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To study the prevalence of metabolic-associated fatty liver disease in a tertiary hospital in Bundelkhand region

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ABSTRACT

Background: Metabolic-associated fatty liver disease (MAFLD) is a growing global health concern associated with metabolic dysfunction, including obesity, type 2 diabetes mellitus, and dyslipidemia. Its prevalence is increasing, particularly in developing regions, necessitating early detection and intervention. **Aims and Objectives:** This study aims to determine the prevalence of MAFLD in a tertiary care hospital and assess the demographic profile and associated risk factors in the Bundelkhand region. **Materials and Methods:** A cross-sectional study was conducted in the Department of General Medicine, Maharani Laxmi Bai Medical College, Jhansi, from June 2023 to June 2024, including 302 patients attending outpatient and inpatient services. Participants underwent clinical evaluations, biochemical tests, and ultrasonography to diagnose MAFLD. Risk factors such as obesity, hypertension, diabetes, and dyslipidemia were analyzed. Statistical tests were applied to assess significant associations. **Results:** The prevalence of MAFLD was 54.63%, with a higher incidence in males (median age: 50 years) than females (48 years). Among MAFLD patients, obesity (81.81%), type 2 diabetes (46.06%), metabolic syndrome (82.42%), dyslipidemia (66.06%), hypothyroidism (37.57%), and hypertension (37.57%) were significantly prevalent ($P<0.05$). Liver function tests and biochemical markers were significantly altered in MAFLD cases. **Conclusion:** The study highlights a high prevalence of MAFLD in the Bundelkhand region, closely linked to metabolic risk factors. Early screening and lifestyle interventions are crucial to prevent disease progression.

Key words: Metabolic-associated fatty liver disease; Obesity; Metabolic syndrome; Diabetes; Hypothyroidism; Hypertension

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INTRODUCTION

Metabolic-associated fatty liver disease (MAFLD) is a relatively recent term that replaced non-alcoholic fatty liver disease (NAFLD) to better reflect the metabolic dysfunction underlying the condition. The redefinition of this disorder emphasized its metabolic origins and provided a clearer diagnostic criterion. Unlike NAFLD, which was defined by the exclusion of secondary causes, MAFLD is diagnosed based on the presence of hepatic steatosis with at least one metabolic abnormality, such as obesity, type 2 diabetes mellitus (T2DM), or metabolic dysfunction.[1]

The prevalence of MAFLD has increased dramatically in recent decades, becoming a global health concern. Studies estimated that approximately 25–32% of the world population is affected, with regional variations observed in different continents. The highest prevalence is reported in the Middle East and South America, followed by Asia, North America, and Europe.[2] In India, the urban population exhibits a prevalence rate similar to other Asian

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countries, ranging between 16% and 32%, whereas rural populations display a significantly lower rate of around 9%.[3]

MAFLD is primarily characterized by excessive fat accumulation in hepatocytes, leading to hepatic steatosis. While the disease often remains asymptomatic in its early stages, it may progress to more severe conditions, including non-alcoholic steatohepatitis, fibrosis, cirrhosis, and hepatocellular carcinoma. In addition, MAFLD is closely linked to extrahepatic complications such as cardiovascular disease, chronic kidney disease, and metabolic syndrome.[4] The disease's pathophysiology is multifactorial, involving insulin resistance, lipid metabolism dysfunction, inflammation, and genetic predisposition.[5]

Diagnosis of MAFLD relies on imaging techniques such as ultrasonography, which serves as the first-line investigation. However, its sensitivity is limited, especially for detecting mild steatosis. More advanced imaging modalities, including transient elastography (FibroScan) and magnetic resonance elastography, provide superior accuracy in assessing liver fat content and fibrosis.[6] In addition, non-invasive biochemical markers such as the hepatic steatosis index (HSI), Fibrosis-4 (FIB-4) Index, and the NAFLD fibrosis score aid in disease assessment and stratification. Liver biopsy remains the gold standard for definitive diagnosis, but it is invasive nature restricts its routine use.[7]

Aims and objectives

- To study the prevalence of MAFLD in a tertiary care hospital in Bundelkhand region
- To determine the demographic profile and risk factors associated with MAFLD based on prevalence.

