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Evaluation of non-alcoholic fatty liver disease in metabolic syndrome patients using fibro scan

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Abstract:
Non-alcoholic fatty liver disease (NAFLD) is common in metabolic syndrome and may progress to advanced liver disease. Therefore, it is of interest to evaluate the hepatic steatosis and fibrosis using FibroScan in 100 adults with metabolic syndrome. Hepatic steatosis was common, while higher CAP and LSM values were associated with increased body mass index, fasting glucose, HbA1c, triglycerides and waist circumference ($p < 0.05$). Obesity and diabetes were identified as independent predictors of advanced fibrosis. Thus, fibroScan facilitates early detection and risk assessment of NAFLD in patients with metabolic syndrome.

Keywords: Non-alcoholic fatty liver disease (NAFLD), metabolic syndrome, fibroscan, transient elastography, controlled attenuation parameter, liver stiffness, hepatic steatosis, fibrosis

Background:
Non-alcoholic fatty liver disease (NAFLD) is one of the most common chronic liver diseases worldwide and is strongly associated with metabolic dysfunction and metabolic syndrome **[1]**. NAFLD is characterized by excessive hepatic fat accumulation in individuals without significant alcohol consumption and encompasses a spectrum ranging from simple steatosis and steatohepatitis to progressive fibrosis, cirrhosis and hepatocellular carcinoma **[2]**. The global burden of NAFLD has increased substantially in parallel with the rising prevalence of obesity, type 2 diabetes mellitus, dyslipidemia, hypertension and other cardiometabolic risk factors **[3]**. Metabolic syndrome comprises interconnected abnormalities, including central obesity, insulin resistance, hyperglycemia, hypertension, hypertriglyceridemia and reduced high-density lipoprotein cholesterol **[4]**. These metabolic abnormalities contribute to hepatic steatosis and fibrosis through increased free fatty acid flux, insulin resistance, oxidative stress and chronic inflammation **[5]**. NAFLD is particularly prevalent among individuals with metabolic syndrome and the presence and number of metabolic abnormalities are associated with greater hepatic fibrosis and disease severity **[6]**. In India, NAFLD represents an important and growing health problem, with population-based studies demonstrating a substantial prevalence and strong associations with obesity, diabetes and other metabolic risk factors **[7]**. Early identification of hepatic steatosis and fibrosis is clinically important because fibrosis stage is a major predictor of liver-related and overall mortality among patients with NAFLD **[8]**. Conventional ultrasonography is widely available for detecting hepatic steatosis but has limited sensitivity for mild steatosis and cannot reliably stage hepatic fibrosis **[9, 10]**. Therefore, it is of interest to evaluate the prevalence and severity of hepatic steatosis and fibrosis among patients with metabolic syndrome using FibroScan® and to evaluate their association with clinical, anthropometric and biochemical parameters.

Methodology:
Study design and setting:
A prospective observational cross-sectional study was conducted in the Department of General Medicine in collaboration with the Department of Gastroenterology at a tertiary care teaching hospital over a period of 12 months. The study was approved by the Institutional Ethics Committee before commencement and written informed consent was obtained from all participants prior to enrolment.

Study population:
Adult patients diagnosed with metabolic syndrome attending the outpatient and inpatient departments were consecutively screened for eligibility. Metabolic syndrome was diagnosed according to the International Diabetes Federation (IDF) criteria.

Sample size:
A total of 100 patients fulfilling the eligibility criteria were included in the study. The sample size was determined based on the anticipated prevalence of NAFLD among patients with metabolic syndrome, with a 95% confidence level and adequate precision, while accounting for possible exclusions and incomplete evaluations.

Inclusion criteria:
[1] Adults aged 18 years and above.
[2] Patients diagnosed with metabolic syndrome according to the IDF criteria.
[3] Patients willing to provide written informed consent.
[4] Patients undergoing FibroScan evaluation during the study period.

Exclusion criteria:
[1] Significant alcohol consumption (>20 g/day for women and >30 g/day for men).
[2] Viral hepatitis (HBV or HCV infection).
[3] Autoimmune liver disease.
[4] Drug-induced liver disease.
[5] Wilson's disease or hemochromatosis.
[6] Pregnancy.
[7] Patients with known chronic liver disease of other etiologies.
[8] Patients with malignant diseases.
[9] Incomplete clinical or FibroScan data.

Data collection:
After obtaining informed consent, demographic information including age and sex was recorded. A detailed medical history regarding diabetes mellitus, hypertension, dyslipidemia, smoking status, medication use and duration of metabolic syndrome was obtained. Anthropometric measurements including height, weight, body mass index (BMI), waist circumference and blood pressure were recorded using standardized procedures.

