IBOM MEDICAL JOURNAL
Vol.19 No.3 | July - September 2026 | Pages 548-558
www.ibommedicaljournal.org

HOMA-IR Values and Dysglycaemic Patterns among Patients with Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) at a Tertiary Health Centre in Nigeria.

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Abstract

Background: Metabolic dysfunction-associated steatotic liver disease (MASLD) coexisting with type 2 diabetes mellitus (T2DM) are increasingly prevalent although the underlying mechanisms remain unlear. This study aimed to determine the prevalence of T2DM and prediabetes among patients with MASLD and to evaluate the role of insulin resistance in the development of dysglycaemia.
Methodology: This hospital-based cross-sectional study included 109 adult patients with MASLD attending the University of Calabar Teaching Hospital over a 12-month period. Anthropometric measurements and fasting blood samples were obtained following informed consent and ethical approval. Fasting plasma glucose and fasting insulin concentrations were measured, and insulin resistance was assessed using the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR).
Results: A total of 109 participants were enrolled, comprising 47 (43.1%) males and 62 (56.9%) females, with a mean age of 54.8 ± 7.2 years. Based on ADA criteria, 51 (46.8%) participants had type 2 diabetes mellitus, 19 (17.4%) had prediabetes, and 39 (35.8%) had normal glycaemic status. The prevalence of diabetes mellitus increased with the severity of hepatic steatosis and was highest among participants with grade 3 fatty liver. HOMA-IR values were elevated in all patients with MASLD, with progressively higher values observed among those with prediabetes and highest among those with type 2 diabetes mellitus.
Conclusion: There is a high prevalence of type 2 diabetes mellitus and prediabetes among patients with MASLD attending a tertiary healthcare facility. The findings suggest that insulin resistance is strongly associated with dysglycaemia and may play an important role in the metabolic disturbances associated with MASLD.

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DOI: 10.61386/imj.v19i3.1179

Keywords: MASLD; Type 2 diabetes mellitus; Prediabetes; Insulin resistance; HOMA-IR; Dysglycaemia.

Introduction

Globally, Metabolically-dysfunction-associated steatotic liver disease(MASLD), formerly called

NAFLD, is the most common chronic liver disease, affecting more than 30% of the world's population.[1,2,3,4] Currently, 1 in 4 people suffer from MASLD4. In 2020, several experts and stakeholders came together and proposed Metabolically-dysfunction-associated steatotic liver disease (MASLD) as the new name for non-alcoholic fatty liver disease (NAFLD).NAFLD had always been a diagnosis of exclusion. Various pathologies must be ruled out before a final diagnosis of NAFLD (deposition of fats in hepatocytes) can be made. These include, but are not limited to, excessive alcohol ingestion (>20g/day in men and >10g/day in women), effects of medications on liver injury, autoimmune conditions, systemic diseases affecting the liver, and chronic viral hepatitis.[5,6] This made it very difficult to make the diagnosis,and there are many possibilities of missed diagnoses. However, the terminology, Metabolically Dysfunctional-Associated Steatotic Liver Disease (MASLD has its link to metabolic conditions. It reflects the pathogenesis of the diseases more than NAFLD, with a view to enhancing patient care.[7] The implication is that MASLD has a higher index of identifying patients with high risk for metabolic dysfunction than NAFLD, as it is associated with metabolic risk factors.[8] The American Association for the Study of Liver Diseases(AASLD) and the European Association for Liver Studies (EALS) have adopted the name henceforth for clinical diagnosis and research.[9]

Hence, MASLD is the new nomenclature, and its diagnosis is based on the presence of 1. hepatic steatosis (diagnosed by histological biopsy showing steatosis or imaging to detect steatosis or identification of blood markers of fat accumulation) PLUS ANY ONE of the three below[10-]

(a) Overweight(BMI≥25kg/m^2) or obesity (BMI>30 kg/m^2)

(b) Presence of type 2 diabetes mellitus

(c) Evidence of metabolic dysfunction/dysregulation

The last point, evidence of metabolic dysfunction/dysregulation, is considered when two or more of the seven following features exist in a patient:

-Waist circumference>90cm in men, 80cm in women.

-Blood pressure >130/80mmHg or on antihypertensive agent

-Triglycerides >1.70mmol/L(150mg/L) or on antilipidemic agent

-High density lipoprotein cholesterol (HDL-C) <1.0mmol/L(40mg/dL) in men, and <1.3mmol/L(50mg/dl) in women

-Fasting plasma glucose (5.6-6.9mmol/l)-prediabetes

-HOMA IR score of 2.5

-Plasma highly sensitive C-reactive protein>2mg/L

Based on the above, MASLD identifies patients with liver-related and cardiovascular events, as it signifies the interconnection of liver and cardiovascular disease (CVD).[11] It explicitly highlights the role of metabolic dysfunction in both conditions and can help identify patients at risk for major adverse cardiovascular events and liver complications.[11] Patients with MASLD exhibit a significantly increased risk of CVD due to shared pathways like inflammation and insulin resistance, making early detection and integrated cardiometabolic risk management crucial for improving outcomes in these individuals.[11,12] MASLD is closely linked to cardiovascular events and involvement, as well as high blood pressure (46.2%)(95CI=33.76-60.30), dyslipidemia, and diabetes mellitus (24.66%)95CI=20.76-28.78)[12,13,14], as well as chronic kidney diseases(23.92%)(95CI=14.13-35.35), which are all comorbidities.[15,16]

Metabolically-dysfunction-associated steatotic liver disease (MASLD) increases the risk of patients developing type 2 diabetes, and also, type 2diabetes mellitus significantly increases the risk of developing MASLD, creating a bidirectional synergy of cause and effect between the two, each raising the risk of the other.[17,18] Several other studies have shown that MASLD increases the incidence of type 2diabetes mellitus by two-fold.[19,20]

Considering the high prevalence of MASLD globally and in Nigeria, and its link to DM, this study aims to determine the prevalence of DM in MASLD patients in our centre and the pattern of HOMA-IR scores in affected participants.

