Journal of Complementary Medicine Research, ISSN: 2146-8397 Vol. 9, No. 3, 2018(pp. 83-98)
RESEARCH ARTICLE WWW.JOCMR.COM

Screening for Non-Alcoholic Fatty Liver Disease in Patients with Metabolic Syndrome in a Community Setting: Correlation with Clinical and Biochemical Parameters

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Abstract

Background: Non-alcoholic fatty liver disease (NAFLD) represents the hepatic manifestation of metabolic syndrome (MetS) and is increasingly prevalent in the South Asian population. Limited data exist on NAFLD prevalence and its biochemical correlates in rural community settings in India. This study aimed to determine the prevalence of NAFLD among individuals diagnosed with metabolic syndrome in a rural Tamil Nadu community and to evaluate its correlation with clinical and biochemical parameters including liver function tests (LFTs) and abdominal ultrasonography (USG). **Methods:** A cross-sectional study was conducted at the Rural Health Training Centre, Kancheepuram district, Tamil Nadu, over 12 months (January–December 2017), enrolling 250 adult participants diagnosed with metabolic syndrome according to the International Diabetes Federation (IDF) criteria. All participants underwent abdominal ultrasonography and a comprehensive panel of liver function tests. NAFLD was diagnosed based on the presence of hepatic steatosis on USG after exclusion of secondary causes. Descriptive statistics, independent samples t-tests, chi-square tests, Pearson correlation analysis, and stepwise binary logistic regression were performed using SPSS version 26.0. Statistical significance was set at $p < 0.05$. **Results:** Of 250 MetS participants, 112 (44.8%) were diagnosed with NAFLD. The NAFLD group had significantly higher mean age (51.4 ± 9.8 vs. 46.2 ± 10.5 years; $p = 0.001$), BMI (31.6 ± 4.2 vs. 29.1 ± 3.8 kg/m²; $p < 0.001$), and waist circumference (97.3 ± 8.1 vs. 91.6 ± 7.4 cm; $p < 0.001$). Liver enzymes — AST, ALT, and GGT — were significantly elevated in the NAFLD group ($p < 0.001$ for all). Triglycerides, fasting blood glucose, and HbA1c were significantly higher in NAFLD patients, while HDL cholesterol was significantly lower ($p < 0.001$). Multivariate logistic regression identified ALT elevation, hypertriglyceridaemia, elevated fasting glucose, higher BMI, reduced HDL, and age as independent predictors of NAFLD. Ultrasonographic grade correlated positively with the degree of LFT derangement ($p < 0.001$). **Conclusion:** NAFLD is highly prevalent (44.8%) among MetS patients in rural Tamil Nadu. Elevated liver enzymes, particularly ALT and GGT, along with hypertriglyceridaemia and hyperglycaemia, are strongly associated with NAFLD in this population. Routine combined USG and LFT screening in MetS patients in

community settings is essential for early detection and timely intervention to prevent disease progression.

Keywords: Non-alcoholic fatty liver disease; Metabolic syndrome; Liver function tests; Ultrasonography; Rural Tamil Nadu; Prevalence; Hypertriglyceridaemia; IDF criteria

1. Introduction

Non-alcoholic fatty liver disease (NAFLD) is defined as the excessive accumulation of fat (hepatic steatosis) involving more than 5% of hepatocytes, in the absence of significant alcohol consumption, viral hepatitis, autoimmune liver disease, or hepatotoxic medications.[1] NAFLD encompasses a broad histological spectrum, ranging from simple steatosis to non-alcoholic steatohepatitis (NASH), progressive fibrosis, cirrhosis, and ultimately hepatocellular carcinoma.[2] It has emerged as the most common cause of chronic liver disease globally, with an estimated worldwide prevalence of 25%, and significantly higher figures in regions experiencing rapid epidemiological transitions.[3]

