Dietary Habits and Physical Activity in Metabolic Syndrome Patients with Hepatic Steatosis in Calabar, Nigeria

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ABSTRACT

Background: *The growing adoption of Western lifestyle in Nigeria has significantly increased the prevalence of non-communicable diseases such as Metabolic syndrome and its hepatic manifestation, such as hepatic steatosis (further subclassified as NAFLD and MAFLD).*

Objectives*: To investigate the dietary habits and physical activity of Met-S patients with hepatic steatosis in Calabar.*

Methodology: *This 12-month cross-sectional analytic study was conducted at the University of Calabar Teaching Hospital in Cross River State and included 118 adults who met diagnostic criteria for Met-S, NAFLD, and MAFLD. Clinical and laboratory data of Met-S patients with hepatic steatosis were obtained using a pre-tested interviewer-administered questionnaire. The SPSS version 25 analysis included Chi-square/Fisher's tests, as well as multivariate logistic regression, to identify independent predictors.*

Results*: NAFLD and MAFLD were highly prevalent in the Met-S patients, affecting 72.9% and 75.4% of participants, respectively. Patients with Met-S were mostly middle-aged females (72.2% and 76.3%, respectively). Bivariate analysis revealed that protein intake (X^2 = 15.864, p = 0.001) and hypertriglyceridemia (X^2 = 6.361, p = 0.012) in Met-S patients with NAFLD were significantly associated. Aerobic exercise showed a borderline association (X^2 = 8.804, p = 0.051). However, Binary logistic regression identified hypertriglyceridemia (95% CI: 1.03-4.09, p=0.042) and low dietary protein intake (95% CI: 2.55-23.90, p <0.01) as independent risk factors for NAFLD, with odds ratios of 2.0 and 7.8, respectively.*

Conclusion: *NAFLD and MAFLD are common in Met-S patients, with low protein intake identified as a significant risk factor. Adopting lifestyle modifications is critical for their management.*

Keywords: Diet, Exercise, Hepatic Steatosis, Metabolic syndrome, MAFLD

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INTRODUCTION

Diet is a major modifiable determinant of non-communicable diseases (NCDs), making the promotion of healthy dietary habits a critical component of disease prevention and public health improvement (1). Although no single die-

tary pattern has been universally identified as optimal for health and disease prevention, several well-established dietary models have been associated with reduced risk of NCDs and improved health outcomes (1). These include the Dietary Approaches to Stop Hypertension (DASH) diet, the Mediterranean diet, the Mediterranean–DASH Intervention for Neurodegenerative Delay (MIND) diet, and the Nordic diet (1). These dietary patterns are characterized by a high intake of fruits, vegetables, whole grains, legumes, nuts, and other nutrient-dense foods, and have been shown to confer protective effects against cardiovascular diseases, metabolic disorders, and other chronic conditions (1).

The adoption of the Western lifestyle in modern Nigerian society has primarily contributed to more sedentary living and unhealthy dietary patterns (2, 3). Improved stable access to a variety of nutritious foods is one of the benefits of urbanization (2). On the contrary, urbanization creates an environment for easy access to ultra-processed and unhealthy food choices due to the convenience of preparing these meals as well as their affordability (2). The consequence of this is an increased risk of developing NCDs such as obesity (including truncal obesity), type 2 diabetes mellitus (T2DM), dyslipidaemia, and hypertension (4,5). The clustering of these interrelated metabolic abnormalities leads to the classification of Metabolic syndrome (Met-S)(6). Met-S is a well-established precursor to both non-alcoholic fatty liver disease (NAFLD) and cardiovascular disease (CVD) (6, 7, 8). Furthermore, there is growing evidence to suggest that even mild hepatic steatosis increases the risk of CVD (such as stroke) and all-cause mortality, particularly when compounded by T2DM (9). Non-alcoholic fatty liver disease has been described as the hepatic manifestation of Met-S, posing a major public health threat due to complications arising from liver cirrhosis and hepatocellular carcinoma (8,10). Traditionally, the diagnosis of NAFLD is established by identifying hepatic steatosis, often incidentally on imaging, in the absence of significant alcohol consumption and other causes of liver disease such as viral hepatitis B and C (10,11). In contrast, the concept of metabolic dysfunction–associated fatty liver disease (MAFLD), while still requiring evidence of hepatic steatosis, adopts a positive diagnostic criterion that emphasizes the presence of metabolic dysfunction rather than the exclusion of other causes of liver disease (7,8).

Diet plays a significant role in the development, progression, and management of NAFLD and associated metabolic comorbidities, including MAFLD (12,13). In general, a high-calorie diet, particularly rich in trans/ saturated fat and cholesterol, and fructose-sweetened beverages, has been associated with increased visceral fat deposition and the stimulation of hepatic lipid accumulation (i.e. intrahepatic triglycerides) and progression into non-alcoholic steatohepatitis (12,13). While a low-calorie diet, increased soy protein and whey consumption, as well as supplementation of monounsaturated fatty acids, omega-3 fatty acids, and probiotics have been found to show preventive and therapeutic effects (12,13). In addition, choline, fibre, coffee, green tea, and avoidance of alcohol might be protective factors for NAFLD (12,13).

Excess hepatic fat accumulation triggered by consumption of saturated fats acts through impairments in lipid storage and/or increased lipolysis, while carbohydrate consumption, especially fructose, has been shown to promote liver fat accumulation through de novo lipogenesis (12). Unhealthy dietary patterns and poor diet quality are not only strongly associated with the development and progression of NAFLD but are also risk factors for obesity and Met-S (6, 12, 13). Interestingly, dietary modification, either alone or in combination with increased physical activity and behavioural interventions, represents an evidence-based strategy for achieving weight loss and improving NAFLD outcomes.