Methodology

Study area

The site of study was the Metabolic,

Gastroenterology, Diabetes, and Radiology Clinics of the University of Calabar Teaching Hospital (UCTH) located within the Calabar Metropolis in Nigeria. The hospital was established in 1979 as an affiliate of the University of Calabar
and offers training of the undergraduate students of the university as well as post-graduate training of resident doctors in various specialties and receives referrals from various hospitals within the state as well as from nearby states of Akwa Ibom, Rivers and Ebonyi; and even from Cameroon, a neighbouring country.

Study design

This was a descriptive cross-sectional study with an analytical component, done over 12-months. The study was conducted among adult diabetic patients aged 20-70years with MASLD.

Study population

The study population consisted of adult MASLD patients (≥18 years) receiving care at the University of Calabar Teaching Hospital who met the inclusion criteria.

Inclusion Criteria

Participants were eligible if they:

- Were aged between 20 and 70 years.
- Had a radiological diagnosis of hepatic steatosis consistent with MASLD.
- Met the accepted diagnostic criteria for MASLD.
- Provided written informed consent.

Exclusion criteria

1. Patients with diabetic nephropathy or type 1 DM
2. Patients with MASLD who objected to this study.
3. Declined participation
4. MASLD patients who were older than 70 years.
5. Patients on insulin therapy or other drugs for liver pathology
6. Patients who were hepatitis B and C seropositive

Sample size determination

The sample size was calculated using the Cochran formula for calculating sample size, for a known prevalence rate.[21]

$N = Z^2PQ/d^2$

Where:
N= the desired sample size
z = the standard normal deviation usually set at 1.96 which corresponds to 95 percent confidence level
p = 0.064(the proportion in the target population estimated to have Type 2 DM in Nigeria)
6.4% (Akinkungbe *et al*., 2013)
q = (0.936) (1-p)
d = degree of accuracy set at 0.05

$N = (1.96)^2 \times 0.064 \times 0.936 / 0.052$

$N = 0.23012 / 0.0025$

$N = 95$

Using an attrition rate of 20%, this comes to about 19 more participants. Added to 95 gives 114 participants which approximates to 120.One hundred and twenty participants were recruited, but eleven were excluded or lost due to incomplete data, leaving 109 participants for final analysis.

Ethical consideration/approval

Ethical approval was obtained from the Health Research Ethics Committee (HREC) of the University of Calabar Teaching Hospital (NHREC/07/10/2012 as UCTH NHREC Registration number and UCTH/HREC/33/Vol.111/123 as the HREC protocol assigned number) before carrying out the study. The study was carried out without coercion of participants, and a signed informed consent was obtained from all participants.
Three research assistants were recruited and trained by me for this study. The training involved filling out the bio data form, taking anthropometric measurements and blood sample collection procedure under aseptic conditions.

Study tools

A pretested, semi-structured, interviewer-administered questionnaire was used for data collection, which was divided into four sections. Section A collected sociodemographic and biodata information from eligible participants; Section B contained information on cardiometabolic risk

factors, while Section C was used to record anthropometric parameters.
Section D contained information on laboratory investigations and the values of calculated and quantified analytes in this study.

Variables and their measurements
Anthropometric measurements were done according to the WHO guidelines for taking physical measurements. These included the weight, height (and by extension, body mass index, BMI), and blood pressure.

Data collection
Five milliliters (5mL) of blood were collected for the following investigations. Three milliliters (3mL) were put into a separator tube for serum fasting insulin assay, and two milliliters (2mL) into a fluoride oxalate vacutainer for fasting plasma glucose estimation. The blood was allowed to stand for 20-60 minutes before it was put into a centrifuge and spun at 3000 revolutions per minute (rpm) for five minutes, using a centrifuge. The resultant serum supernatant was transferred to an appropriate storage tube and assayed for fasting plasma insulin, fasting plasma glucose. HOMA-IR values were calculated from the above two by the equation:

HOMA-IR = (fasting plasma glucose in mmol/L X fasting insulin in microU/L)/22.5[22]
All analyses were done according to standard procedures and methodology.
The diagnosis of diabetes mellitus was made by the current WHO guidelines and that of the American Diabetes Association (ADA); in which a fasting plasma glucose value of 7mmol/L(126 mg/dl) defines diabetes, while Impaired fasting glucose(prediabetes) values were pegged at between 5.7 and 6.9mmol.

Quality Control
Internal quality control procedures were implemented throughout the study.High-level and low-level quality control materials were analyzed with each batch of glucose and insulin assays. Within-run and between-run coefficients of variation were monitored to ensure analytical precision and accuracy.Research assistants received standardized training in questionnaire administration, anthropometric measurements, and specimen collection procedures before commencement of the study.