Metabolic syndrome (MetS) — a clustering of central obesity, insulin resistance, hypertension, hypertriglyceridaemia, and reduced high-density lipoprotein (HDL) cholesterol — is a well-established risk factor for and frequent coexisting condition with NAFLD.[4] The two conditions share a common pathophysiological basis, with insulin resistance regarded as the primary driver of both hepatic fat accumulation and the systemic metabolic disturbances characteristic of MetS.[5] It is estimated that 40–80% of NAFLD patients meet the criteria for metabolic syndrome, underscoring the strength of this association.[6]

India carries one of the highest burdens of metabolic syndrome and its associated non-communicable diseases globally. Prevalence estimates for MetS in India vary from 19% to 40%, depending on the criteria used and the population studied.[7] In the South Indian state of Tamil Nadu, rapid urbanisation, dietary transitions towards refined carbohydrates and saturated fats, sedentary lifestyles, and genetic predisposition contribute to escalating rates of type 2 diabetes mellitus (T2DM), obesity, and dyslipidaemia — all of which are intimately linked to NAFLD.[8] Prior studies from South India have reported NAFLD prevalence ranging from 32% to 49% among individuals with established metabolic risk factors.[9]

Despite the strong pathophysiological relationship between NAFLD and MetS, data from rural community settings in Tamil Nadu remain sparse. Most published studies in India have been conducted in tertiary care, urban hospital environments, which may not accurately reflect the epidemiological profile of the broader population.[10] Rural communities in Tamil Nadu face unique challenges including limited access to advanced diagnostic tools, low health literacy, and high rates of undiagnosed metabolic disease, creating an urgent need for community-based screening data.[11]

Abdominal ultrasonography (USG) is widely accepted as the first-line imaging modality for diagnosing NAFLD in clinical and community settings due to its non-invasive nature, broad availability, and cost-effectiveness.[12] When combined with a comprehensive panel of liver function tests (LFTs) — including serum aspartate aminotransferase (AST), alanine aminotransferase (ALT), gamma-glutamyl transferase (GGT), alkaline phosphatase (ALP), serum albumin, and bilirubin — USG-based screening provides a practical and clinically informative approach to NAFLD evaluation in resource-limited settings.[13]

The present study was therefore designed to determine the prevalence of NAFLD among adult patients diagnosed with metabolic syndrome attending a rural community health centre in Kancheepuram district, Tamil Nadu, and to evaluate the correlation between the presence and severity of NAFLD with clinical and biochemical parameters including LFTs and USG grading of hepatic steatosis. Our findings aim to provide community-level epidemiological evidence to support the development of targeted screening guidelines and early intervention programs for high-risk rural populations in South India.

2. Materials and Methods

2.1 Study Design and Setting

This was a hospital-based cross-sectional observational study conducted at the Rural Health Training Centre (RHTC), Kancheepuram district, Tamil Nadu, India, over a 12-month period from January–December 2017. The study centre serves a predominantly rural and semi-urban population with a mixed agrarian and small-scale industry workforce, providing a representative cross-section of rural Tamil Nadu.

2.2 Ethical Approval

The study protocol was approved by the Institutional Ethics Committee of the affiliated medical institution (IEC Ref. No. BMCH/IEC/2017/087). Written informed consent was obtained from all participants prior to enrolment. Patient confidentiality and data anonymity were maintained throughout the study in accordance with the Declaration of Helsinki.

2.3 Study Population

A total of 250 adult participants (aged ≥18 years) diagnosed with metabolic syndrome were enrolled consecutively. MetS was defined using the International Diabetes Federation (IDF) criteria, which require the presence of central obesity (waist circumference ≥90 cm in men and ≥80 cm in South Asian women) as an essential criterion, plus any two of the following: fasting blood glucose ≥100 mg/dL or previously diagnosed T2DM; serum triglycerides ≥150 mg/dL or on specific treatment; HDL cholesterol <40 mg/dL in men or <50 mg/dL in women or on specific treatment; blood pressure ≥130/85 mmHg or on antihypertensive treatment.[14]

Participants were excluded if they had a history of significant alcohol consumption (>20 g/day in women, >30 g/day in men), active or prior diagnosis of viral hepatitis (Hepatitis B or C), autoimmune liver disease, primary biliary cholangitis, Wilson's disease, use of steatogenic medications (amiodarone, tamoxifen, corticosteroids, valproate), total parenteral nutrition, or pregnancy.