MATERIALS AND METHODS

Study design

This cross-sectional study was conducted to determine the prevalence of MAFLD in a tertiary care hospital. The study includes a sample size of 302 participants and was carried out from June 2023 to June 2024. The research was conducted in the Department of General Medicine at Maharani Laxmi Bai Medical College, Jhansi, Uttar Pradesh, under the supervision of experienced faculty members.

Inclusion criteria

- Age 18–65 years (males and females)
- Blood pressure >130/85 mmHg or specific pharmacological treatment
- Plasma triglycerides >150 mg/dL or specific pharmacological treatment
- Plasma high-density lipoprotein cholesterol (HDL-C) <40 mg/dL for men and <50 mg/dL for women, or specific pharmacological treatment
- Prediabetes (fasting glucose: 100–125 mg/dL) [5.6–6.9 mmol/L] or 2 h after glucose tolerance test: 140–199 mg/dL [7.8–11.0 mmol/L] or hemoglobin A1c (HbA1c): 5.7–6.4%)
- Diabetic (fasting plasma glucose ≥126 mg/dL) [≥7.0 mmoL/L], 2 h after glucose tolerance test ≥200 mg/dL [≥11.1 mmoL/L] HbA1C ≥6.5%)
- Waist circumference ≥102 cm in men and ≥88 cm in women.

Exclusion criteria

- Patients denying consent
- Patient <18 years and >65 years
- Pregnant females
- Alcoholic liver disease
- Viral hepatitis B and C
- Autoimmune hepatitis
- Drug-induced hepatitis
- Hepatitis B and human immunodeficiency virus or hepatitis C virus co-infection
- History of cirrhosis and other liver diseases.

Methodology

A total of 302 individuals were enrolled in the study, and the individuals underwent detailed history evaluation, physical examination, anthropometric measurement, laboratory investigations, and ultrasonography of the abdomen. A history of diabetes mellitus, hypertension, hypothyroidism, and alcohol consumption was obtained. Individuals taking excessive alcohol consumption (>20 g/day for women and >30 g/day for men) were excluded from the study. Anthropometric measurements included weight, height, and waist circumference. Body mass index (BMI) was calculated using the Quetelet index. According to WHO, a BMI of 18.5–24.9 kg/m^2 is normal weight, BMI ≥25.0 kg/m^2 is overweight, and BMI ≥30.0 is obesity.

The existence of at least two of the following findings was deemed to indicate the metabolic syndrome.

- Waist circumference ≥88 cm in women and ≥102 cm in men
- Blood pressure ≥130/85 mmHg or specific antihypertensive drugs
- Plasma Triglycerides ≥150 mg/dL or specific TG-lowering drugs
- Plasma HDL-C <40 mg/dL for men and <50 mg/dL for women or specific lipid-lowering drugs
- Fasting plasma glucose levels between 100 and 125 mg/dL or 2 h postprandial glucose levels between 140 and 199 mg/dL or glycosylated hemoglobin (HbA1c) between 5.7% and 6.4%
- Homeostasis model assessment for assessing insulin resistance (HOMA-IR) ≥2.5

- High-sensitivity C-reactive protein (Hs-CRP) levels >2 mg/L.

Blood samples for fasting plasma glucose levels, fasting insulin levels, thyroid function test, and fasting lipid profile were collected following an overnight fast of 12 h. Biochemical parameters, including aspartate aminotransferase (AST), alanine transaminase (ALT), gamma-glutamyl transferase (GGT), thyroid function test, and fasting insulin level were measured using ARCHITECT i2000SR (Abbott, IL). Lipid profile levels were measured using a Cholestech LDX analyzer based on the spectrophotometer principle. HbA1C level is measured using HbA1C analyzer (Bio Red) based on a high-performance liquid chromatography method. Hs-CRP level is measured using selection Pro-M based on the immunoturbidimetric method. Ultrasound (USG) of the abdomen was performed by a single-blinded radiologist using a VIVID T8 GE Machine for diagnosis of fatty liver.