Excess energy intake relative to expenditure can lead to the accumulation of fat in visceral adipose tissue and the liver, resulting in hepatic steatosis (10). Although total caloric intake may not be significantly increased in patients with NAFLD, their dietary patterns are typically characterized by higher consumption of saturated fats and simple carbohydrates compared with healthy individuals (12). The development and progression of NAFLD are strongly linked to such unhealthy dietary patterns (13). Consequently, evidence-based, diet-induced weight loss has been shown to improve liver enzymes and reduce hepatic steatosis. In line with this, clinical practice guidelines recommend weight reduction through a hypocaloric diet, either alone or combined with increased physical activity, as a cornerstone in the management of NAFLD (10,11). Weight loss of at least 3–5% is required to improve steatosis, while greater weight loss (approximately 10%) may be necessary to improve necroinflammation and fibrosis. These benefits are mediated through improvements in insulin resistance, enhanced fat metabo-

lism, and increased mitochondrial function (10,14).

The landmark Rotterdam study demonstrated that engagement in physical activity, irrespective of intensity, is associated with a reduced risk of NAFLD (12). Similar studies have reported a beneficial association between physical activity and NAFLD; however, there remains limited consensus regarding the optimal duration and intensity of exercise required for maximal benefit (14,15).

There is a notable paucity of research within our population examining the dietary habits and physical activity of patients with metabolic syndrome (Met-S) who develop hepatic steatosis, including NAFLD and MAFLD. Specifically, the contribution of diet and lifestyle to the development and progression of these metabolically driven conditions remains insufficiently explored.

This pioneer study was therefore undertaken to assess dietary habits and physical activity (exercise habits) among Met-S patients with hepatic steatosis in Calabar. Given the critical role of diet and lifestyle in the aetiopathogenesis and management of Met-S and hepatic steatosis, there is an urgent need for locally generated data to provide preliminary insights into these factors. Such evidence will serve as a basis for future large-scale studies and the development of homegrown interventions within the Nigerian setting.

METHODS

Study Location

This study was conducted at the Gastroenterology, Cardiology, Diabetic, and Metabolic Clinics of the University of Calabar Teaching Hospital (UCTH), Calabar, Cross River State (CRS), Nigeria. UCTH is a 600-bed tertiary healthcare institution located in the capital city of Cross River State. The hospital serves as a major referral centre for patients from CRS and neighbouring states, including Akwa Ibom, Abia, and Benue, as well as parts of Cameroon.

Study Design and Population

A 12-month cross-sectional analytical study was conducted among adults aged 18 years and above who provided written informed consent. Participants were recruited from the Gastroenterology, Cardiology, Diabetic, and Metabolic Clinics of UCTH.

Data were collected using a pre-tested, semi-structured interviewer-administered questionnaire designed to obtain information on socio-demographic characteristics, clinical symptoms, medical history, medication use, alcohol consumption, previous abdominal surgery, and family history of diabetes mellitus, liver disease, and cardiovascular disease. Dietary habits (including the frequency with which they consumed fruits, vegetables, processed/fast foods, legumes/protein, and carbohydrates) were assessed by asking the respondents how often a week they consumed these foods. Additionally, physical activity was also assessed, including the frequency of participating in exercise, e.g., aerobics lasting at least 30 minutes per session.

Metabolic syndrome was defined according to the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) criteria as the presence of at least three of the following components: central obesity (waist circumference ≥102 cm in men or ≥88 cm in women), elevated triglycerides (≥1.69 mmol/L), reduced high-density lipoprotein (HDL) cholesterol (≤1.03 mmol/L in men or ≤1.29 mmol/L in women), elevated fasting blood glucose (≥5.5 mmol/L), and blood pressure ≥130/85 mmHg (6).

Hepatic steatosis was diagnosed by abdominal ultrasonography and defined by increased hepatic echogenicity relative to the renal cortex and spleen, with either diffuse or focal involvement (16).

The diagnosis of non-alcoholic fatty liver disease was based on the presence of hepatic steatosis after excluding significant alcohol consumption and other causes of chronic liver disease, including viral hepatitis. Clinical features such as right upper quadrant discomfort, hepatomegaly, dyslipidaemia, and elevated liver enzymes were considered supportive findings (10,11).

Metabolic dysfunction-associated fatty liver disease was diagnosed according to established criteria, requiring evidence of hepatic steatosis in addition to overweight/obesity (BMI ≥25 kg/m^2) or type 2 diabetes mellitus. In the absence of these conditions, the diagnosis required the presence of at least two metabolic risk abnormalities, including increased waist circumference, elevated triglycerides, reduced HDL cholesterol, hypertension, prediabetes, elevated high-sensitivity C-reactive protein, or a Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) score ≥2.5 (8, 17).

Screening for hepatitis B surface antigen (HBsAg) and hepatitis C virus antibody (anti-HCV) was performed using rapid diagnostic test kits to exclude alternative causes of liver disease.

Inclusion and Exclusion Criteria

Eligible participants were consenting adults aged 18 years and above with sonographic evidence of hepatic steatosis who fulfilled the diagnostic criteria for metabolic syndrome. Participants with jaundice, shrunken liver suggestive of advanced chronic liver disease, malignancy, pregnancy, severe systemic illnesses such as heart failure, advanced liver disease, thyroid disorders, or renal disease, as well as those receiving medications associated with secondary hepatic steatosis, were excluded from the study.

Sample Size Determination

The sample size was initially estimated using Cochran's formula for prevalence studies, assuming a prevalence of 12.3% (18), a 95% confidence level, and a margin of error of 5%, yielding a minimum sample size of 166 participants. After adjusting for a possible non-response rate, the sample size increased to 183 participants. However, because the target clinic population was finite, the Taro Yamane formula was applied using an estimated clinic population of 320 patients, resulting in a minimum sample size of 117 participants. A total of 118 eligible participants were eventually recruited using a purposive convenience sampling technique.

Data Collection

Data collection was conducted using a pre-tested, semi-structured interviewer-administered questionnaire. The questionnaire was pilot-tested among approximately 10% of the study population to ensure clarity, reliability, and appropriateness of the study instrument. Anthropometric measurements included weight, height, body mass index (BMI), and waist circumference. Blood pressure measurements were obtained using standard procedures. Laboratory investigations included fasting blood glucose, lipid profile, liver function tests, C-reactive protein, and screening for hepatitis B and hepatitis C infection. Abdominal ultrasonography was performed to assess the presence of hepatic steatosis.