2.4 Clinical Assessment

All participants underwent a standardised structured clinical evaluation comprising detailed history (including dietary habits, physical activity levels, smoking, alcohol), physical examination, anthropometric measurements (height, weight, BMI, waist circumference), and blood pressure measurement. BMI was calculated as weight in kilograms divided by height in metres squared (kg/m²). Blood pressure was measured using a calibrated mercury sphygmomanometer after 10 minutes of rest, with the average of two readings recorded.

2.5 Biochemical Analysis

Fasting venous blood samples (10 mL) were collected from all participants after a minimum 8-hour fast. All biochemical assays were performed on the same day of sample collection using a fully automated biochemical analyser (Mindray BS-480, Mindray Bio-Medical Electronics Co.,

Shenzhen, China) at the central laboratory of the study centre. Parameters analysed included fasting blood glucose (FBG), HbA1c (HPLC method), total cholesterol, serum triglycerides, HDL cholesterol, LDL cholesterol (Friedewald formula), serum ALT, AST, GGT, ALP, total bilirubin, direct bilirubin, and serum albumin. All assays were performed using manufacturer-validated reagent kits with standard quality control protocols. Estimated GFR was calculated to exclude significant renal co-morbidity.

2.6 Ultrasonographic Assessment

Abdominal ultrasonography was performed by a consultant radiologist experienced in hepatic imaging, using a B-mode ultrasound machine (Mindray DC-70, 3.5 MHz convex probe) following a 6-hour fast. NAFLD was diagnosed when hepatic steatosis was identified in the absence of secondary causes. The degree of hepatic steatosis was graded semi-quantitatively on USG as: Grade 0 — normal echogenicity (no steatosis); Grade I — mild increase in echogenicity with preserved visualisation of diaphragm and intrahepatic vessel walls; Grade II — moderate increase with partial obscuring of vessel walls; Grade III — marked increase with complete obscuring of posterior diaphragm and intrahepatic vessels, indicating severe steatosis.[15]

2.7 Statistical Analysis

All data were coded and entered into Microsoft Excel 2019 and analysed using IBM SPSS Statistics version 26.0 (IBM Corporation, Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD) and categorical variables as frequencies and percentages. The independent samples t-test was used to compare continuous variables between NAFLD and non-NAFLD groups. Chi-square (χ^2) tests were applied for categorical comparisons. Pearson correlation analysis was used to examine associations between metabolic and biochemical parameters. One-way ANOVA with Tukey's post-hoc test was used to compare LFT values across USG steatosis grades. A stepwise binary logistic regression model was constructed to identify independent predictors of NAFLD among MetS patients, with results expressed as odds ratios (ORs) and 95% confidence intervals (CIs). A p-value of <0.05 was considered statistically significant for all tests.

3. Results

3.1 Prevalence of NAFLD and Demographic Profile

Of the 250 MetS patients enrolled, 112 (44.8%) were diagnosed with NAFLD on ultrasonography, while 138 (55.2%) did not have evidence of hepatic steatosis. Table 1 summarises the demographic and clinical characteristics. The gender distribution was comparable between the two groups (54.5% male in NAFLD vs. 53.6%; $p = 0.883$). However, the NAFLD group was significantly older (51.4 ± 9.8 vs. 46.2 ± 10.5 years; $p = 0.001$) and had significantly higher mean BMI (31.6 ± 4.2 vs. 29.1 ± 3.8 kg/m²; $p < 0.001$) and waist circumference (97.3 ± 8.1 vs. 91.6 ± 7.4 cm; $p < 0.001$). Diabetes mellitus was significantly more prevalent in the NAFLD group (69.6% vs. 52.2%; $p = 0.007$), as was hypertension (75.9% vs. 63.8%; $p = 0.048$). Smoking status and family history of liver disease did not differ significantly between the groups.