HOMA-IR

$$\text{HOMA} - \text{IR} = \frac{\text{Glucose}\left(\text{mg} / \text{dl}\right) \times \text{Insulin m IU} / \text{L}}{22.5}$$

Insulin Resistance = ≥2.5.

Biochemical scores for hepatic steatosis

HSI

HSI = 8×(ALT/AST) + BMI + 2(if type2 diabetes) + 2(if female)

HSI <30 excludes MAFLD, HSI ≥36 confirms a diagnosis of MAFLD.

Biochemical scores for advanced fibrosis

Fibrosis score (FIB-4):

$$\text{FIB} - 4 = \frac{\text{Age}\left(\text{years}\right) \times \text{AST}\left(\text{U} / \text{L}\right)}{\text{Platelets}\left(10^9 / \text{L}\right) \times \text{ALT}^{1/2}\left(\text{U} / \text{L}\right)}$$

The upper limit for AST was 40 (IU/L)

- FIB-4 values (<1.30) exclude the presence of advanced fibrosis
- FIB-4 values (>2.67) indicate advanced fibrosis.

Data analysis

Statistical analysis was performed using the Statistical Package for Social Sciences version 23 IBM (USA). The data were initially processed and coding was performed on MS Excel. The normality of data was checked using Kolmogorov–Smirnov, Shapiro–Wilk tests, and student t-test data were found to be non-normally distributed. For categorical variables, frequency and proportions were used in the descriptive analysis, whereas mean and standard deviations were used for continuous variables. P<0.05 was considered significant.

Ethical considerations

This study follows the Declaration of Helsinki and has been approved by the Institutional Ethical and Research Committee (Certificate No. 2126/IEC/I/2023-2024). Written informed consent was obtained from all participants, ensuring confidentiality, anonymity, and voluntary participation.

RESULTS

The study included 302 cases of these 165 were diagnosed with MAFLD, thus resulting in a prevalence of 54.63% which was significantly higher among participants, with notable differences in anthropometric and biochemical parameters between MAFLD and non-MAFLD groups, whereas the hepatic steatosis score was markedly elevated in MAFLD cases, confirming the association between metabolic dysfunction and liver fat accumulation (Table 1). We found that in both males and females, the prevalence of MAFLD rose with age, with the majority of cases occurring in the 50–59 years age group (Table 2). Ultrasonography findings revealed that a substantial proportion of MAFLD cases had fatty liver changes, predominantly Grade I (53.3%) and Grade II (35.75%), whereas Grade III was rare (10.92%) (Table 3). The prevalence of comorbidities such as obesity (81.81%), metabolic syndrome (82.42%), dyslipidemia (66.06%), hypertension (37.57%), hypothyroidism (37.57%), and T2DM (46.06%) were significantly higher in the MAFLD group compared to the non-MAFLD group (Table 4). Furthermore, fibrosis risk assessment using the FIB-4 Index demonstrated that MAFLD patients were more likely to have an intermediate or high risk of advanced fibrosis compared to non-MAFLD cases, highlighting the potential for disease progression (Table 5).