Data Analysis

Data were entered and analyzed using the Statistical Package for Social Sciences (SPSS) version 25. Continuous variables were summarized using means and standard deviations, while categorical variables were presented as frequencies and percentages. Associations between categorical variables were assessed using Chi-square or Fisher's exact tests as appropriate. Variables that demonstrated significant associations at the bivariate level were entered into a multivariable logistic regression model to identify independent predictors of NAFLD. Results were reported as adjusted odds ratios (AORs) with corresponding 95% confidence intervals (CIs). Statistical significance was set at $p < 0.05$.

Ethical Considerations

Ethical approval for the study was obtained from the University of Calabar Teaching Hospital Health Research Ethics Committee (Protocol Number: UCTH/HREC/33/Vol.III/118). Written informed consent was obtained from all participants before enrolment. Three eligible individuals declined participation, while one participant died during the study period and was therefore excluded from the final analysis. The study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki.

RESULTS

Demographic and Clinical Characteristics of the Met-S Patients

Females constituted the majority (64.4%), while males accounted for 35.6%. Most patients were within the 45–65 years age group (76.3%), followed by those over 65 years (20.3%), and a small proportion (3.4%) aged 18–45 years.

Anthropometric Profile of the Met-S Patients

Table 2 showed that 30.5% of patients are overweight, and 27.1% are in the obese class I category. Waist circumference was elevated in more than half (52.5%) of the Met-S patients, indicating a high prevalence of central obesity.

Prevalence of NAFLD and MAFLD

Among the 118 patients with Met-S, NAFLD and MAFLD were highly prevalent, affecting 72.9% and 75.4% of participants, respectively, indicating a substantial overlap between the two conditions in our study participants.

Table 1: Socio-Demographic Characteristics of the Met-S Patients

Variable	Frequency (118)	Percentage (%)
Sex		
Male	42	35.6
Female	76	64.4
Age group(years)		
18-44	4	3.4
45-64	90	76.3
≥65	24	20.3
Mean age ± SD years	58.1 ± 9.7	
Senatorial district		
Southern	12	10.2
Central	10	8.5
Northern	8	6.8
Others (Ibo, Yoruba, Hausa)	88	74.6
Religion		
Christianity	115	97.5
Muslim	3	2.5
Marital status		
Single	7	5.9
Married	79	66.9
Divorced	5	4.2
Widowed	27	22.9
Occupation		
Civil service	25	21.2
Public servant	19	16.1
Business	32	27.1
Others-Artisans, farmers, etc	42	35.6
History of diabetes mellitus		
Present	100	84.7
Absent	18	15.3
History of hypertension		
Present	117	99.2
Absent	1	0.8
History of stroke		
Present	8	6.8
Absent	110	93.2
Takes alcohol		
Yes	42	35.6
No	76	64.4
Abdominal swelling		
Present	6	5.1
Absent	112	95
Pedal oedema		
Present	2	1.7
Absent	116	98.3
RUQ pain		
Present	30	25.4
Absent	88	74.6

TABLE 2: Anthropometric Measurements among Met-S Patients

Variable	Frequency (118)	Percentage (%)
BMI category (kg/m²)		
18.5 to 24.9 (Normal)	14	11.9
25.0 to 29.9 (overweight)	36	30.5
30.0 to 34.9 (Class 1 obesity)	32	27.1
35.0 to 39.9 (Class 2 obesity)	29	24.6
≥40.0 (Class 3 obesity)	7	5.9
Mean BMI ± SD	31.3 ± 6.0	
Waist circumference/cm		
Normal	56	47.5
Increased (≥ 88, F: ≥ 110)	62	52.5
Mean WC ± SD	99.6 ± 17.4	

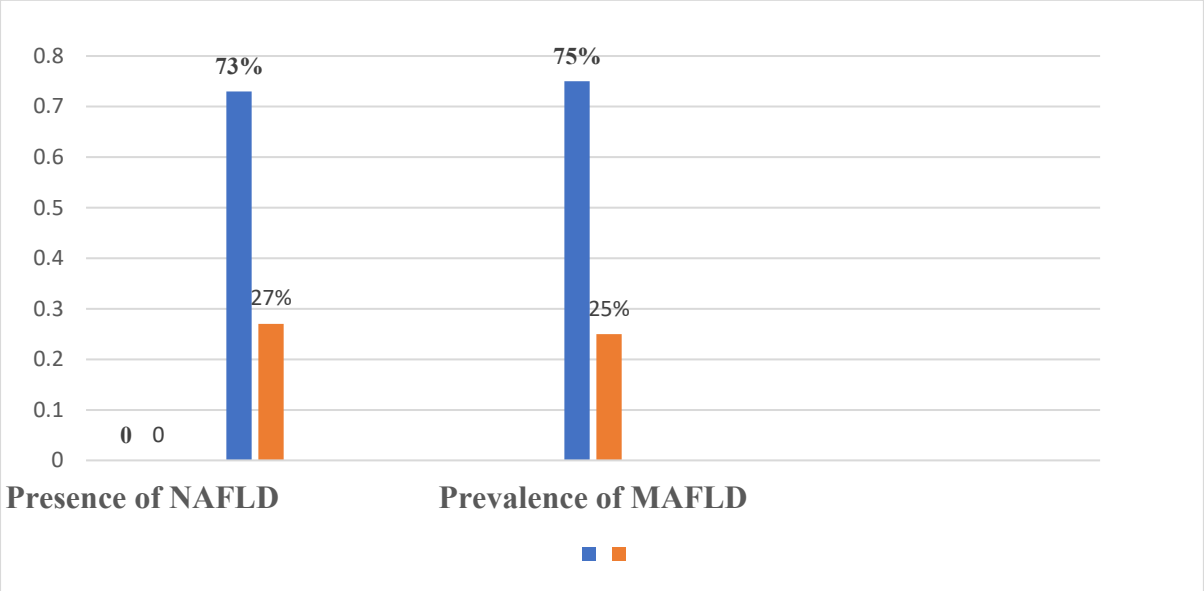

Figure 1: Prevalence of NAFLD and MAFLD in Met-S Patients

Association between Socio-Demographic Characteristics, Dietary Patterns and Exercise in Patients with Met-S and NAFLD