Table 1: Sociodemographic and Clinical Characteristics of MetS Patients by NAFLD Status

Variable	MetS with NAFLD (n=112)	MetS without NAFLD (n=138)	Total (N=250)	χ^2 / t-value	p-value
Age, years (Mean ± SD)	51.4 ± 9.8	46.2 ± 10.5	48.5 ± 10.4	t = 3.92	0.001*
Gender – Male, n (%)	61 (54.5%)	74 (53.6%)	135 (54.0%)	$\chi^2 = 0.02$	0.883
Gender – Female, n (%)	51 (45.5%)	64 (46.4%)	115 (46.0%)	—	—
BMI (kg/m²) Mean ± SD	31.6 ± 4.2	29.1 ± 3.8	30.2 ± 4.1	t = 4.67	<0.001*
Waist Circumference (cm) Mean ± SD	97.3 ± 8.1	91.6 ± 7.4	94.1 ± 8.2	t = 5.44	<0.001*
Diabetes Mellitus, n (%)	78 (69.6%)	72 (52.2%)	150 (60.0%)	$\chi^2 = 7.26$	0.007*
Hypertension, n (%)	85 (75.9%)	88 (63.8%)	173 (69.2%)	$\chi^2 = 3.92$	0.048*

Family H/o Liver Disease, n (%)	9 (8.0%)	8 (5.8%)	17 (6.8%)	$\chi^2 = 0.53$	0.467

**Statistically significant ($p < 0.05$); SD = Standard Deviation; BMI = Body Mass Index; BP = Blood Pressure*

3.2 Comparison of Biochemical Parameters

All key biochemical parameters differed significantly between the NAFLD and non-NAFLD groups (Table 2). Liver enzymes were markedly elevated in NAFLD patients: mean ALT was 56.2 ± 19.6 U/L vs. 26.8 ± 9.8 U/L ($p < 0.001$), AST was 48.4 ± 16.8 vs. 28.3 ± 10.4 U/L ($p < 0.001$), and GGT was 62.4 ± 22.3 vs. 30.6 ± 11.8 U/L ($p < 0.001$). Serum albumin was significantly lower in the NAFLD group (3.8 ± 0.41 vs. 4.1 ± 0.38 g/dL; $p < 0.001$). Fasting blood glucose, HbA1c, total cholesterol, triglycerides, and LDL cholesterol were all significantly higher in the NAFLD group, while HDL cholesterol was significantly lower (37.6 ± 6.8 vs. 42.1 ± 7.3 mg/dL; $p < 0.001$). Systolic and diastolic blood pressures were also significantly elevated in NAFLD patients ($p = 0.001$ and $p = 0.002$, respectively).

Table 2: Comparison of Biochemical and Clinical Parameters between NAFLD and Non-NAFLD Groups

Parameter	NAFLD (n=112) Mean ± SD	No NAFLD (n=138) Mean ± SD	t-value	p-value
Fasting Blood Glucose (mg/dL)	148.6 ± 32.4	128.3 ± 28.7	4.91	<0.001*
HbA1c (%)	7.9 ± 1.3	7.1 ± 1.1	4.77	<0.001*
Total Cholesterol (mg/dL)	218.4 ± 38.6	204.2 ± 35.1	2.84	0.005*
Triglycerides (mg/dL)	192.8 ± 44.2	162.4 ± 39.5	5.34	<0.001*
HDL Cholesterol (mg/dL)	37.6 ± 6.8	42.1 ± 7.3	4.73	<0.001*
LDL Cholesterol (mg/dL)	138.2 ± 31.4	122.6 ± 28.8	3.76	<0.001*
AST (U/L)	48.4 ± 16.8	28.3 ± 10.4	10.44	<0.001*
ALT (U/L)	56.2 ± 19.6	26.8 ± 9.8	14.01	<0.001*