DISCUSSION

In our study, a total of 302 individuals were included, with a median age of 50 years for males and 48 years for females among MAFLD patients. The prevalence of MAFLD was 54.63%, which aligns with findings from similar studies conducted in Asian populations, where MAFLD prevalence ranges from 45% to 60%.[8] The association between MAFLD and obesity (81.81%) was significant, consistent with a study by Chalasani et al., (2023) which reported an 80% prevalence of obesity among MAFLD patients.[7]

A further breakdown of demographic data revealed that metabolic syndrome was highly prevalent among MAFLD

Table 1: Comparison of anthropometric and biochemical parameters between MAFLD and non-MAFLD patients

Parameters	MAFLD (n=165)	Non-MAFLD (n=137)	P-value
Anthropometric (Mean±SD)			
BMI (kg/m^2)	28.7903±04.4688	24.6624±03.2485	<0.0001
Mean W.C (cm)	108.50±12.95	95.40±12.86	<0.0001
Biochemical parameters (Mean±SD)			
Sr. protein (mg/dL)	7.011±0.701	7.04±0.747	0.7181
Sr. albumin (mg/dL)	3.86±0.437	3.84±0.514	0.7181
SGOT (IU/L)	44.95±19.96	38.74±19.98	0.0075
SGPT (IU/L)	48.19±33.48	36.13±33.41	0.0020
GGT (IU/L)	62.02±47.85	23.33±12.57	<0.0001
Sr. ALP (IU/L)	210.32±63.374	182.54±62.459	0.0395
Sr. cholesterol (mg/dL)	209.23±62.73	172.20±62.59	<0.0001
Sr. triglycerides (mg/dL)	166.34±59.71	146.63±59.46	0.0045
HDL (mg/dL)	48.77±10.27	49.46±08.95	0.5385
LDL (mg/dL)	102.12±46.20	77.12±45.84	<0.0001
VLDL (mg/dL)	34.04±15.88	30.56±13.11	0.0413
Sr.TSH (uIU/mL)	21.19±26.81	08.11±26.69	<0.0001
F.T4 (ng/dL)	0.90±0.32	01.13±0.32	<0.0001
F.T3 (pg/mL)	1.89±0.85	02.38±0.85	<0.0001
Fasting insulin level (uIU/ML)	13.5221±10.6462	8.2397±10.6178	<0.0001
Fasting blood sugar level (mg/dL)	135.7212±38.5358	125.0802±38.5205	0.0175
HOMA-IR	4.91±4.41	2.78±4.40	<0.0001
HbA1c (%)	7.81±2.65	6.49±2.55	<0.0001
hs-CRP	3.33±3.35	1.07±3.18	<0.0001
Hepatic steatosis score	39.1761±05.0351	32.256±5.0010	<0.0001

BMI: Body mass index, HDL: High-density lipoprotein, LDL: Low-density lipoprotein, VLDL: Very-low-density lipoprotein, HOMA-IR: Homeostasis model assessing of insulin resistance, HbA1c: Hemoglobin A1c, hs-CRP: High-sensitivity C-reactive protein, SGOT: Serum glutamic oxaloacetic transaminase, SGPT: Serum glutamate pyruvate transaminase, GGT: Gamma-glutamyl transferase, ALP: Alkaline phosphatase, WC: Waist circumference

Table 2: Distribution of study participants in terms of gender and age group among MAFLD

Age group (years)	Male (n=64)		Female (n=101)	
	No.	%	No.	%
18–39	12	18.75	25	24.75
40–49	10	15.63	26	24.74
50–59	29	45.31	30	29.70
60–65	13	20.31	21	20.79
Mean and SD	50.23±09.321		48.762±09.262	

Table 3: USG findings in study participants among MAFLD and non-MAFLD

USG finding	MAFLD cases (n=165)	Percentage	Non-MAFLD cases (n=137)	Percentage
Fatty liver grade-I	88	53.33	20	14.59
Fatty liver grade-II	59	35.75	1	0.72
Fatty liver grade-III	18	10.92	0	0

USG: Ultrasound, MAFLD: Metabolic-associated fatty liver disease

individuals, affecting 82.42% of cases. Hypertension and hypothyroidism were also more frequent among the MAFLD group, at 37.57% each. These results support earlier studies that have linked metabolic disorders with the pathogenesis of MAFLD.[9]