Statistical Analysis
The data obtained from the study were compiled in the form of tables and entered into a Microsoft Excel spreadsheet, and statistical analyses were performed using Statistical Package for Social Sciences (SPSS) Version 25.0 (SPSS, Chicago, Illinois, USA). The sex, age, BMI, and levels of glucose, insulin, and HOMAIR were analyzed using mean, standard error of mean, and analysis of variance (ANOVA). The levels of significance at a 95% confidence interval were taken at $p<0.05$. Violation of these assumptions was addressed by the transformation of variables. Results of normally distributed parameters were presented as mean ± standard deviation (SD). Between groups, differences were assessed using analysis of variance (ANOVA) for normally distributed. Pearson's correlation and regression studies were also employed in the data analysis. The level of statistical significance was set at $p<0.005$.

Results
Sociodemographic and Clinical Characteristics of Study Participants
A total of 109 participants with Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) were included in the study. Of these, 47 (43.1%) were males and 62 (56.9%) were females, giving a male-to-female ratio of 1:1.3. The mean age of the study population was 54.8 ± 7.2 years, with the majority of participants (62.4%) aged between 56 and 65 years.Based on the American Diabetes Association (ADA) criteria, 51 (46.8%) participants had type 2 diabetes mellitus, 19 (17.4%) had impaired fasting glucose (prediabetes), while 39 (35.8%) had normal fasting plasma glucose levels.Fasting insulin concentrations were elevated in 17 (15.6%) participants, whereas 92 (84.4%) had values within the reference range.Assessment of insulin resistance using HOMA-IR showed that 29 (26.6%) participants were insulin sensitive, 18 (16.5%) had normal insulin sensitivity, 7 (6.4%) had early insulin resistance, and 55 (50.5%) had established (late) insulin resistance.

Table 1: Socio-demographic and Clinical Characteristics of Participants with MASLD (N = 109)

Variable	Frequency	Percentage
Sex		
Male	47	43.1
Female	62	56.9
Age group/years		
36-45	7	6.4
46-55	24	22.0
56-65	68	62.4
>65	10	9.2
Glycemic status		
Fasting glucose category		
Diabetes	51	46.8
Impaired fasting glucose	19	17.4
Normal	39	35.8
Insulin level		
Normal	92	84.4
Elevated	17	15.6
HOMA-IR Category		
Insulin sensitivity	29	26.6
Normal	18	16.6
Early insulin resistance	7	6.4
Late insulin resistance	55	50.5
Waist circumference		
Low risk	46	42.2
High risk	21	19.3
Very high risk	42	38.5
BMI category		
Underweight	4	3.7
Normal	42	38.5
Overweight	48	44.0
Obesity	15	13.8
Systolic BP (mmHg)		
Normal	80	73.4
Raised	29	26.6
Diastolic BP (mmHg)		
Normal	104	95.4
Raised	5	4.6

Correlation Between HOMA-IR and Plasma Glucose

The fasting glucose values show that 51(46.8%) were diabetic, 19(17.4%) had impaired fasting glucose (prediabetes), while 39(35.8%) had normal fasting glucose levels(see Figure 1). Insulin level was normal for 92(84.4%) but high for 17(15.6%). HOMA IR values showed insulin sensitivity among 29(26.6%), normal among 18(16.6%), early insulin resistance among 7(6.4%), and late insulin resistance among 55(50.5%). Those who had normal BMI were 42(38.5%), obese individuals constituted 15(13.8%), systolic blood pressure was raised among 29(26.6%), while diastolic blood pressure was raised among only 5(4.6%).

The proportion with a severe grade of fatty level (grade 3) was higher among those with type 2 diabetes mellitus compared with their non-diabetic counterparts (60.0% versus 40.0%). Conversely, the proportion of those with a mild level of fatty liver was highest among those who were not diabetic compared with those who were diabetic. However, the difference was not statistically significant (p=0.081)(see Table 2 and Figure 2).

Table 2: Comparison of the pattern of fatty liver among those with and without Type 2 diabetes mellitus

Variable	Diabetes Present n=51	Diabetes Absent n=58	Total N=109	*p*-value
Severity/pattern of fatty liver				
Grade 1 (Mild)	15(34.1)	29(65.9)	44(100.0)	0.081
Grade 2 (Moderate)	24(53.3)	21(46.7)	45(100.0)	
Grade 3(Severe)	12(60.0)	8(40.0)	20(100.0)	

Table 3: Factors associated with the severity of fatty liver among study participants (n=109)

Variable	Mild n=44	Moderate n=45	Severe n=20	Total N=109	FET	p-value
Sex						
Male	18(38.3)	16(34.9)	13(27.7)	47(100.0)	5.941	0.080
Female	26(41.9)	29(46.8)	7(11.3)	62(100.0)		
Age group/years						
36-45	2(28.6)	3(42.9)	2(28.6)	7(100.0)	4.563	0.696
46-55	8(33.3)	11(45.8)	5(20.8)	24(100.0)		
56-65	30(44.1)	25(36.8)	13(19.1)	68(100.0)		
>66	4(40.0)	6(60/0)	0(0.0)	10(100.0)		
Fasting glucose category						
Diabetes	15(29.4)	24(47.1)	12(23.5)	51(100.0)	7.948	0.073
Impaired	7(36.8)	10(52.6)	2(10.5)	19(100.0)		
Fasting glucose						
Normal	22(56.4)	11(28.2)	6(15.4)	39(100.0)		
Insulin level						
Normal	36(39.1)	40(43.5)	16(17.4)	92(100.0)	1.359	0.550
High insulin	8(47.1)	5(29.4)	4(23.5)	17(100.0)		
HOMA IR						
Insulin sensitivity	17(58.6)	8(27.6)	4(13.8)	29(100.0)	7.817	0.266

Table 3 shows factors associated with the severity of fatty liver among study participants.