Table 3a and b showed that the majority of the 118 participants were female (64.4%), aged 45–65 years (76.3%), married (66.9%), and had tertiary education (65.3%). Most participants were engaged in business or other informal forms of employment, while only a small proportion reported a previous history of stroke (6.8%) or right upper quadrant abdominal pain (25.4%). Physical activity levels were generally low, with 84.7% reporting no aerobic exercise lasting more than 30 minutes. Dietary assessment revealed low consumption of whole grains and cereals, while over half of the participants reported daily intake of protein-rich foods and 41.5% consumed fruits and vegetables daily. The prevalence of NAFLD among participants was high, affecting 72.9% of the study population. In bivariate analysis, only protein intake showed a statistically significant association with NAFLD ($\chi^2 = 15.864$, $p = 0.001$), with lower protein consumption being more common among affected participants. No significant associations were found between NAFLD and sociodemographic characteristics, medical history, physical activity, or other dietary variables ($p > 0.05$).

TABLE 3a: The Association between Socio-Demographic Characteristics, Dietary Patterns, and Exercise in Met-S Patients with NAFLD

Variable	Non-alcoholic fatty liver disease Present n=86	Absent n=32	Total N=118	Chi-square test	p-value
Sex					
Male	28(66.7)	14(33.3)	42(100.0)	1.274	0.259
Female	58(76.3)	18(23.7)	76(100.0)		
Age group/years					
18-45	2(50.0)	2(50.0)	4(100.0)	1.696	0.466*
45-65	65(72.2)	25(27.8)	90(100.0)		
≥65	19(79.2)	5(20.8)	24(100.0)		
Marital status					
Single	5(71.4)	2(28.6)	7(100.0)	1.070	0.856*
Married	59(74.7)	20(25.3)	79(100.0)		
Divorced	3(60.0)	2(40.0)	5(100.0)		
Widowed	19(70.4)	8(29.6)	27(100.0)		
Highest education					
Primary	10(71.4)	4(28.6)	14(100.0)	0.532	0.949*
Secondary	15(71.4)	6(28.6)	21(100.0)		
Tertiary	57(74.0)	20(26.0)	77(100.0)		
Informal	4(66.7)	2(33.3)	6 (100.0)		
Occupation					
Civil service	17(68.0)	8(32.0)	25(100.0)	2.944	0.400*
Public servant	13(68.4)	6(31.6)	19(100.0)		
Business	27(84.4)	5(15.6)	32(100.0)		
Others	29(69.0	13(31.0)	42(100.0)		
History of stroke					
Present	7 (87.5)	1 (12.5)	8 (100.0)	0.445	0.308*
Absent	79 (71.8)	31 (28.2)	110 (100.0)		
RUQ pain					
Present	21 (70.0)	9 (30.0)	30 (100.0)	0.169	0.681
Absent	65 (73.9)	23 (26.1)	88 (100.0)		
Aerobic exercise > 30 minutes					
Everyday	1 (50.0)	1 (50.0)	2 (100.0)	8.804	0.051*
Thrice/week	3 (75.0)	1 (25.0)	4 (100.0)		
Twice/week	0 (0)	2 (100)	2 (100.0)		
Once/week	5 (50.0)	5 (50.0)	10 (100.0)		
None	77 (77.0)	23 (23.0)	100 (100.0)		
Whole grain intake (Oats, wheat swallow, wheat bread)					
Everyday	14 (77.8)	4 (22.2)	18 (100.0)	8.456	0.102*
5-6 times/week	2 (100)	0 (0)	2 (100.0)		
3-4 times/week	17 (94.4)	1 (5.6)	18 (100.0)		
1-2 times/week	11 (55.0)	9 (45.0)	20 (100.0)		
0 times/week	42 (70.0)	18 (30.0)	60 (100.0)		
Protein intake (beans, eggs, meat etc)					
Everyday	53 (81.5)	12 (18.5)	65 (100.0)	15.864	**0.001***
5-6 times/week	16 (80.0)	4 (20.0)	20 (100.0)		
3-4 times/week	9 (75.0)	3 (25.0)	12 (100.0)		
1-2 times/week	7 (35.0)	13 (65.0)	20 (100.0)		
0 times/week	1 (100)	0 (0)	1 (100.0)		

TABLE 3b: The Association between Socio-Demographic Characteristics, Dietary Patterns, and Exercise in Met-S Patients with NAFLD

Variable	Non-alcoholic fatty liver disease Present n=86	Absent n=32	Total N=118	Chi-square test	p-value
CHO intake, i.e., staple starches (yam, rice, garri etc)					
Everyday	22 (59.5)	15 (40.5)	37 (100)	5.525	0.220*
5-6 times/week	10 (90.9)	1 (9.1)	11 (100)		
3-4 times/week	22 (75.9)	7 (24.1)	29 (100)		
1-2 times/week	30 (76.9)	9 (23.1)	39 (100)		
0 times/week	2 (100)	0 (0)	2 (100)		
Cereals (pap, cornflakes, Golden morn etc)					
Everyday	8 (66.7)	4 (33.3)	12 (100)	0.714	0.907*
5-6 times/week	0 (0)	0 (0)	0 (0)		
3-4 times/week	9 (62.9)	4 (30.8)	13 (100)		
1-2 times/week	24 (72.7)	9 (27.3)	33 (100)		
0 times/week	45 (75.0)	15 (25.0)	60 (100)		
Fruits & Vegetables					
Everyday	34 (69.4)	15 (30.6)	49 (100)	0.959	0.966*
5-6 times/week	12 (75.0)	4 (25.0)	16 (100)		
3-4 times/week	20 (76.9)	6 (23.1)	26 (100)		
1-2 times/week	18 (72.0)	7 (28.0)	25 (100)		
0 times/week	2 (100)	0 (100)	2 (100)		

** - Fisher's Exact Test; **RUQ**- Right Upper Quadrant; **CHO**- Carbohydrates*

Association between Anthropometric Indices and Routine Laboratory Parameters among Met-S Patients with NAFLD

Table 4 presents the association between anthropometric, biochemical, and metabolic parameters and the presence of NAFLD among the study participants. Although the prevalence of NAFLD increased with increasing BMI, from 50.0% among participants with normal BMI to 87.5% among those with class I obesity, the association was not statistically significant ($\chi^2 = 8.068$, $p = 0.076$). Similarly, participants with increased waist circumference had a higher prevalence of NAFLD (77.4%) than those with normal waist circumference (67.9%), but this difference was not significant ($\chi^2 = 1.361$, $p = 0.243$).