A significant proportion of MAFLD patients (46.06%) were diagnosed with T2DM, emphasizing the close interplay between insulin resistance and hepatic steatosis. Our study revealed that fasting insulin levels were significantly elevated in MAFLD patients compared to non-MAFLD individuals (13.52±10.64 uIU/mL vs. 8.23±10.61 uIU/mL, P<0.0001). Comparatively, a study by Targher et al. (2022) found higher fasting insulin levels (15.21±9.87 uIU/mL) in MAFLD patients. However, both studies confirm that insulin resistance is a major driver of MAFLD.[10]

Dyslipidemia was significantly more prevalent in the MAFLD group (66.06%) than in non-MAFLD individuals (45.25%, P<0.0001). Lipid profile analysis revealed elevated LDL (102.12±46.20 mg/dL vs. 77.12±45.84 mg/dL, P<0.0001), triglycerides (166.34±59.71 mg/dL vs. 146.63±59.46mg/dL, P=0.0045), and VLDL (34.04±15.88mg/dL vs. 30.56±13.11 mg/dL, P=0.0413) in MAFLD patients. Byrne and Targher (2023) reported similar findings, with LDL levels elevated in 68% of MAFLD cases, further reinforcing the association between lipid abnormalities and hepatic steatosis.[4]

Hypothyroidism was significantly more prevalent in the MAFLD group (37.57%) than in non-MAFLD individuals (13.15%, P<0.0001). Thyroid function test results revealed

significantly higher serum TSH levels in MAFLD patients (21.19±26.81 uIU/mL) compared to non-MAFLD individuals (8.11±26.69 uIU/mL, P<0.0001). A study by Bernal-Reyes et al. (2022) also reported an increased prevalence of hypothyroidism (35%) among MAFLD patients, closely matching our findings. This supports the hypothesis that thyroid dysfunction exacerbates metabolic abnormalities in MAFLD.[4]

BMI was significantly higher in MAFLD patients (28.79±4.46 kg/m^2) compared to non-MAFLD individuals (24.66±3.24 kg/m^2, P<0.0001). In addition, mean waist circumference was significantly greater in MAFLD patients (108.50±12.95 cm) than in non-MAFLD individuals (95.40±12.86 cm, P<0.0001). These findings align with Eslam et al. (2023), who reported a similar trend, with a mean BMI of 29.2 kg/m^2 among MAFLD patients. This consistency highlights the universal impact of central obesity on MAFLD risk.[11]

Liver function tests showed significantly elevated levels of AST, ALT, and GGT in MAFLD patients compared to non-MAFLD individuals. Elevated transaminase levels, particularly ALT, have been recognized as an early biochemical marker of hepatic dysfunction in MAFLD. This supports the results of Bernal-Reyes et al., (2022) who found higher transaminase levels in MAFLD patients.[4] Notably, the AST/ALT ratio remained below 1 in the early stages but reversed in cases progressing toward fibrosis, reinforcing the importance of liver enzyme monitoring in MAFLD patients.

Table 4: Comparison of comorbidity prevalence between study participants with MAFLD and non-MAFLD

Comorbidity	MAFLD (n=165)		Non-MFALD (n=137)	
	No.	%	No.	%
Obesity	135	81.81	64	46.71
Type 2 diabetes mellitus	76	46.06	45	32.84
Metabolic syndrome	136	82.42	56	40.87
Dyslipidemia	109	66.06	62	45.25
Hypertension	62	37.57	29	21.16
Hypothyroidism	62	37.57	18	13.15

MAFLD: Metabolic-associated fatty liver disease

In addition, serum albumin levels were relatively lower in MAFLD patients. The elevated GGT levels in MAFLD patients suggest an underlying oxidative stress component, as previously reported in a study by Younossi et al. (2022).[2] This suggests that oxidative damage plays a pivotal role in the pathogenesis of MAFLD, potentially accelerating its progression to more severe liver conditions.

