty liver cases [67 (49–83) vs. 31 (17–50); P < 0.001] [23].

In our study, anemia was more frequently observed among MAFLD cases, indicating a possible association between reduced hemoglobin levels and presence of fatty liver disease. This contrasts with a study found higher hemoglobin levels in MAFLD cases (15.34 ± 1.50 vs. 14.23 ± 1.50; P < 0.0001) (21), indicating less anemia. Differences in population characteristics or confounding factors may explain this variation, highlighting the need for further investigation.

In this study, TG levels were markedly higher in participants with MAFLD, reinforcing the link between hypertriglyceridemia and fatty liver disease. Similar associations were reported by 2 studies finding (150.91 ± 143.04 vs. 98.8 ± 64.16; P < 0.0001) and [1.32 (0.96–1.86) vs. 0.82 (0.58–1.23); P < 0.001] [21, 22], highlighting dyslipidemia as a key factor in MAFLD pathogenesis and suggesting TG as a useful biomarker.

Our study showed significantly elevated GGT levels in MAFLD cases, indicating hepatic dysfunction and oxidative stress. This finding aligns with previous studies finding (72.79 ± 215.35 vs. 36.76 ± 66.86; P = 0.007) and [25.00 (18.00–40.00) vs. 17.00 (12.00–25.00); P < 0.001] [21, 22], supporting GGT as a marker of liver injury and metabolic disturbance in MAFLD.

Our study found higher WBC counts in MAFLD participants, suggesting an inflammatory role in disease pathogenesis. This aligns with a study reported significantly elevated WBC levels in the MAFLD group (6722.82 ± 1950.21) compared to non-MAFLD (6019.63 ± 1735.57; P < 0.0001) [21], supporting the link between systemic inflammation and MAFLD progression.

Serum ferritin levels were significantly higher in MAFLD cases in our study, reflecting underlying inflammation and metabolic dysregulation. These findings harmonize with results of a meta-analysis showed higher ferritin levels in FLD cases (mean difference = 1.54 ng/mL; 95% CI: 0.85–2.23; P < .001) [24]. Elevated ferritin in MAFLD likely indicates hepatocellular injury, oxidative stress, and increased iron stores due to insulin resistance.

In our study, age showed limited predictive value for MAFLD, with an AUC of 0.648. A cutoff above 45 years yielded high specificity but low sensitivity, indicating age alone is not a strong standalone predictor. This aligns with previous studies finding (AUC = 0.647; P < 0.001), (AUC = 0.683; sensitivity 0.646, specificity 0.628), and (AUC = 0.725; 95% CI: 0.674–0.766; P < 0.001) [25] [26], all of them reported fair predictive performance of age in MAFLD risk assessment [27], [28].

In our study, BMI showed strong predictive value for MAFLD, with an AUC of 0.745. A cutoff above 30.48 kg/m² provided high sensitivity and acceptable specificity. This aligns with previous studies finding (AUC = 0.743; P < 0.001) and (AUC = 0.824; 95% CI: 0.782–0.865; P < 0.001), which confirmed BMI's reliability, especially when combined with age (AUC = 0.838) [25, 26]. Although another study reported a lower AUC of 0.593 for overweight status, specificity remained high (0.753). Collectively, these findings validate BMI as a key MAFLD predictor [19].

In this study, GGT demonstrated fair to good predictive ability for MAFLD, with an AUC of 0.714 and a cutoff above 17.8 U/L providing high sensitivity and moderate specificity. This aligns with numerous studies finding (AUC = 0.668; 95% CI: 0.622–0.714) and (AUC = 0.640; 95% CI: 0.618–0.661) [21, 23], supporting GGT's role as a useful biomarker for detecting hepatic steatosis and liver dysfunction in MAFLD.

TGs showed fair predictive ability for MAFLD in our study (AUC = 0.663), with a cutoff >80 mg/dL yielding moderate sensitivity but low specificity. Similarly, a study reported an AUC of 0.659 (95% CI: 0.596–0.722), with high specificity (0.928) but low sensitivity (0.309) [29]. Another one found an AUC of 0.682 (95% CI: 0.661–0.703) [23]. These findings indicate that while TG is a relevant marker, its diagnostic utility is stronger when combined with other indicators.