Elevated alanine transaminase (ALT) levels were observed in nearly half of the participants, with NAFLD occurring slightly more frequently among those with abnormal ALT values (75.4%) compared with those with normal levels (70.5%); however, no significant association was demonstrated ($\chi^2 = 0.365$, $p = 0.546$). Likewise, HDL (high-density lipoprotein)-cholesterol, LDL (low-density lipoprotein)-cholesterol, and C-reactive protein (CRP) showed no statistically significant relationships with NAFLD ($p > 0.05$), although higher proportions of NAFLD were observed among participants with abnormal HDL-cholesterol and elevated CRP levels.

Significant associations were found between NAFLD and selected metabolic parameters. Total cholesterol was significantly associated with NAFLD ($\chi^2 = 4.163$, $p = 0.041$), while abnormal triglyceride levels showed a stronger association ($\chi^2 = 6.361$, $p = 0.012$). Elevated fasting blood sugar was also significantly related to NAFLD ($\chi^2 = 4.670$, $p = 0.031$), with a greater proportion of participants with hyperglycaemia having NAFLD compared with those with normal glucose levels. Overall, elevated total cholesterol, abnormal triglycerides, and increased fasting blood sugar emerged as the factors significantly associated with NAFLD.

Multivariate Binary Logistic Regression of the Predictors of NAFLD among Patients with Met-S

Table 5 presents the multivariable logistic regression analysis of factors associated with NAFLD. Variables that were significantly associated with NAFLD at the bivariate level were entered into the model to identify independent predictors. Following adjustment for potential confounders, abnormal serum triglyceride level remained a significant independent predictor of NAFLD. Participants with elevated triglyceride levels had approximately twice the odds of having NAFLD compared with those with normal triglyceride levels (AOR = 2.046, 95% CI: 1.025–4.086, $p = 0.042$).

TABLE 4: Association between Anthropometric Indices and Routine Laboratory Parameters among Met-S Patients with NAFLD

Variable	Non-alcoholic Fatty Liver Disease Present: n=86	Absent: n=32	Total: N=118	Chi-square test	p-value
BMI category (kg/m²)					
18.5 to 24.9	7(50.0)	7(50.0)	14(100.0)	8.068	0.076*
25.0 to 29.9	24(66.7)	12(33.3)	36(100.0)		
30.0 to 34.9	28(87.5)	4(12.5)	32(100.0)		
35.0 to 39.9	22(75.9)	7(24.1)	29(100.0)		
≥40.0	5(71.4)	2(28.6)	7(100.0)		
Waist circumference/cm					
Normal	38(67.9)	18(32.1)	56(100.0)	1.361	0.243
Increased	48(77.4)	14(22.6)	62(100.0)		
ALT					
Normal	43(70.5)	18(29.5)	61(100.0)	0.365	0.546
Abnormal	43(75.4)	14(24.6)	57(100.0)		
Total cholesterol					
Normal	45(81.8)	10(18.2)	55(100.0)	4.163	**0.041**
Elevated	41(65.1)	22(34.9)	63(100.0)		
HDL-cholesterol					
Normal	58(69.9)	25(30.1)	83(100.0)	1.276	0.259
Abnormal	28(80.0)	7(20.0)	35(100.0)		
LDL-cholesterol					
Normal	44(81.5)	10(18.5)	54(100.0)	3.726	0.054
Abnormal	42(65.6)	22(34.4)	64(100.0)		
Triglyceride					
Normal	57(81.4)	13(18.6)	70(100.0)	6.361	**0.012**
Abnormal	29(60.4)	19(39.6)	48(100.0)		
Fasting blood sugar					
Normal	18(58.1)	13(41.9)	31(100.0)	4.670	**0.031**
Elevated	68(78.2)	19(21.8)	87(100.0)		
C-reactive protein					
Normal	45(67.2)	22(32.8)	67(100.0)	2.564	0.109
Elevated	41(80.4)	10(19.6)	51(100.0)		

*= *Fisher's Exact Test*

Although total cholesterol and fasting blood sugar were significantly associated with NAFLD in the bivariate analysis, these relationships did not persist after adjustment. Elevated total cholesterol was not independently associated with NAFLD (AOR = 1.251, 95% CI: 0.942–1.660, p = 0.122), while fasting blood sugar showed a borderline association that did not reach statistical significance (AOR = 0.837, 95% CI: 0.700–1.002, p = 0.052).

Dietary protein intake also emerged as an important predictor of NAFLD. Compared with participants who consumed protein-rich foods daily, those who consumed such foods only one to two times per week had significantly increased odds of NAFLD (AOR = 7.810, 95% CI: 2.552–23.903, p = 0.001). However, protein consumption frequencies of three to four days per week and four to six days per week were not significantly associated with NAFLD (p > 0.05).

Overall, abnormal triglyceride levels and low protein intake (one to two times per week) were identified as the independent predictors of NAFLD in this study, underscoring the importance of lipid metabolism and dietary protein adequacy in the development of hepatic steatosis.