Fasting blood glucose and HOMA-IR levels were significantly higher in the MAFLD group compared to non-MAFLD individuals, indicating a strong link between insulin resistance and hepatic steatosis. This aligns with Chalasani et al. (2023), who found that insulin resistance was present in nearly 75% of MAFLD cases.[7] The correlation between increased HOMA-IR and hepatic steatosis supports the hypothesis that insulin resistance plays a pivotal role in MAFLD pathogenesis.

HbA1C levels were also significantly elevated among MAFLD patients, suggesting chronic glycemic dysregulation. This finding is in concordance with a study by Byrne and Targher (2023), which demonstrated that poor glycemic control accelerates the progression of MAFLD to more severe forms of liver disease, including cirrhosis.[4]

Hs-CRP levels were markedly elevated among MAFLD patients, reflecting increased systemic inflammation. Research by Tilg et al. (2023) identified hs-CRP as a reliable predictor of hepatic fibrosis in MAFLD patients, reinforcing its importance in disease monitoring.[12]

USG findings revealed that fatty liver changes were significantly more prevalent in MAFLD patients, with 53.33% classified as Grade I, 35.75% as Grade II, and 10.92% as Grade III. These results are consistent with Younossi et al. (2022), who found that 55% of MAFLD patients had Grade I steatosis, whereas Grade III cases remained under 12%. This suggests that USG findings are relatively consistent across different populations.

The HSI was significantly higher in MAFLD patients, with a P<0.0001, confirming the strong association between steatosis severity and metabolic dysfunction. This finding

Table 5: Risk for advanced fibrosis according to fibrosis-4 index score

Study Group	Low risk F1B4 <1.3		Intermediate risk F1B4=1.3-2.67		High risk F1B4 >2.67		Chi-squared test
	No.	%	No.	%	No.	%	
MAFLD cases (n=165)	77	46.66	54	32.72	34	20.62	χ^2=14.1
Non-MAFLD cases (n=137)	81	59.12	48	35.03	08	05.85	P<0.001

MAFLD: Metabolic-associated fatty liver disease

supports previous studies by Eslam et al. (2023), which emphasized the utility of non-invasive indices such as HSI in predicting disease severity and progression.[11]

FIB-4 index score and risk of advanced fibrosis: FIB-4 index analysis indicated that MAFLD patients were at a significantly higher risk of developing advanced fibrosis. Among MAFLD cases, 20.62% had a high-risk FIB-4 score (>2.67) compared to only 5.85% of non-MAFLD individuals (P<0.001). This closely matches the findings of Tilg et al. (2023), who reported that 22% of MAFLD patients exhibited high-risk FIB-4 scores.[12] Such consistency highlights the predictive value of FIB-4 for fibrosis progression.

Limitations of the study

This single-center, cross-sectional study has limited generalizability, potential recall bias, and less sensitive ultrasonography for hepatic steatosis detection.

CONCLUSION

The study highlights a high prevalence of MAFLD in a tertiary care hospital in Bundelkhand, strongly linked to obesity, T2DM, dyslipidemia, metabolic syndrome, hypertension, elevated BMI, and increased waist circumference. A significant proportion of cases showed an elevated risk of hepatic fibrosis, emphasizing the need for early screening, lifestyle modifications, and long-term monitoring. Given the rising burden of metabolic disorders, targeted public health strategies, including awareness programs and preventive measures, are essential to mitigate MAFLD progression and reduce its complications in this region.

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Authors' Contributions:
NR, KC, AS, AAS, and GS- Definition of intellectual content, literature survey, prepared the first draft of the manuscript, implementation of study protocol, data collection, data analysis, manuscript preparation and submission of article, concept, design, clinical protocol, manuscript preparation, editing, and manuscript revision, design of study, statistical analysis and interpretation, review manuscript, review manuscript, literature survey, coordination, and manuscript revision.

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