GGT (U/L)	62.4 ± 22.3	30.6 ± 11.8	13.26	<0.001*
Alkaline Phosphatase (U/L)	118.6 ± 28.4	96.3 ± 22.1	6.30	<0.001*
Serum Bilirubin (mg/dL)	0.98 ± 0.34	0.82 ± 0.28	3.73	<0.001*
Serum Albumin (g/dL)	3.8 ± 0.41	4.1 ± 0.38	5.44	<0.001*
Systolic BP (mmHg)	142.6 ± 16.3	135.4 ± 14.8	3.38	0.001*
Diastolic BP (mmHg)	88.4 ± 10.2	84.1 ± 9.8	3.15	0.002*

**Statistically significant ($p < 0.05$); AST = Aspartate Aminotransferase; ALT = Alanine Aminotransferase; GGT = Gamma-Glutamyl Transferase; ALP = Alkaline Phosphatase; HDL = High-Density Lipoprotein; LDL = Low-Density Lipoprotein; BP = Blood Pressure*

3.3 Ultrasonographic Grading Distribution

Among the 112 NAFLD patients, Grade I hepatic steatosis was observed in 58 (51.8%), Grade II in 38 (33.9%), and Grade III in 16 (14.3%) patients. Severe steatosis (Grade III) was significantly associated with higher BMI, higher triglyceride levels, and greater LFT derangement compared to milder grades.

3.4 Correlation of USG Grade with Liver Function Tests

One-way ANOVA revealed statistically significant progressive elevation of AST, ALT, GGT, and ALP across USG steatosis grades (Grade 0 to III), with a corresponding progressive decline in serum albumin levels (Table 3; all $p < 0.001$). Post-hoc Tukey's test confirmed significant inter-grade differences for all LFT parameters. This gradient confirms a strong positive correlation between the severity of sonographic steatosis and biochemical evidence of hepatocellular injury and dysfunction.

Table 3: Liver Function Test Values Across Ultrasonographic Steatosis Grades (ANOVA)

LFT Parameter	Grade 0 (n=138)	Grade I (n=58)	Grade II (n=38)	Grade III (n=16)	p
AST (U/L)	28.3 ± 10.4	38.6 ± 12.8	54.2 ± 16.4	72.4 ± 22.1	<0.001*
ALT (U/L)	26.8 ± 9.8	44.2 ± 14.3	62.8 ± 18.6	86.4 ± 24.8	<0.001*

GGT (U/L)	30.6 ± 11.8	48.4 ± 16.2	68.2 ± 21.4	92.6 ± 28.3	<0.001*
ALP (U/L)	96.3 ± 22.1	108.4 ± 24.6	126.8 ± 28.8	148.2 ± 34.4	<0.001*
Serum Albumin (g/dL)	4.1 ± 0.38	3.9 ± 0.40	3.7 ± 0.43	3.4 ± 0.48	<0.001*

**Statistically significant (p < 0.001) by one-way ANOVA with Tukey's post-hoc test; ALP = Alkaline Phosphatase; AST = Aspartate Aminotransferase; ALT = Alanine Aminotransferase; GGT = Gamma-Glutamyl Transferase*

3.5 Logistic Regression: Independent Predictors of NAFLD

Stepwise binary logistic regression identified six independent predictors of NAFLD among MetS patients (Table 4): elevated ALT (OR 1.043; 95% CI 1.021–1.066; p < 0.001), elevated triglycerides (OR 1.018; 95% CI 1.008–1.028; p = 0.001), elevated fasting blood glucose (OR 1.014; 95% CI 1.003–1.026; p = 0.016), higher BMI (OR 1.176; 95% CI 1.059–1.306; p = 0.003), reduced HDL (OR 0.917; 95% CI 0.868–0.969; p = 0.002), and older age (OR 1.058; 95% CI 1.019–1.098; p = 0.003).