TABLE 5: Multivariate Binary Logistic Regression of Predictors of NAFLD among Patients with Met-S

	AOR	95% Confidence Interval		p-value
		Lower	Upper	
Total cholesterol	1.251	0.942	1.660	0.122
Triglyceride	2.046	1.025	4.086	0.042*
Fasting blood sugar	0.837	0.700	1.002	0.052
Protein intake				
Everyday	RC*			
4-6 days/week	1.181	0.328	4.246	0.799
3-4 days/week	1.513	0.348	6.574	0.580
1-2 days/week	7.810	2.552	23.903	0.001*

*=independent predictor of hepatic steatosis; Reference category

DISCUSSION

Anthropometric assessment in patients with Met-S and hepatic steatosis, including NAFLD and MAFLD, provides key insights into body composition and central adiposity, both critical in disease pathogenesis (19). Dietary patterns are also major determinants of metabolically driven diseases, with lifestyle interventions significantly impacting metabolic health and overall well-being (20). In contemporary Nigeria, rapid urbanization, improved food systems, increasing affluence, and sedentary lifestyles have promoted unhealthy dietary habits and overnutrition, leading to excess intrahepatic triglyceride accumulation and an increased risk of hepatic steatosis, particularly among individuals with Met-S. This pilot study was designed to examine the dietary habits and physical activity behaviours of Met-S patients with hepatic steatosis, who fulfilled the diagnostic criteria for NAFLD and MAFLD.

Overnutrition affects approximately 31% of the population, particularly in women; this is mainly influenced by factors such as parity, reduced post-pregnancy activity, age, education, wealth, employment, and geographic region (21). In this study, the prevalence of NAFLD increased with increasing BMI, which was considerably higher than the national estimate of 14.5% reported in a 2022 meta-analysis (22). This marked difference is not surprising, given that the present study was conducted among individuals with Met-S, a population already at risk of obesity and other cardiometabolic risk factors. Furthermore, the study was undertaken in southern Nigeria, where obesity rates have been reportedly higher than the national average due to rapid urbanization, dietary transitions, increasing affluence, and sedentary lifestyles (22). These findings underscore the close relationship between obesity and hepatic steatosis. As a key component of Met-S, obesity not only contributes to the development and progression of hepatic steatosis but also increases the risk of cardiovascular morbidity and mortality. Hence, effective weight management through dietary modification, increased physical activity, and other lifestyle interventions remains crucial in both liver and cardiovascular disease prevention and management.

Although a high prevalence of elevated anthropometric indices, particularly increased BMI and waist circumference (WC), was observed in this study, neither parameter showed a statistically significant association with NAFLD following bivariate analysis. This finding is consistent with the report by Chukwurah et al., who similarly found no significant relationship between these anthropometric measures and NAFLD, despite documenting slightly higher mean BMI and waist circumference values than those observed in our study (23). The lack of association may reflect the relatively homogeneous metabolic risk profile of the study population, all of whom had Met-S, thereby limiting the ability of these anthropometric indices to discriminate between participants with and without NAFLD. Waist circumference is increasingly recognized as a more reliable indicator of visceral obesity than the waist-to-hip ratio (24). Elevated WC, reflecting visceral adiposity, is associated with increased release of free fatty acids and pro-inflammatory mediators, which promote hepatic steatosis, inflammation, and fibrosis, creating a metabolically adverse environment (24). The relationship between WC, waist-to-hip ratio, and NAFLD is well established (24, 25), and the prevalence of NAFLD rises with visceral obesity and elevated BMI (25). These patterns were similarly observed in our cohort, reinforcing existing evidence.

Furthermore, morbidly obese individuals are reported to exhibit more pronounced insulin resistance, increasing their risk of Met-S and NAFLD (24). In this study, although a majority (78.2%) of patients had elevated fasting blood glucose, regression analysis did not demonstrate a statistical-

ly significant association with NAFLD. This finding suggests a potential limitation of relying solely on fasting glucose as a marker of metabolic dysfunction and underscores the superiority of the MAFLD framework, which incorporates measures such as the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) to more accurately identify at-risk individuals (7,8).

In our study, a small subset of patients with NAFLD (n = 7, 50%) had a BMI below 25 kg/m^2, although this was not statistically significant. This aligns with the concept of "lean NAFLD," which describes hepatic steatosis in individuals who are not overweight or obese according to region-specific BMI cut-offs (26). While reports on lean NAFLD are uncommon in Nigeria, where most reported cases occur in overweight or obese individuals, it highlights that hepatic steatosis can develop even in the absence of traditional anthropometric risk factors. Lean NAFLD has been associated with sarcopenia, insulin resistance, and visceral adiposity, which together promote metabolic dysfunction and ectopic fat accumulation (27). Although lean NAFLD was not a primary focus of our study, recognizing its existence underlines the need for comprehensive metabolic evaluation in all patients with Met-S, irrespective of BMI.

This study furthermore identified suboptimal lifestyle patterns in 77% of Met-S patients with NAFLD, with more than three-quarters of the cohort reporting inadequate physical activity (aerobic exercise lasting at least 30 minutes). Although this finding was not statistically significant, it reflects a sedentary lifestyle within the study population. Similar trends have been reported in other Nigerian studies, where low levels of physical activity and poor lifestyle practices are common among individuals with metabolic risk factors (28,29). Evidence from international studies, including the Rotterdam Study, demonstrates that physical activity, irrespective of intensity, is associated with a reduced risk of NAFLD (14). Furthermore, a modest weight reduction of approximately 5% has been shown to slow disease progression and may even result in fibrosis regression (14). Importantly, clinically meaningful improvements may also be achieved with weight loss below 5% or exceeding 10%. These findings emphasize that achieving a "normal" BMI (<25 kg/m^2) may not always be necessary, as improvements in metabolic health can occur with sustained weight reduction regardless of baseline body weight (30).