In our study, FLI showed fair to good predictive ability for MAFLD (AUC = 0.707), with a cutoff >62 providing high sensitivity and moderate specificity, supporting its value as a screening tool. A study similarly reported an AUC of 0.776 (95% CI: 0.737–0.816; P < 0.0001) with 71.3% specificity and 71.2% sensitivity at a cutoff of 30 [21]. Another study found higher accuracy with an AUC of 0.813 (95% CI: 0.797–0.830), specificity of 81% and sensitivity of 62%, at a cutoff of 60 [23]. Additional one also demonstrated strong performance of FLI (AUC = 0.834), with 79.89% sensitivity and 71.51% specificity at a cutoff of 30 in a large Chinese cohort [30].

In our study, serum ferritin demonstrated good predictive accuracy for MAFLD, with an AUC of 0.760 and a cutoff >307 ng/mL providing excellent sensitivity and negative predictive value. Similarly, a study reported ferritin as a significant MAFLD predictor with an AUC of 0.723 (P < 0.001) [25]. Higher accuracy was reported by another study which found an AUC of 0.995 (95% CI: 0.978–1.013; P = 0.005) for serum ferritin in diagnosing liver fibrosis and steatosis using FibroScan in pediatric FLD cases [31], supporting its utility as a non-invasive diagnostic marker.

In our study, logistic regression confirmed serum ferritin as an independent predictor of MAFLD, underscoring its role as a marker of hepatic inflammation and iron overload. This aligns with a study found significant associations between SF and FLD risk in men (OR = 2.36, 95% CI: 1.41–3.93; P = 0.001) and women (OR = 2.93, 95% CI: 1.83–4.69; P < 0.001). After adjusting for confounders, SF remained independently predictive in both men (OR = 2.24, 95% CI: 1.64–3.05; P < 0.001) and women (OR = 3.30, 95% CI: 2.13–5.11; P < 0.001), reinforcing its utility in MAFLD risk stratification [24].

Our analysis identified FLI as an independent predictor of MAFLD, supporting its value as a practical, non-invasive diagnostic tool. Similarly, a study reported a significant association between FLI and FLD after adjusting for confounders (OR = 1.05, 95% CI: 1.04–1.05; P < 0.001), with FLI also correlating with disease severity (β = 0.048, 95% CI: 0.043–0.052; P < 0.001) [23]. Another study found that individuals with FLI >30.1 had a 6.135-fold higher risk of MAFLD, and each one-point increase in FLI raised MAFLD risk by 4.4% (OR = 1.044) [21].

Although BMI was significantly associated with MAFLD in our study, its borderline significance in the multivariate model suggests it may be a contributing rather than an independent determinant when serum ferritin and FLI are considered. Nonetheless, previous research has consistently identified BMI as an independent risk factor for MAFLD. A study reported an OR of 1.095 (95% CI: 1.036–1.158; P = 0.001) [25], another one observed a stronger association (OR = 10.986, 95% CI: 5.317–22.698) [32]. Also, a study found a similarly significant link (OR = 1.51; 95% CI: 1.33–1.72; P < 0.001), underscoring the pivotal role of adiposity in MAFLD development [33].

In our logistic regression analysis, TG levels were linked to MAFLD but with borderline significance in the multivariate model. This contrasts with a study reporting TG as an independent risk factor (OR = 1.651; 95% CI: 1.627–1.691; P < 0.001) [34]. The discrepancy may reflect variations in population characteristics or confounding by other metabolic indicators like BMI and FLI. Still, elevated TG remains a key marker of metabolic

dysfunction and an essential component of MAFLD diagnostic criteria.

This study was limited by its single-center, cross-sectional design, which restricts causal inference. The sample size was relatively small and may not represent the general population.

Conclusion:

MAFLD is strongly associated with several metabolic syndrome components and systemic complications, including cardiovascular, renal, and hematological abnormalities. Serum ferritin and FLI emerge as independent predictors, highlighting their clinical utility. This underscores the importance of proactive screening and early metabolic health interventions, even in non-obese individuals, to curb the growing burden of MAFLD in occupational settings.

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None.

Author contribution

Authors contributed equally to the study.

Conflicts of interest

None.

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