



Original Research Article

ASSESSMENT OF LIVER FIBROSIS IN MASLD: A COMPARISON OF FIBROSCAN WITH FIB-4 SCORE, NFS, BARD SCORE AND FIBROMETER

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ABSTRACT

Background: Metabolic dysfunction-associated steatotic liver disease (MASLD), formerly known as non-alcoholic fatty liver disease (NAFLD), is a common chronic liver disease that can progress to fibrosis, cirrhosis, and hepatocellular carcinoma. Non-invasive methods for fibrosis assessment are increasingly used as alternatives to liver biopsy. This study compared FibroScan with FIB-4, NAFLD Fibrosis Score (NFS), BARD score, and FibroMeter in evaluating liver fibrosis.

Materials and Methods: This cross-sectional analytical study included 101 participants (66 MASLD patients and 35 controls). Clinical, anthropometric, and biochemical parameters were recorded. FIB-4, NFS, BARD, and FibroMeter scores were calculated using validated formulas. Liver stiffness was assessed using transient elastography (FibroScan). Correlations between FibroScan and serum-based fibrosis scores were analyzed using Spearman's rho correlation.

Results: MASLD patients were significantly older and had higher adiposity indices, insulin resistance markers, dyslipidemia, ALT, ferritin levels, FibroMeter scores, FibroScan liver stiffness values, and NFS compared with controls ($p < 0.05$). FibroScan showed weak positive correlations with FibroMeter ($r = 0.191$, $p = 0.056$) and FIB-4 ($r = 0.194$, $p = 0.052$), which were not statistically significant. Significant positive correlations were observed between FibroScan and BARD score ($r = 0.213$, $p = 0.032$) and NFS ($r = 0.258$, $p = 0.009$), with NFS showed the strongest association. Strong correlations were found among serum-based fibrosis markers, particularly between FIB-4 and NFS ($r = 0.743$, $p = 0.0001$).

Conclusion: Serum-based fibrosis scores showed high concordance with each other but limited correlation with FibroScan. NFS showed strongest association with liver stiffness. A stepwise approach using serum fibrosis scores for screening followed by FibroScan may improve non-invasive fibrosis assessment in MASLD.

Keywords: NAFLD, MASLD, NFS.

INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) which is recently redefined as metabolic dysfunction-

associated steatotic liver disease (MASLD) is a common chronic hepatic condition worldwide with a reported prevalence of 30%.^[1,2] The clinical spectrum of MASLD ranges from mild to severe non-alcoholic

steatohepatitis (NASH) and may progress to fibrosis and cirrhosis. Patients with MASLD are at increased risk of death related to cardiovascular diseases and hepatocellular carcinoma (HCC).^[3] Therefore, it is important to accurately identify individuals at risk for NASH and subsequent progression to cirrhosis. Since, liver biopsy, the gold standard method is invasive and carries potential risks, reliable non-invasive methods for assessing liver fibrosis are essential for effective disease management, prognostic evaluation, and timely intervention.^[4] Non-invasive methods such as FibroScan, FIB-4 score, and FibroMeter have gained prominence as viable alternatives for evaluating fibrosis in patients with MASLD.^[5,6]

Transient elastography (TE) is a reliable, non-invasive method for evaluating fibrosis. Transient elastography using FibroScan has been commonly recommended for assessing liver stiffness and fibrosis.^[7-9] However, scarce availability of TE in resource limited settings necessitated the practical need for a laboratory-based risk stratification system.^[10,11]

The Fibrosis-4 (FIB-4) index, Non-Alcoholic Fatty Liver Disease Fibrosis Score (NFS) and BARD score incorporates routinely measured laboratory parameters in the assessment of hepatic fibrosis. FIB-4 uses parameters like age, aspartate transaminase (AST), alanine aminotransferase (ALT), and platelet count whereas NFS includes BMI, albumin, and diabetes in addition to the parameters used for FIB-4 index.^[12-14]

FibroMeter tests which uses serum biomarkers have been shown to have adequate level of accuracy in detecting patients with NAFLD and advanced fibrosis.^[6,15] These non-invasive methods are economical and easily accessible making them practical choices for evaluating hepatic fibrosis, especially in resource-limited settings where advanced imaging is not readily accessible. However, evidence on their diagnostic accuracy remains limited. Therefore, this study aimed to assess the effectiveness of FIB-4, NFS, BARD and FibroMeter scores in evaluating hepatic fibrosis using FibroScan as the reference standard.