Multivariate regression analysis in this study showed that Met-S patients consuming only 1–2 servings of protein-rich foods per week (e.g., eggs, milk, beans, or meat) had a 7.18-fold higher risk of NAFLD, indicating a significant inverse relationship between protein intake and disease risk. Adequate dietary protein, alongside weight reduction, may reduce hepatic fat, whereas high protein intake typical of Western diets has been linked to increased NAFLD prevalence and severity (31). Dysregulation of plasma amino acids, including branched-chain and aromatic amino acids, may further drive intrahepatic fat accumulation (31). Additionally, our study showed a significant association (following regression analysis) between elevated serum triglycerides and NAFLD, suggesting the possible role of increased dietary fat or hepatic de novo lipogenesis.

Studies in our local context support the role of diet in the development of metabolic conditions. For instance, Olatona et al reported that urban Nigerian adults often consume energy-dense, nutrient-poor diets with inadequate intake of fruits and vegetables, and this contributed to the high prevalence of obesity among the study cohorts (28). Similarly, in Kenya, Okube et al found that individuals who consumed large amounts of carbohydrates, proteins, processed/fast foods, and sugar were predisposed to having Met-S (29). In our study, most patients reported infrequent consumption of whole grains and cereals, modest fruit and vegetable intake, and frequent consumption of carbohydrate-rich staples such as yams, garri, fufu, and rice. Evidence from international studies has consistently demonstrated that the Mediterranean diet, characterized by low saturated fat intake, high dietary fibre content, rich in antioxidants, and a favourable balance of monounsaturated and omega-3 fatty acids, is associated with a reduced risk of Met-S, T2DM, cardiovascular disease, and NAFLD (32). However, widespread adoption of this dietary pattern in Nigeria may be unattainable due to high cost, lack of availability, and differences in local food preferences. This highlights the need to develop and evaluate culturally acceptable, affordable, and locally sourced dietary models that can deliver similar metabolic and hepatic benefits. Given the limited evidence on dietary habits and physical activity among Nigerian patients with Met-S and hepatic steatosis, including NAFLD and MAFLD, further context-specific research is required to inform effective lifestyle interventions and public health strategies aimed at reducing the growing burden of metabolic and liver-related diseases.

CONCLUSION

This study demonstrated a slightly higher prevalence of MAFLD compared to NAFLD, reflecting the central role of metabolic dysfunction in liver disease and the need for heightened clinical suspicion in high-risk individuals. The high burden of obesity, increased waist circumference, and physical inactivity support weight reduction through caloric restriction and structured lifestyle modification as key management strategies. A culturally appropriate "Wazobia" or "Naija" diet (comparable with the Mediterranean diet) emphasizing whole grains, legumes, fruits, vegetables, and healthy fats alongside increased physical activity may improve metabolic health and hepatic outcomes in our environment.

A key limitation of this study was the inability to quantify nutrient (macro and micro) composition of local diets, underscoring the need for further home-grown nutritional research.

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DISCLOSURE AND CONFLICTS OF INTEREST

We hereby disclose no conflicts of interest that might influence the results or interpretation of this manuscript.

REFERENCES

1. Fischer, D., & Ouyang, P. (2025). Lifestyle-related factors for improving diet quality. *Nutrients, 17*(3), 448.
2. Ameye, H., Hülsen, V., Glatzel, K., Laar, A., & Qaim, M. (2025). Urbanizing food environments in Africa: Challenges and opportunities for improving accessibility, affordability, convenience, and desirability of healthy diets. *Food Policy, 137*.
3. Popkin, B. M., Corvalan, C., & Grummer-Strawn, L. M. (2020). Dynamics of the double burden of malnutrition and the changing nutrition reality. *The Lancet, 395*(10217), 65–74.
4. World Health Organization. (2023). *Non-communicable diseases*. Retrieved August 1, 2026, from https://www.who.int/news-room/fact-sheets/detail/noncommunicable-diseases
5. Saklayen, M. G. (2018). The global epidemic of the metabolic syndrome. *Current Hypertension Reports, 20*(2), 12.
6. Dobrowolski, P., Prejbisz, A., Kuryłowicz, A., Baska, A., Burchardt, P., Chlebus, K., Dzida, G., Jankowski, P., Jaroszewicz, J., Jaworski, P., Kamiński, K., Kapłon-Cieślicka, A., Klocek, M., Kukla, M., Mamcarz, A., Mastalerz-Migas, A., Narkiewicz, K., Ostrowska, L., Śliż, D., . . . Bogdański, P. (2022). Metabolic syndrome: A new definition and management guidelines: A joint position paper by the Polish Society of Hypertension, Polish Society for the Treatment of Obesity, Polish Lipid Association, Polish Association for Study of Liver, Polish Society of Family Medicine, Polish Society of Lifestyle Medicine, Division of Prevention and Epidemiology Polish Cardiac Society, "Club 30" Polish Cardiac Society, and Division of Metabolic and Bariatric Surgery Society of Polish Surgeons. *Archives of Medical Science, 18*(5), 1133–1156.
7. Eslam, M., Newsome, P. N., Sarin, S. K., Anstee, Q. M., Targher, G., Romero-Gomez, M., Zelber-Sagi, S., Wong, V. W. S., Dufour, J. F., Schattenberg, J. M., Kawaguchi, T., Arrese, M., Valenti, L., Shiha, G., Tiribelli, C., Yki-Järvinen, H., Fan, J. G., Grønbæk, H., Yilmaz, Y., . . . George, J. (2020). A new definition for metabolic dysfunction-associated fatty liver disease: An international expert consensus statement. *Journal of Hepatology, 73*(1), 202–209.
8. Eslam, M., Sanyal, A. J., George, J., & International Consensus Panel. (2020). MAFLD: A consensus-driven proposed nomenclature for metabolic-associated fatty liver disease. *Gastroenterology, 158*(7), 1999–2014.
9. Kim, K. S., Hong, S., Han, K., & Park, C. Y. (2024). 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, 384*, e076388.
10. Powell, E. E., Wong, V. W., & Rinella, M. (2021). Non-alcoholic fatty liver disease. *The Lancet, 397*(10290), 2212–2224.
11. Chalasani, N., Younossi, Z., Lavine, J. E., Charlton, M., Cusi, K., Rinella, M., Harrison, S. A., Brunt, E. M., & Sanyal, A. J. (2018). The diagnosis and management of non-alcoholic fatty liver disease: Practice guidance from the American Association for the Study of Liver Diseases. *Hepatology, 67*(1), 328–357.
12. Hydes, T., Alam, U., & Cuthbertson, D. J. (2021). The impact of macronutrient intake on non-alcoholic fatty liver disease (NAFLD): Too much fat, too much carbohydrate, or just too many calories? *Frontiers in Nutrition, 8*, 640557.