Laboratory investigations:
After overnight fasting, venous blood samples were collected for estimation of fasting blood glucose, glycated hemoglobin (HbA1c), lipid profile (total cholesterol, triglycerides, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol), liver function tests (alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase, total bilirubin, serum albumin), complete blood count, serum creatinine and viral hepatitis markers (HBsAg and anti-HCV) to exclude secondary causes of liver disease.

FibroScan assessment:
Hepatic steatosis and liver fibrosis were evaluated using FibroScan® (Echosens, Paris, France) performed by a trained operator following standard manufacturer guidelines. Participants were instructed to fast for at least three hours before the examination. Liver stiffness measurement (LSM) was expressed in kilopascals (kPa), while hepatic steatosis was assessed using the Controlled Attenuation Parameter (CAP) expressed in decibels per meter (dB/m). At least ten valid measurements with a success rate greater than 60% and an interquartile range to median ratio of less than 30% were considered reliable.

Steatosis was graded according to CAP values as:
[1] **S0:** <248 dB/m
[2] **S1:** 248–267 dB/m
[3] **S2:** 268–279 dB/m
[4] **S3:** ≥280 dB/m

Liver fibrosis was staged using liver stiffness values:
[1] **F0–F1:** <7.0 kPa
[2] **F2:** 7.0–8.6 kPa
[3] **F3:** 8.7–10.2 kPa
[4] **F4:** >10.3 kPa

Outcome measures:
The primary outcome was the prevalence and severity of NAFLD among patients with metabolic syndrome as assessed by FibroScan. Secondary outcomes included the association between liver steatosis, liver fibrosis and individual components of metabolic syndrome, including obesity, diabetes mellitus and hypertension, dyslipidemia and biochemical parameters.

Statistical analysis:
Data were entered into Microsoft Excel and analyzed using IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD), whereas categorical variables were presented as frequency and percentage. Comparisons between groups were performed using the independent Student's *t*-test or one-way analysis of variance (ANOVA) for continuous variables and the Chi-square test or Fisher's exact test for categorical variables. Correlations between FibroScan parameters (CAP and LSM) and clinical or biochemical variables were assessed using Pearson's or Spearman's correlation coefficient as appropriate. Multivariable logistic regression analysis was performed to identify independent predictors of significant liver fibrosis. A two-tailed p-value <0.05 was considered statistically significant.

Results:
A total of 100 patients with metabolic syndrome were included in the study. The mean age was 52.8 ± 10.7 years, and males constituted 58% of the study population. The mean body mass index (BMI) was 29.3 ± 4.2 kg/m², and the mean waist circumference was 101.8 ± 9.6 cm. Type 2 diabetes mellitus and hypertension were present in 72% and 68% of participants, respectively, while dyslipidemia was observed in 74% **(Table 1).** The mean fasting blood glucose was 156.7 ± 48.2 mg/dL, and the mean HbA1c was 7.9 ± 1.4%. The mean serum triglyceride, total cholesterol, HDL cholesterol, and LDL cholesterol levels were 218.6 ± 74.5, 209.4 ± 41.8, 39.8 ± 8.1, and 126.5 ± 32.4 mg/dL, respectively. The mean alanine aminotransferase (ALT) and aspartate aminotransferase (AST) levels were 54.8 ± 28.6 U/L and 47.2 ± 21.3 U/L, respectively **(Table 2).** FibroScan evaluation revealed hepatic steatosis in 84% of patients. Severe steatosis (S3) was the most prevalent grade, affecting 39% of participants, followed by S2 in 25%, S1 in 20%, and S0 in 16%. The mean Controlled Attenuation Parameter (CAP) was 292.8 ± 41.6 dB/m, while the mean Liver Stiffness Measurement (LSM) was 8.1 ± 3.4 kPa **(Tables 3 and 4).** Regarding liver fibrosis staging, 54% of participants had significant fibrosis (F2–F4), including 20% with F2, 24% with F3, and 10% with F4. The remaining 46% were classified as F0–F1 **(Table 5).** Patients with obesity (BMI ≥30 kg/m²) had significantly higher mean CAP scores than those without obesity (311.6 ± 37.5 vs. 272.4 ± 35.8 dB/m; p. Patients with obesity (BMI ≥30 kg/m²) had significantly higher mean CAP scores than those without obesity (311.6 ± 37.5 vs. 272.4 ± 35.8 dB/m; $p < 0.001$) and significantly higher liver stiffness