MATERIALS AND METHODS

In this cross-sectional analytical study, a total of one hundred and one (n=101) consecutive patients with suspected MASLD attending gastroenterology department were enrolled. Patients with significant alcohol consumption, viral hepatitis, autoimmune liver disease, other known causes of chronic liver disease, history of hepatotoxic drug use, or incomplete data were excluded. Sociodemographic and clinical details such as age, sex, body mass index, duration of illness, and associated metabolic comorbidities were recorded.

All patients underwent laboratory evaluation including HbA1c, liver function tests (serum AST, ALT, bilirubin, albumin, and globulin), lipid profile,

renal function tests (blood urea and creatinine), complete blood count including platelet count, prothrombin time, ferritin, and ceruloplasmin. Non-invasive fibrosis scores were calculated using standard validated formulas, and patients were categorized into fibrosis risk groups based on established cut-off values.^[16]

Liver stiffness measurement was performed using transient elastography (FibroScan) by a trained operator following standard protocols, with at least 10 valid measurements obtained per patient; results were expressed as median liver stiffness values in kilopascals and considered reliable when the interquartile range was less than 30% of the median. Statistical analysis was carried out using SPSS software, with continuous variables expressed as median (interquartile range) and categorical variables as frequencies and percentages. Correlation and diagnostic performance between FibroScan and non-invasive fibrosis scores (FIB-4 Score, NFS, BARD score and Fibrometer score) were assessed using Spearman's rho Correlations, and a p-value <0.05 was considered statistically significant.

RESULTS

A total of 101 subjects (35 controls and 66 MASLD patients) were included in the analysis. Table 1 shows clinical characteristics of study population. MASLD patients were significantly older than controls (median 49 vs. 32 years, $p = 0.0001$). Measures of adiposity, including body weight, waist and hip circumference, BMI, and mid-arm circumference, were significantly higher in the MASLD group ($p = 0.0001$ for all), while height did not differ between groups. MASLD subjects demonstrated significantly higher fasting insulin, fasting blood glucose, and HbA1c levels ($p = 0.0001$), indicating greater insulin resistance. Diastolic blood pressure was significantly elevated ($p = 0.002$), whereas systolic blood pressure was comparable between groups. Lipid profile analysis revealed significantly higher triglycerides, total cholesterol, LDL, and VLDL levels, along with lower HDL levels in MASLD patients ($p < 0.05$ for all). Serum ALT, ferritin, total bilirubin, and conjugated bilirubin levels were significantly higher in the MASLD group, while AST, SAP, GGT, albumin, coagulation parameters, and ceruloplasmin showed no significant differences. Platelet counts were significantly increased and mean platelet volume was reduced in MASLD subjects ($p < 0.05$). Non-invasive fibrosis assessment showed significantly higher FibroMeter scores, FibroScan liver stiffness values, and NAFLD fibrosis scores in NAFLD patients compared to controls ($p < 0.05$), whereas BARD score and FIB-4 index did not differ significantly between the two groups.

[Table 2] presents the Spearman's rho correlation analysis between FibroScan and non-invasive fibrosis scores (FibroMeter, BARD, FIB-4, and NFS). FibroScan showed a weak positive correlation with FibroMeter ($r = 0.191$) and FIB-4 ($r = 0.194$);

however, these associations did not reach statistical significance ($p = 0.056$ and $p = 0.052$, respectively). A significant positive correlation was observed between FibroScan and BARD score ($r = 0.213$, $p = 0.032$). The strongest association with FibroScan was seen for the NAFLD Fibrosis Score (NFS), which demonstrated a significant positive correlation ($r = 0.258$, $p = 0.009$).

Among the serum-based fibrosis indices, strong and highly significant correlations were observed. FibroMeter correlated significantly with BARD ($r =$

0.373), FIB-4 ($r = 0.559$), and NFS ($r = 0.649$), all with $p = 0.0001$. Similarly, BARD showed moderate positive correlations with FIB-4 ($r = 0.355$) and NFS ($r = 0.454$). The strongest correlation overall was observed between FIB-4 and NFS ($r = 0.743$, $p = 0.0001$).

These findings indicate that while FibroScan shows only weak-to-moderate correlations with serum-based fibrosis scores, the non-invasive scoring systems demonstrate strong internal agreement, particularly between FIB-4 and NFS.

Table 1: Clinical characteristics of study population.