13. Romero-Gómez, M., Zelber-Sagi, S., & Trenell, M. (2017). Treatment of NAFLD with diet, physical activity and exercise. *Journal of Hepatology, 67*(4), 829–846.
14. Van Kleef, L. A., Hofman, A., Voortman, T., & de Knegt, R. J. (2022). Objectively measured physical activity is inversely associated with non-alcoholic fatty liver disease: The Rotterdam Study. *The American Journal of Gastroenterology, 117*(2), 311–318.
15. Golabi, P., Locklear, C. T., Austin, P., Afdhal, S., Byrns, M., Gerber, L., & Younossi, Z. M. (2016). Effectiveness of exercise in hepatic fat mobilization in non-alcoholic fatty liver disease: Systematic review. *World Journal of Gastroenterology, 22*(27), 6318–6327.
16. Kolawole, O. J., Oje, M., Betiku, O. A., Ijarotimi, O., Adekanle, O., & Ndububa, D. A. (2024). 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 Gastroenterology, 24*(1), 147.
17. Ng, C. H., Huang, D. Q., & Nguyen, M. H. (2022). Non-alcoholic fatty liver disease versus metabolic-associated fatty liver disease: Prevalence, outcomes and implications of a change in name. *Clinical and Molecular Hepatology, 28*(4), 790–801.
18. Obasi, E., Adekanle, E., Ajayi, N. A., & Ndububa, D. A. (2022). Clinical correlates of non-alcoholic steatohepatitis in Nigerian patients with metabolic syndrome. *West African Journal of Medicine, 39*(4), 407–414.
19. Silveira Almeida, N. S., Rocha, R., Cotrim, H. P., & Daltro, C. (2018). Anthropometric indicators of visceral adiposity as predictors of non-alcoholic fatty liver disease: A review. *World Journal of Hepatology, 10*(10), 695–701.
20. Chen, M., Chen, Y., Lin, J., Hu, R., Liu, D., Chen, J., Li, K., & Jiang, X. (2025). Evidence summary of lifestyle interventions in adults with metabolic dysfunction-associated steatotic liver disease. *Frontiers in Nutrition, 11*, 1421386.
21. Badru, O., & Alabi, T. A. (2023). There is a high prevalence of overnutrition among married and cohabiting women in Nigeria: Findings from the 2018 Nigeria Demographic and Health Survey. *Qeios*. https://doi.org/10.32388/ONGOQQ
22. Chukwuonye, I. I., Ohagwu, K. A., Ogah, O. S., John, C., Oviasu, E., Anyabolu, E. N., Ezeani, I. U., Iloh, G. U. P., Chukwuonye, M. E., Raphael, C. O., Onwuchekwa, U., Okafor, H. U., Oladele, C., Obi, E. C., Okwuonu, C. G., Iheji, O., Nwabuko, O. C., Nnoli, M. A., & Okpechi, I. G. (2022). Prevalence of overweight and obesity in Nigeria: Systematic review and meta-analysis of population-based studies. *PLOS Global Public Health, 10*(6).
23. Chukwurah, N. S., Okonkwo, U. C., & Ihekwaba, A. E. (2019). Comparative analysis of indices of the metabolic syndrome in patients with and without non-alcoholic fatty liver disease at a teaching hospital in Nnewi, South-East, Nigeria. *Asian Journal of Medical Sciences, 10*(2), 40–45.
24. Onyia, C. P., Asogwa, P., Adiri, W., Obienu, O., Ijoma, U. N., Nwokediuko, S. C., & Okeke, E. N. (2024). Non-alcoholic fatty liver disease and associated risk factors in obese Nigerians: A cross-sectional study. *Nigerian Journal of Clinical Practice, 27*(3), 352–360.
25. Suri, S., Singh, S., Rapelly, S. S., Rungta, S., & Ahmad, E. (2025). Waist circumference as a predictor for severity of liver fibrosis in non-alcoholic fatty liver disease patients. *Journal of Family Medicine and Primary Care, 14*(3), 1073–1078.
26. Kumar, R., & Mohan, S. (2017). Non-alcoholic fatty liver disease in lean subjects: Characteristics and implications. *Journal of Clinical and Translational Hepatology, 28*(3), 216–223.
27. Chen, M., Cao, Y., Ji, G., & Zhang, L. (2023). Lean non-alcoholic fatty liver disease and sarcopenia. *Frontiers in Endocrinology, 14*, 1217249.
28. Olatona, F. A., Adeniyi, D. B., Obrutu, O. E., & Ogunyemi, A. O. (2023). Nutritional knowledge, dietary habits and nutritional status of adults living in urban communities in Lagos State. *African Health Sciences, 23*(1), 711–724.
29. Okube, O. T., Kimani, S., & Waithira, M. (2020). Association of dietary patterns and practices on metabolic syndrome in adults with central obesity attending a mission hospital in Kenya: A cross-sectional study. *BMJ Open, 10*, e039131.
30. Ryan, D. H., & Yockey, S. R. (2017). Weight loss and improvement in comorbidity: Differences at 5%, 10%, 15%, and over. *Current Obesity Reports, 6*(2), 187–194.
31. Tricò, D., Biancalana, E., & Solini, A. (2021). Protein and amino acids in non-alcoholic fatty liver disease. *Current Opinion in Clinical Nutrition and Metabolic Care, 24*(1), 96–101.

32. Anania, C., Perla, F. M., Olivero, F., Pacifico, L., & Chiesa, C. (2018). Mediterranean diet and non-alcoholic fatty liver disease. *World Journal of Gastroenterology, 24*(19), 2083–2094.