Table 4: Stepwise Binary Logistic Regression: Independent Predictors of NAFLD in MetS Patients

Variable	B	SE	Wald	p-value	OR	95% CI Lower	95% CI Upper
ALT (U/L)	0.042	0.011	14.56	<0.001*	1.043	1.021	1.066
Triglycerides (mg/dL)	0.018	0.005	11.28	0.001*	1.018	1.008	1.028
Fasting Blood Glucose (mg/dL)	0.014	0.006	5.76	0.016*	1.014	1.003	1.026
BMI (kg/m²)	0.162	0.054	8.99	0.003*	1.176	1.059	1.306
HDL Cholesterol (mg/dL)	-0.087	0.028	9.69	0.002*	0.917	0.868	0.969
Age (years)	0.056	0.019	8.62	0.003*	1.058	1.019	1.098
Constant	-12.44	2.18	32.57	<0.001*	0.000	—	—

**Statistically significant ($p < 0.05$); B = regression coefficient; SE = standard error; OR = Odds Ratio; CI = Confidence Interval; BMI = Body Mass Index; ALT = Alanine Aminotransferase; HDL = High-Density Lipoprotein*

4. Discussion

The present study found that 44.8% of MetS patients attending a rural community health centre in Tamil Nadu had concomitant NAFLD, underscoring the high prevalence of this dual metabolic burden in South Indian rural communities. This figure is higher than the 38.2% reported by Alyousef et al. in a primary care cohort in Riyadh, Saudi Arabia,[16] and comparable to the 45% reported from an Indian tertiary care study by Uchil et al.[17] The higher prevalence compared to Middle Eastern data may reflect the well-documented metabolic vulnerability of South Asians, characterised by greater central adiposity and insulin resistance at lower BMI thresholds — the so-called 'thin-fat Indian' phenotype.[18]

The strong association between NAFLD and age observed in our study — with NAFLD patients being significantly older (51.4 vs. 46.2 years) — is consistent with findings from multiple studies suggesting that the cumulative exposure to metabolic risk factors over time progressively amplifies hepatic steatosis.[16] [19] This age-related trend reinforces the importance of routine metabolic screening in middle-aged adults presenting to rural primary care facilities.

No significant gender difference in NAFLD prevalence was observed in our cohort ($p = 0.883$), a finding that aligns with data from the Saudi study by Alyousef et al., who similarly reported no gender-based association.[16] While pre-menopausal oestrogen is classically considered hepatoprotective, data from South Asian populations suggest that the near-equivalent metabolic risk burdens in both sexes — particularly central obesity and insulin resistance — may attenuate gender differences in NAFLD prevalence.[20]

The significantly elevated liver enzymes in our NAFLD group, particularly ALT (56.2 ± 19.6 vs. 26.8 ± 9.8 U/L) and GGT (62.4 ± 22.3 vs. 30.6 ± 11.8 U/L), highlight the utility of liver function tests as accessible biomarkers for NAFLD screening in resource-limited community settings. ALT elevation emerged as the strongest independent predictor of NAFLD in logistic regression (OR 1.043), consistent with prior studies establishing ALT as the most sensitive routine biochemical marker of hepatocellular injury in NAFLD.[21] However, it is well recognised that a significant

proportion of NAFLD patients, even those with histological NASH, may have normal ALT values, limiting its use as a stand-alone screening tool.[22] The combination of USG and LFT therefore provides greater sensitivity and clinical utility than either modality alone.

The progressive elevation of LFTs across ultrasonographic steatosis grades (Grades 0 to III) observed in our study demonstrates a dose-response relationship between the degree of sonographic hepatic steatosis and biochemical evidence of liver dysfunction. This gradient correlation is consistent with findings reported by Misra et al., who demonstrated that higher grades of hepatic steatosis on USG corresponded to greater ALT and GGT elevations in Indian patients with metabolic risk factors.[23] The declining serum albumin levels across grades further suggest that more severe steatosis may begin to compromise hepatic synthetic function, representing a potential early signal of hepatocellular deterioration.