Association Between Type 2 Diabetes Mellitus and Severity of Fatty Liver

The proportion of those with severe fatty liver was higher among males compared with females (27.7% versus 11.3%), but the difference was not statistically significant (p=0.080). Age was inversely related to severe fatty liver, although the relationship was not statistically significant (p=0.696). Also, those who were diabetic accounted for a higher proportion of those with severe fatty liver compared with either impaired fasting glucose or normal glucose; however, the difference was not statistically significant (p=0.073). Furthermore,

there was no statistically significant relationship between severity of fatty liver and HOMA-IR (p=0.266).

HOMA-IR as a Predictor of Type 2 Diabetes Mellitus

Regression studies showed that HOMA IR (t=8.427, p=0.000, 95% CI=0.145-0.235) is a strongly significant positive predictor (Table 4). This study further showed that the severity of fatty liver was not associated with age and sex of the studied subjects (p-value =0.08). However, subjects with type 2DM had a severe grade of fatty liver, compared to those with either impaired fasting glucose (prediabetes) or normal glucose values. There was a very strong correlation between HOMA-IR and the plasma glucose levels (r=0.597, p=0.000). This was well established by the scatter plots in the above(see Figure 3).

Table 4. Regression Analysis of HOMA-IR as a Predictor of Type 2 Diabetes Mellitus Standardized 95.0% Confidence Interval

	Coefficient Beta	Lower Bound	Upper Bound	T	*P*- value	Rank
(Constant)		6.379	7.562	23.376	0.000	
HOMA_IR	0.604	0.145	0.235	8.427	0.000	High

As shown in Table 4, HOMA IR (t=8.427, p=0.000; 95% CI: 0.145 to 0.235) is a strong and significant positive predictor of type 2diabetes mellitus.

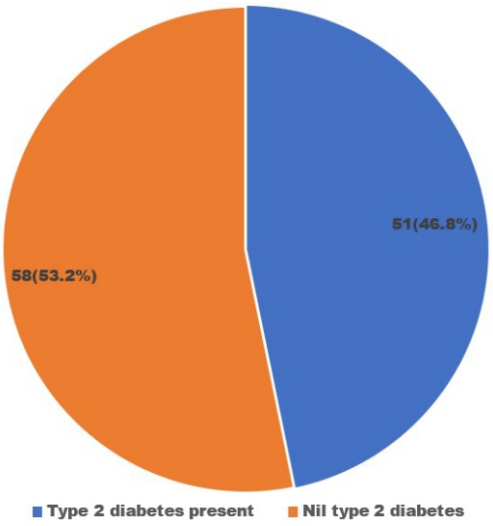
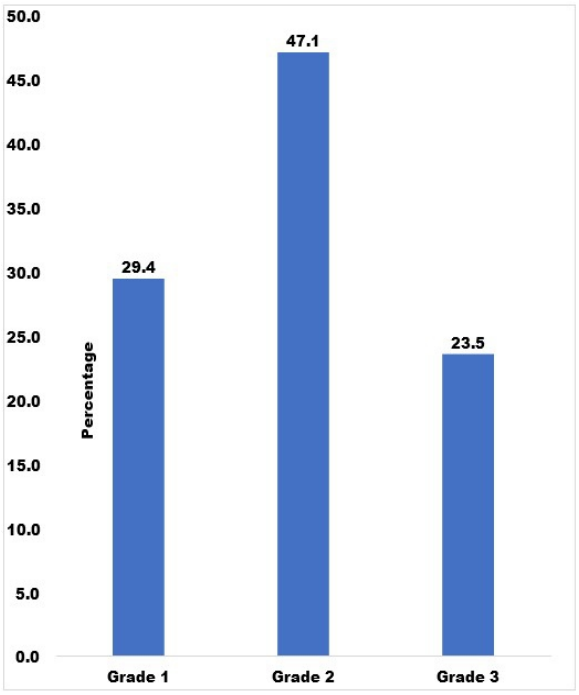


Fig. 1: Prevalence of type 2 diabetes mellitus among masld participants



Proportion of diabetics at different severities of fatty liver

Fig. 2:Type 2 diabetes stratified to compare with the severity of fatty liver

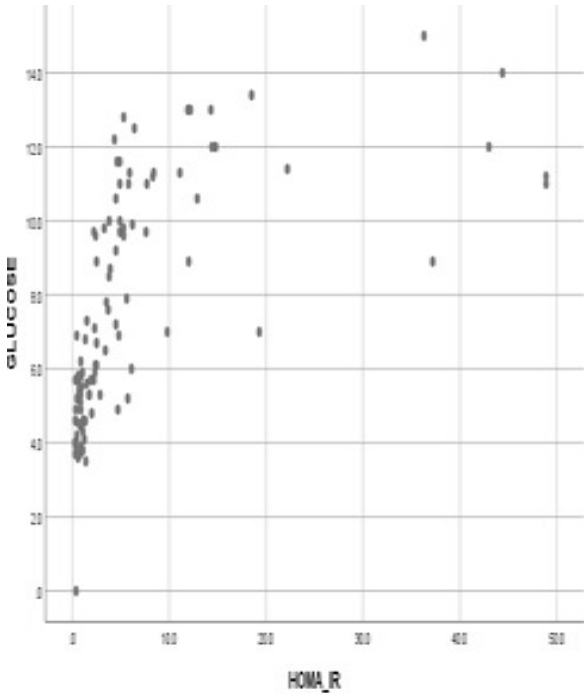


Fig. 3:Scatter-plot showing positive (direct) correlation between homa ir and glucose level (dm) among patients with fatty liver