measurements (9.3 ± 3.7 vs. 6.8 ± 2.1 kPa; p < 0.001) **(Table 6)**. Similarly, patients with diabetes mellitus had significantly higher mean CAP scores (301.8 ± 38.6 vs. 269.5 ± 34.9 dB/m; p < 0.001) and liver stiffness measurements (8.8 ± 3.6 vs. 6.3 ± 2.2 kPa; p = 0.002) than those without diabetes **(Table 7)**. CAP scores demonstrated significant positive correlations with BMI (r = 0.58), waist circumference (r = 0.54), fasting blood glucose (r = 0.43), HbA1c (r = 0.47), and triglyceride levels (r = 0.41), with all correlations reaching statistical significance (p < 0.001) **(Table 8)**. In multivariable logistic regression analysis, obesity (adjusted OR = 3.48, 95% CI: 1.42–8.52; p = 0.006) and diabetes mellitus (adjusted OR = 2.91, 95% CI: 1.18–7.16; p = 0.020) were independently associated with significant liver fibrosis (≥F2). Hypertension, hypertriglyceridemia, and age greater than 50 years were not significantly associated with significant fibrosis (p > 0.05) **(Table 9).**

Table 1: Baseline demographic and clinical characteristics (n=100)

Variable	Value
Age (years), Mean ± SD	52.8 ± 10.7
Male, n (%)	58 (58.0)
Female, n (%)	42 (42.0)
BMI (kg/m²), Mean ± SD	29.3 ± 4.2
Waist circumference (cm), Mean ± SD	101.8 ± 9.6
Systolic BP (mmHg), Mean ± SD	142.6 ± 15.8
Diastolic BP (mmHg), Mean ± SD	88.7 ± 9.4
Diabetes mellitus, n (%)	72 (72.0)
Hypertension, n (%)	68 (68.0)
Dyslipidemia, n (%)	74 (74.0)

Table 2: Laboratory characteristics

Parameter	Mean ± SD
Fasting blood glucose (mg/dL)	156.7 ± 48.2
HbA1c (%)	7.9 ± 1.4
Total cholesterol (mg/dL)	209.4 ± 41.8
Triglycerides (mg/dL)	218.6 ± 74.5
HDL cholesterol (mg/dL)	39.8 ± 8.1
LDL cholesterol (mg/dL)	126.5 ± 32.4
ALT (U/L)	54.8 ± 28.6
AST (U/L)	47.2 ± 21.3

Table 3: Distribution of hepatic steatosis according to CAP score

Steatosis Grade	CAP Value (dB/m)	n (%)
S0	<248	16 (16.0)
S1	248–267	20 (20.0)
S2	268–279	25 (25.0)
S3	≥280	39 (39.0)

Table 4: FibroScan parameters

Parameter	Mean ± SD
CAP (dB/m)	292.8 ± 41.6
Liver stiffness (kPa)	8.1 ± 3.4

Table 5: Liver fibrosis staging

Fibrosis Stage	Liver Stiffness	n (%)
F0–F1	<7.0 kPa	46 (46.0)
F2	7.0–8.6 kPa	20 (20.0)
F3	8.7–10.2 kPa	24 (24.0)
F4	>10.3 kPa	10 (10.0)

Table 6: Association of obesity with FibroScan findings

Variable	BMI <30 kg/m² (n=48)	BMI ≥30 kg/m² (n=52)	p-value
CAP (dB/m)	272.4 ± 35.8	311.6 ± 37.5	<0.001
Liver stiffness (kPa)	6.8 ± 2.1	9.3 ± 3.7	<0.001

Table 7: Association of diabetes with FibroScan parameters

Variable	Diabetes (n=72)	No Diabetes (n=28)	p-value
CAP (dB/m)	301.8 ± 38.6	269.5 ± 34.9	<0.001
Liver stiffness (kPa)	8.8 ± 3.6	6.3 ± 2.2	0.002

Table 8: Correlation of CAP score with metabolic variables

Variable	Correlation (r)	p-value
BMI	0.58	<0.001
Waist circumference	0.54	<0.001
Fasting blood glucose	0.43	<0.001
HbA1c	0.47	<0.001
Triglycerides	0.41	<0.001

Table 9: Multivariable logistic regression for significant fibrosis (≥F2)

Variable	Adjusted OR	95% CI	p-value
BMI ≥30 kg/m²	3.48	1.42–8.52	0.006
Diabetes mellitus	2.91	1.18–7.16	0.020
Hypertension	1.46	0.63–3.37	0.372
Hypertriglyceridemia	1.88	0.82–4.31	0.136
Age >50 years	1.71	0.74–3.92	0.208