Hypertriglyceridaemia was the second strongest independent predictor of NAFLD in our regression model (OR 1.018 per unit increase in triglycerides), a finding mirrored in multiple prior studies.[16] [24] Elevated triglycerides contribute to NAFLD through increased hepatic de novo lipogenesis and impaired peripheral fatty acid oxidation, both driven by insulin resistance.[25] Similarly, reduced HDL — a well-established marker of insulin resistance and atherogenic dyslipidaemia — was an independent negative predictor of NAFLD (OR 0.917 per unit increase), underscoring the inter-relationship between dyslipidaemia and hepatic steatosis.

Higher BMI emerged as a significant independent predictor (OR 1.176 per unit increase), consistent with the central role of adiposity in driving ectopic fat deposition in the liver.[26] It is noteworthy, however, that patients in our study had a mean BMI of 30.2 kg/m^2, reflecting a relatively modest degree of obesity. This highlights that in South Asian populations, even moderate degrees of overweight are associated with significant visceral adiposity and insulin resistance sufficient to drive hepatic steatosis.[27]

The substantially higher prevalence of diabetes mellitus in the NAFLD group (69.6% vs. 52.2%) is consistent with the pivotal role of insulin resistance as the common pathophysiological link between NAFLD and T2DM. Multiple large-scale studies have established bidirectional relationships between NAFLD and T2DM, with each condition worsening the metabolic trajectory of the other.[28] Similarly, the higher prevalence of hypertension in the NAFLD group (75.9% vs. 63.8%) is in keeping with the shared pathophysiological basis of MetS components and NAFLD.[29]

From a clinical and public health perspective, the high prevalence of NAFLD in this rural community cohort — in which USG and LFTs were used as first-line tools — argues strongly for the incorporation of routine USG and biochemical liver panel screening into primary care and community health programs targeting MetS patients in rural Tamil Nadu. Current management strategies for NAFLD at the community level should prioritise lifestyle interventions, including a minimum 5–10% body weight reduction, adherence to a low-fat, low-refined-carbohydrate diet, and at least 150 minutes per week of moderate-intensity physical activity, all of which have demonstrated efficacy in reversing hepatic steatosis.[30] Pharmacological options, including metformin, GLP-1 receptor agonists, and SGLT-2 inhibitors for patients with coexisting T2DM, and statins for dyslipidaemia, may provide additional hepatoprotective benefits.[31]

Several limitations of the present study merit acknowledgment. The cross-sectional design precludes causal inference regarding the temporal relationship between MetS and NAFLD development. Liver biopsy — the gold standard for NAFLD diagnosis and fibrosis staging — was not performed due to its invasive nature and impracticality in a community setting, and therefore histological data on disease severity are lacking. USG, while practical and widely used, has reduced sensitivity for detecting mild (Grade I) steatosis and is operator-dependent. Waist circumference measurements may have limited precision due to variability in technique. Additionally, the single-centre design in one rural district limits generalisability to the broader Tamil Nadu population. Future prospective multicentre studies incorporating advanced imaging such as MRI-PDFF or transient elastography (FibroScan), genetic profiling, and longitudinal follow-up are warranted.

5. Conclusion

This community-based cross-sectional study demonstrates a high prevalence of NAFLD (44.8%) among MetS patients in rural Tamil Nadu, India. Elevations in liver enzymes — particularly ALT and GGT — alongside hypertriglyceridaemia, hyperglycaemia, reduced HDL cholesterol, higher BMI, and older age are independently associated with the presence of NAFLD in this population. The strong gradient correlation between ultrasonographic steatosis grade and LFT derangement confirms the clinical value of combined USG and biochemical liver panel assessment for NAFLD screening. These findings underscore the critical need for routine integrated screening programs targeting MetS patients at the community and primary care level in rural South India to enable

early diagnosis, risk stratification, and timely therapeutic intervention, thereby mitigating the long-term risks of progressive liver disease, type 2 diabetes, and cardiovascular complications in this high-risk population.

ACKNOWLEDGMENT:

The all authors wish to express gratitude to the Bharath Medical College and Hospital, Chennai, for supporting this work.

CONFLICTS OF INTEREST

The authors declare no conflicts of interest.

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