Summary of Principal Findings

The principal findings of this study are:

1. Nearly one-half (46.8%) of patients with MASLD had type 2 diabetes mellitus.
2. Prediabetes was present in 17.4% of participants.
3. The prevalence of diabetes increased with increasing severity of hepatic steatosis.
4. More than half of the participants demonstrated established insulin resistance.
5. HOMA-IR was a strong and statistically significant predictor of type 2 diabetes mellitus.

A positive correlation existed between HOMA-IR and fasting plasma glucose levels, emphasizing the role of insulin resistance in the metabolic abnormalities associated with MASLD.

Discussion

This study assesses the dysglycemic (DM and prediadiabetes) pattern and HOMAIR values in MASLD patients attending a tertiary hospital in Nigeria. From the study, more females (67.5%) formed the study group than males (46.1%).The mean age of the study population was 54.8 ± 7.2 years, with the majority of participants (62.4%) aged between 56 and 65 years.This tallies with the findings of other authors and researchers who opined that the mean age of onset with MASLD in most people was around 56±6.4years.[23,24]

.In this index study, type 2 diabetes mellitus was identified as 46.8% participants with MASLD, compared with non- diabetic patients (31 out of 109).

The predominance of female participants and the mean age observed in this study are consistent with previous reports.The high prevalence of diabetes and prediabetes observed in our cohort is comparable to findings from previous studies.Based on the American Diabetes Association (ADA) criteria, 46.8% participants had type 2 diabetes mellitus, 17.4% had impaired fasting glucose (prediabetes), while 35.8% had normal fasting plasma glucose levels.Onyekwere *et al* and Odenigbo *et al* opined that there is a high prevalence of DM in MASLD patients.[25,26] Other related research on this subject also found a high prevalence of DM among MASLD patients. Latif *et al* had a DM prevalence of 54.3% in their study population in those with MASLD.[27] The reverse also holds; many patients with NAFLD have a higher prevalence of type 2 diabetes mellitus than non-diabetic patients[28,29]. This suggests a bidirectional relationship between the two. With the aforementioned, it is apparent that NAFLD is a risk factor for type 2DM. In some studies, the prevalence of NAFLD in patients with type 2 DM was found to be two-fold higher than that found in the normal population[22,30]. Further studies done by other researchers have also shown that the risk of DM in patients with NAFLD appears to be a two-fold rise, even after adjusting for many risk factors[22,31]. Yet in another study, fatty liver is associated with the development of type 2 DM, independent of classical risk factors, among Korean adults.[32]

Similarly, impaired fasting glucose (prediabetes) was identified in 17.4% of the study participants with NAFLD. This prediabetic condition has the potential of progressing to type 2 diabetes mellitus if adequate intervention is not brought to bear. The value of 17.4% in our study of those with prediabetes is in tandem with the work of Byrne *et al* (20.6%)[33], Nobili(19.8%)..This prevalence is similar to a study conducted by Taharboucht *et al*. in Algeria in 2020 on 213 MASLD patients, in which prediabetes prevalence was 14.6%. Cuthbertson et al in Finland found that 25.44% of MASLD patients studied had prediabetes. Similarly, Kitazawa *et al*. reported that 2.96% (770/26,014 cases) of individuals with NAFLD met the criteria for prediabetes. It is believed that IR is the central hallmark of MASLD. Hence, the reason for the prediabetes and subsequent DM is found in most people with MASLD.[38,39] This explains the elevated HOMA IR in MASLD patients compared with the general population.[40] The HOMA IR reflects the ongoing IR before the manifestation of prediabetes or DM. This elevation was found to be higher in MASLD patients with type 2 diabetes mellitus than in those without type 2 diabetes mellitus. This finding arouses the curiosity entertained by some researchers that insulin resistance may play a significant role in the pathogenesis of MASLD via the insulin resistance pathway. These thoughts are shared with several authors (Singh *et al.*, 2014).[41] This study shows that HOMAR IR was a positive predictor of glucose levels, and by extension, the DM status. The regression weight was (p=0.000, 95% CI=0.145-0.235)(see Table 4). All patients

studied with MASLD had elevated HOMA IR, but it was higher in prediabetes, and highest in DM. This proves the MASLD has a measure of insulin resistance in its pathophysiology, and was probably responsible for the progression to prediabetes and DM in this study population. This again further explains from this study, the reason for the most severe forms of MASLD (they have higher IR than the mild or moderate MASLD) having an increased tendency to have DM (see table 5). This agrees with the works of Zheng *et al*, where elevated HOMAR IR scores were observed in MASLD subjects.[41]

This showed that high glucose values, as seen in Type 2DM patients, are usually associated with insulin resistance. It concurs with the works of Begoni *et al*[42]. A multivariable linear regression analysis for the contribution of HOMA IR to diabetes mellitus was done among adults with fatty liver, and it was found to have a high regression. This shows that 45.5 percent of the dependent variable (type 2 diabetes mellitus) may be predicted by HOMAIR, while the remaining 54.5% is attributed to other extraneous variables. The p-value of 0.000 shows this significant relationship. This further proves the high possibility of MAFLD leading to type 2 diabetes mellitus.