Discussion:

The present study evaluated the prevalence and severity of non-alcoholic fatty liver disease (NAFLD) among patients with metabolic syndrome using FibroScan. Hepatic steatosis was detected in 84% of participants, while 54% showed significant liver fibrosis (F2–F4). CAP and liver stiffness measurements showed significant associations with obesity, diabetes mellitus, waist circumference, fasting blood glucose, HbA1c and serum triglycerides, highlighting the close relationship between metabolic syndrome and progressive liver disease. Our findings are consistent with those reported by Alfadda *et al.* (2022) **[11]**, who investigated 490 patients with type 2 diabetes using transient elastography. They reported hepatic steatosis in 80.8% of participants, with severe steatosis being the predominant grade. Similar to our study, increasing obesity and poor glycemic control were strongly associated with higher CAP values, emphasizing the importance of FibroScan as a non-invasive screening tool in high-risk metabolic populations. The association between metabolic abnormalities and hepatic steatosis observed in our study also agrees with the findings of Valenti *et al.* (2023) **[12]**, who evaluated over 1,200 individuals with metabolic dysfunction. Their study demonstrated that abdominal circumference, body mass index, diabetes, fasting insulin, triglycerides and reduced HDL cholesterol were independent determinants of CAP values. Likewise, we observed significant positive correlations between CAP score and BMI, waist circumference, fasting blood glucose, HbA1c and triglyceride levels, supporting the concept that insulin resistance and visceral obesity are major contributors to hepatic fat accumulation. Our results further support the diagnostic utility of FibroScan for assessing hepatic fibrosis. Seo *et al.* (2023) **[13]** compared transient elastography with controlled attenuation parameter (CAP) against two-dimensional shear wave elastography in patients with NAFLD and reported comparable

diagnostic performance for liver fibrosis assessment. They concluded that transient elastography remains a reliable and practical method for evaluating both steatosis and fibrosis in routine clinical practice. The present study similarly demonstrated successful assessment of both parameters using a single non-invasive examination, reinforcing FibroScan as an effective modality for evaluating NAFLD severity. Comparable findings were also reported by Cao *et al.* (2022) **[14]**, whose systematic review and meta-analysis demonstrated that CAP and liver stiffness measurement possess high diagnostic accuracy for grading hepatic steatosis and fibrosis in NAFLD. The pooled evidence supported the routine use of FibroScan as a non-invasive alternative to liver biopsy for screening and disease staging. Our findings corroborate these conclusions by demonstrating significant associations between FibroScan-derived measurements and established metabolic risk factors in patients with metabolic syndrome. The prognostic significance of liver stiffness observed in our study is also supported by Wong *et al.* (2023) **[15]**, who evaluated non-invasive fibrosis assessment in over 1,200 patients with NAFLD. They demonstrated that liver stiffness measurement obtained using vibration-controlled transient elastography effectively stratified patients according to their risk of liver-related adverse outcomes and outperformed several conventional clinical scores **[16]**. The significant association between obesity, diabetes mellitus and advanced fibrosis in our study similarly highlights the value of FibroScan for identifying high-risk individuals who may benefit from early intervention and long-term surveillance. Overall, the findings of the present study are in agreement with contemporary literature demonstrating that metabolic syndrome is strongly associated with hepatic steatosis and fibrosis and that FibroScan provides a rapid, reproducible and non-invasive assessment of disease severity. Early identification of patients with advanced fibrosis allows timely implementation of lifestyle modification, optimization of metabolic risk factors and referral for specialized hepatology care, thereby reducing the risk of progression to cirrhosis and hepatocellular carcinoma.

Conclusion:

Non-alcoholic fatty liver disease was highly prevalent among patients with metabolic syndrome, with 84% exhibiting hepatic steatosis and 54% demonstrating significant liver fibrosis. Obesity, diabetes mellitus, waist circumference, glycemic parameters, and triglyceride levels were significantly associated with FibroScan findings. FibroScan may facilitate early detection and risk stratification of hepatic steatosis and fibrosis, supporting timely intervention in patients with metabolic syndrome.

Limitations:

This study has several limitations. It was conducted at a single tertiary care center with a relatively small sample size of 100 participants, which may limit the generalizability of the findings. The cross-sectional design precluded assessment of disease progression and causal relationships between metabolic syndrome and NAFLD. Liver biopsy, the reference standard for diagnosing and staging NAFLD, was not performed because of its invasive nature; therefore, FibroScan findings could not be histologically validated. Additionally, long-term clinical outcomes and the effects of therapeutic interventions were not evaluated.

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