This study does not fully establish a relationship that men have a greater risk than women of the occurrence of DM in MASLD. This is contrary to earlier studies, which emphasized that men have DM in MASLD than women (Riazi et al., 2022; Teng *et al*., 2023).[43,44]

Clinical Implications

The high prevalence of diabetes mellitus and prediabetes observed in this study has important clinical implications. Routine screening for dysglycaemia should be incorporated into the management of patients with MASLD. Assessment of fasting plasma glucose, glycated haemoglobin (HbA1c), and, where feasible, HOMA-IR may facilitate early identification of individuals at high metabolic risk.Given the bidirectional relationship between MASLD and T2DM, an integrated multidisciplinary approach involving hepatologists, endocrinologists, chemical pathologists, family physicians, and nutrition specialists is essential for effective management.

Strengths of the Study

This study provides important local data on the relationship between MASLD, dysglycaemia, and insulin resistance in a Nigerian population. It also employed HOMA-IR as an objective marker of insulin resistance and evaluated both diabetes mellitus and prediabetes among participants with confirmed MASLD.

Conclusion

In conclusion, this study has been able to pinpoint the high prevalence of DM among MASLD patients and the possible link to the co-existing insulin resistance as a possible pathophysiology, as evidenced by the correlating HOMA IR values.

Recommendations and policy implications

1. Based on this study, we recommend that, additionally, there should be initiatives in place to offer screening for diabetes in all patients with MASLD and a follow-up done for those who are negative for DM. Because of the 2-3-fold increased risk of diabetes incidence in MASLD patients, most guidelines recommend screening for T2DM in MASLD patients. Active surveillance with serum fasting glucose and HbA1c should be included for MASLD patients. Since MASLD is a strong risk factor for T2DM, we recommend screening every 1-2 years, particularly in obese subjects or subjects with a family history of T2DM.
2. Furthermore, there should be constant sensitization programs designed to educate the public about screening for diabetes and related diseases when they are diagnosed with fatty liver.

Acknowledgment: We deeply appreciate Mrs. Precious for typing this manuscript

Grant: There was no grant obtained for this research

Conflict of interest: None declared by the authors

References

1. Boccatonda A, Andreetto L, D'Ardes D, Cocco G, Rossi I, Vicari S, Schiavone C, Cipollone F, Guagnano MT. From NAFLD to MAFLD: Definition, Pathophysiological Basis and

Cardiovascular Implications. Biomedicines. 2023 Mar 13;11(3):883. doi: 10.3390/biomedicines11030883.
2. Kolawole OJ, Oje MM, Betiku OA, Ijarotimi O, Adekanle O, Ndububa DA, et al. Correlation of alanine aminotransferase levels and a histological diagnosis of steatohepatitis with ultrasound-diagnosed metabolic-associated fatty liver disease in patients from a centre in Nigeria. BMC Gastroenterol. 2024;24:147. doi:10.1186/s12876-024-03237-4.
3. Liu, Q., Zhao, G., Li, Q. et al. A comparison of NAFLD and MAFLD diagnostic criteria in contemporary urban healthy adults in China: a cross-sectional study. BMC Gastroenterol 22, 471 (2022). https://doi.org/10.1186/s12876-022-02576-4
4. Chan WK, Chuah KH, Rajaram RB, Lim LL, Ratnasingam J, Vethakkan SR. Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD): A State-of-the-Art Review. J Obes Metab Syndr. 2023 Sep 30;32(3):197-213. doi: 10.7570/jomes23052.
5. Lupsor M, Badea R. Imaging diagnosis and quantification of hepatic steatosis: is it an accepted alternative to needle biopsy? Rom J Gastroenterol. 2005;14(4):419–25.
6. Chalasani N, Younossi Z, Lavine JE, Charlton M, Cusi K, Rinella M, Harrison SA, Brunt EM, Sanyal AJ. The diagnosis and management of nonalcoholic fatty liver disease: practice guidance from the American Association for the Study of Liver Diseases. Hepatology. 2018;67(1):328–57.
7. Alharthi, Jawahera,b; Gastaldelli, Amaliac; Cua, Ian Homerd; Ghazinian, Hasmike; Eslam, Mohammeda. Metabolic dysfunction-associated fatty liver disease: a year in review. Current Opinion in Gastroenterology 38(3):p 251-260, May 2022. | DOI: 10.1097/MOG.0000000000000823
8. Méndez-Sánchez, Nahum, Abate, María Lorena. Global multi-stakeholder endorsement of the MALD definition. Lancet Gastroenterology & Hepatology, Volume 7, Issue 2022;7(5):388-390. doi:10.1016/S2468-1253(21)00444-8. 5, 388 - 390
9. Eslam M, Newsome PN, Sarin SK, Anstee QM, Targher G, Romero-Gomez M, et al. A new definition for metabolic dysfunction-associated fatty liver disease: An international expert consensus statement. J Hepatol. 2020;73(1):202-209. doi:10.1016/j.jhep.2020.03.039.
10. Tang SY, Tan JS, Pang XZ, Lee GH. Metabolic dysfunction associated with fatty liver disease: The new nomenclature and its impact. World J Gastroenterol. 2023 Jan 21;29(3):549-560. doi: 10.3748/wjg.v29.i3.549.
11. Wang TY, Wang RF, Bu ZY, Targher G, Byrne CD, Sun DQ, Zheng MH. Association of metabolic dysfunction-associated fatty liver disease with kidney disease. Nat Rev Nephrol. 2022 Apr;18(4):259-268. doi: 10.1038/s41581-021-00519-y.
12. Tilg, H. · Effenberger, M.From NAFLD to MAFLD: when pathophysiology succeeds Nat Rev Gastroenterol Hepatol. 2020; 17:387-388
13. Lim GEH, Tang A, Ng CH, et al. An observational data meta-analysis on the differences in prevalence and risk factors between MAFLD vs NAFLD. Clin Gastroenterol Hepatol. 2023;21(3):619-629.e7. doi:10.1016/j.cgh.2022.08.037.
14. American Association for the Study of Liver Diseases (AASLD). Introducing MASLD and MetALD: New fatty liver disease nomenclature. Available from: https://www.aasld.org/new-masld-nomenclature.[cited 2026 Jun 11]. Available from: https://www.aasld.org/new-masld-nomenclature
15. Younossi ZM, Koenig AB, Abdelatif D, Fazel Y, Henry L, Wymer M. Global epidemiology of nonalcoholic fatty liver disease-Meta-analytic assessment of prevalence, incidence, and outcomes. Hepatology 2016;64(1):73–84.
16. Musso G, Gambino R, Cassader M, Pagano G. Meta-analysis: natural history of non-alcoholic fatty liver disease (NAFLD) and diagnostic accuracy of non-invasive tests for liver disease severity. Ann Med. 2011;43(8):617–49.
17. Cao, L., An, Y., Liu, H. et al. Global epidemiology of type 2 diabetes in patients with NAFLD or MAFLD: a systematic review and meta-analysis. BMC Med 22, 101 (2024). https://doi.org/10.1186/s12916-024-03315-0
18. Padda J, Khalid K, Khedr A, et al. (August 20, 2021) Non-Alcoholic Fatty Liver Disease and

Its Association With Diabetes Mellitus. Cureus 13(8): e17321. doi:10.7759/cureus.17321
19. Kosmalski, M.; Śliwińska, A.; Drzewoski, J. Non-Alcoholic Fatty Liver Disease or Type 2 Diabetes Mellitus—The Chicken or the Egg Dilemma. Biomedicines 2023, 11, 1097. https://doi.org/10.3390/biomedicines11041097
20. Rabiee B, Roozafzai F, Hemasi GR, Poustchi H, Keyvani H, Khonsari MR, Ajdarkosh H, Maadi M, Sima Saeedian F, Zamani F. The Prevalence of Non-alcoholic Fatty Liver Disease and Diabetes Mellitus in an Iranian Population. Middle East J Dig Dis. 2017 Apr;9(2):86-93. doi: 10.15171/mejdd.2017.56.
21. Cochran WG. Sampling Techniques. 3rd ed. New York: John Wiley & Sons; 1977.
22. David R. Matthews, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and β-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia. 1985;28(7):412-419. doi:10.1007/BF00280883.
23. Harrison SA, Gawrieh S, Roberts K, Lisanti CJ, Schwope RB, Cebe KM, Paradis V, Bedossa P, Aldridge Whitehead JM, Labourdette A, Miette V, Neubauer S, Fournier C, Paredes AH, Alkhouri N. Prospective evaluation of the prevalence of non-alcoholic fatty liver disease and steatohepatitis in a large middle-aged US cohort. J Hepatol. 2021 Aug;75(2):284-291. doi: 10.1016/j.jhep.2021.02.034.
24. Perumpail BJ, Manikat R, Wijarnpreecha K, Cholankeril G, Ahmed A, Kim D. The prevalence and predictors of metabolic dysfunction-associated steatotic liver disease and fibrosis/cirrhosis among adolescents/young adults. J Pediatr Gastroenterol Nutr. 2024 Jul;79(1):110-118. doi: 10.1002/jpn3.12219.
25. Onyekwere C.A., Ogbera, A.O., Balogun, B.O. (2011). Non-alcoholic fatty liver and the metabolic syndrome an urban hospital serving an African Community. Annals of Hepatology 10(2):119-124.
26. Odenigbo, C. U., Oguejiofor, O.C., Ezejiofor, O. I., Jisieke-Onuigbo, N. N., Nwaneli, C. U. Nonalcoholic Fatty Liver Disease Among Adults Attending Medical Outpatient Clinic Using Ultrasound. Orient Journal of Medicine 2020; 32:(3-4).
27. Ferdous SE, Ferrell JM. Pathophysiological Relationship between Type 2 Diabetes Mellitus and Metabolic Dysfunction-Associated Steatotic Liver Disease: Novel Therapeutic Approaches. Int J Mol Sci. 2024 Aug 10;25(16):8731. doi: 10.3390/ijms25168731.
28. Latif S, Ahsan T. Prevalence of Metabolically Dysfunctional-associated Steatotic Liver Disease (MASLD) in Persons with Obesity and Type 2 Diabetes Mellitus: A Cross-sectional Study. Euroasian J Hepatogastroenterol. 2024 Jul-Dec;14(2):129-133. doi: 10.5005/jp-journals-10018-1437.
29. Barrera F, Uribe J, Olvares N, Huerta P, Cabrera D, Romero-Gómez M. The Janus of a disease: Diabetes and metabolic dysfunction-associated fatty liver disease. Ann Hepatol. 2024 Jul-Aug;29(4):101501. doi: 10.1016/j.aohep.2024.101501.
30. Lee BW, Lee Yh, Park CY, Rhee EJ, Lee WY, Kim NH, Choi KM, Park KG, Choi YK, Cha BS, Lee DH; Korean Diabetes Association (KDA) Fatty Liver Research Group. Non-Alcoholic Fatty Liver Disease in Patients with Type 2 Diabetes Mellitus: A Position Statement of the Fatty Liver Research Group of the Korean Diabetes Association. Diabetes Metab J. 2020;44(3):382-401.
31. Dharmalingam M, Yamasandhi PG. Nonalcoholic Fatty Liver Disease and Type 2 Diabetes Mellitus. Indian J Endocrinol Metab. 2018 May-Jun;22(3):421-428. doi: 10.4103/ijem.IJEM_585_17.
32. Kim KS, Hong S, Han K, Park CY. Association of non-alcoholic fatty liver disease with cardiovascular disease and all-cause death in patients with type 2 diabetes mellitus: nationwide population-based study. BMJ. 2024 Feb 13;384:e076388. doi: 10.1136/bmj-2023-076388.
33. Byrne, Christopher D .Type 2 diabetes, prediabetes, and MASLD: who and when to screen and how to treat?The Lancet Diabetes & Endocrinology, Volume 13, Issue 8, 645– 646
34. Nobili V, Mantovani A, Cianfarani S, Alisi A, Mosca A, Sartorelli MR, Maffeis C, Loomba R, Byrne CD, Targher G. Prevalence of prediabetes and diabetes in children and adolescents with

biopsy-proven non-alcoholic fatty liver disease. J Hepatol. 2019 Oct;71(4):802-810. doi: 10.1016/j.jhep.2019.06.023.

35. Taharboucht S, Guermaz R, Brouri M, Chibane A. Pre-diabetes and NAFLD: A study of an Algerian population sample. Endocr Metab Sci. 2020;1(3-4):100060. doi:10.1016/j.endmts.2020.100060.
36. Cuthbertson DJ, Koskinen J, Brown E, Magnussen CG, Hutri-Kähönen N, Sabin M, et al. Fatty liver index predicts incident risk of prediabetes, type 2 diabetes and non-alcoholic fatty liver disease (NAFLD). Ann Med. 2021;53(1):1257-1265. doi:10.1080/07853890.2021.1956685
37. Atsushi Kitazawa, Shotaro Maeda, Yoshiharu Fukuda. Fatty liver index as a predictive marker for the development of diabetes: A retrospective cohort study using Japanese health check-up data. PLOS ONE 2021. doi.org/10.1371/journal.pone.0257352
38. Gao X, Chen T, Zhou F, Sun Y, Zhang J, Li X, Zhao W, Li Y, Shi Y, Niu K, Wang Y, Zhang Y, Zhang W. The association between different insulin resistance surrogates and all-cause mortality and cardiovascular mortality in patients with metabolic dysfunction-associated steatotic liver disease. Cardiovasc Diabetol. 2025 May 9;24(1):200. doi: 10.1186/s12933-025-02758-w. PMID: 40346671; PMCID: PMC12065324.
39. Citation: Cao W, Jiang T, Deng W, Wang S, Li X, Zhang Z, Zhang L, Lu Y, Chang M, Liu R, Wu S, Shen G, Gao Y, Hao H, Chen X, Hu L, Xu M, Yi W, Xie Y and Li M (2024) Insulin resistance has closer correlation with the occurrence of metabolic dysfunction associated steatotic liver disease diagnosed by liver biopsy. Front. Med. 11:1384927. doi: 10.3389/fmed.2024.1384927
40. Peng H, Xiang J, Pan L, Zhao M, Chen B, Huang S, Yao Z, Liu J, Lv W. METS-IR/HOMA-IR and MAFLD in U.S. adults: dose-response correlation and the effect mediated by physical activity. BMC Endocr Disord. 2024 Aug 1;24(1):132. doi: 10.1186/s12902-024-01646-w. PMID: 39085855; PMCID: PMC11293050.
41. Zeng P, Cai X, Yu X, Huang L, Chen X. HOMA-IR is an effective biomarker of non-alcoholic fatty liver disease in the non-diabetic population. J Int Med Res. 2023 Oct;51(10):3000605231204462. doi: 10.1177/03000605231204462.
42. Bedogni G, Miglioli L, Masutti F, Tiribelli C, Marchesini G, Bellentani S. Prevalence of and risk factors for nonalcoholic fatty liver disease: the Dionysos nutrition and liver study. Hepatology. 2005 Jul;42(1):44-52. doi: 10.1002/hep.s 20734.
43. Teng ML, Ng CH, Huang DQ, Chan KE, Tan DJ, Lim WH, Yang JD, Tan E, Muthiah MD. Global incidence and prevalence of nonalcoholic fatty liver disease. Clin Mol Hepatol. 2023 Feb;29(Suppl):S32-S42. doi: 10.3350/cmh.2022.0365.
44. Genuth S, Alberti KG, Bennett P, Buse J, Defronzo R, Kahn R, et al. Follow-up report on the diagnosis of diabetes mellitus. Diabetes Care. 2003;26(11):3160-3167. doi:10.2337/diacare.26.11.3160.