Clinical Parameter	CONTROL (n=35) Median (IQR)	NAFLD (n=66)	p Value
Age (Yrs)	32 (24-43)	49 (42-56)	0.0001
Height	162 (157-165)	162 (154-169.25)	0.977
weight	58.4 (49.5-67)	74.45 (67.975-86.625)	0.0001
WC	80 (69-90)	100.50 (94.75-109.25)	0.0001
HC	90 (81-92)	103 (98-111)	0.0001
BMI (kg/m ²)	21.4 (18.9-24)	28.6 (25.75-31.7)	0.0001
Fasting insulin μ U/ml	6.7 (4.8-9.7)	10.05 (8.025-12.450)	0.0001
HbA1C (%)	5.1 (4.9-5.5)	5.65 (5.3-6.5)	0.0001
Systolic Blood Pressure (mmHg)	110 (110-120)	120 (110-120)	0.226
Diastolic Blood Pressure (mmHg)	70 (70-80)	80 (70-80)	0.002
Triglyceride (mg/dl)	74 (46-107)	118 (99-166.75)	0.0001
Cholesterol (mg/dl)	166 (149-184)	185 (164-214)	0.012
HDL (mg/dl)	48 (43-59)	41.5 (38-48.05)	0.007
LDL (mg/dl)	106 (92-126)	122 (100-148.50)	0.010
VLDL	14.8 (9.2-21.4)	23.2 (19.8-31)	0.0001
CERULOPLASMIN (mg/dl)	0.240 (0.210-0.280)	0.2300 (0.2100-0.2700)	0.415
ALT IU/L	25 (20-30)	32.50 (25-42.25)	0.0001
AST IU/L	18 (15-22)	19 (16-26.25)	0.238
SAP IU/L	72 (61-89)	77.5 (63.75-97)	0.191
GGT IU/L	28 (19-39)	33 (27-45.25)	0.070
PT (secs)	14.3 (13.8-14.7)	14.10 (13.475-14.725)	0.338
INR	1.07 (1.02-1.11)	1.07 (1.0075-1.140)	0.920
Ferritin (ng/ml)	26.7 (16.4-42.3)	57.3 (25.675-93.350)	0.005
Urea (mg/dl)	8 (7-11)	8 (7-10.25)	0.506
Creatinine (mg/dl)	0.72 (.61-.96)	0.8350 (0.7375-0.9700)	0.051
Total Bilirubin (mg/dl)	0.49 (0.38-0.71)	0.6450 (0.4900-0.8525)	0.004
Conjugated Bilirubin (mg/dl)	0.100 (.060-.130)	0.1400 (0.1175-0.1900)	0.0001
Albumin (g/dl)	4.3 (4.1-4.5)	4.2 (3.975-4.5)	0.240
Globulin (mg/dl)	3.5 (3.2-3.7)	3.450 (3.175-3.725)	0.833
WBC	-	7.4 (6.175-8.6)	
Hb (g/dl)	13.2 (12.5-15.4)	13.65 (12.5-15.2)	0.537
HCT (%)	39.6 (37.9-44.1)	41.25 (38-45.05)	0.484
MCV (fl)	87.3 (82-91)	84.3 (80.8-87.7)	0.079
MCH (pg)	29.6 (27.4-30.3)	28.4 (26.55-29.625)	0.055
MCHC (g/dl)	33.5 (33-34.1)	33.55 (32.9-34)	0.708
Platelet/L	249 (180-291)	287.5 (237.25-320.75)	0.013
RDW	12.2 (11.7-13)	12.55 (12.20-13.125)	0.136
MPV (fl)	9.1 (8.3-9.6)	8.6 (8.1-9.1)	0.033
RBS mg/dl	100.5 (69.25-269)	-	
FBG mg/dl	84 (80-89)	92.5 (87-114.25)	0.0001
Fibrometer	0.00 (0.00-0.030)	0.0300 (0.0100-0.0900)	0.001
Fibroscan (kPa)	4.6 (4.20-5.60)	5.35 (4.30-7.625)	0.038
Fibroscan	4.6 (4.10-5.50)	5.35 (4.3-7.525)	0.021
BARD score	0.00 (0.00-2.0)	1 (0.0-2)	0.143
FIB4	0.50 (0.35-0.81)	0.5600 (0.4775-0.8625)	0.189
NFS	-0.93700 (-1.99700--0.04000)	0.12250 (-1.41625-1.04850)	0.025
Mid-arm Circumference Right (cms)	26 (23-27)	30 (28-32)	0.0001
Mid-arm Circumference Left (cms)	25 (22-27)	30 (28-32)	0.0001
Biceps Right (cms)	3 (3-4)	4 (3-5)	0.346
Biceps Left (cms)	3 (3-5)	4 (3-5)	0.507
Triceps Right (cms)	5 (4-5)	5 (4-6.25)	0.796
Triceps Left (cms)	5 (4-5)	5 (4-6.25)	0.813

Table 2: Correlation between FibroScan and non-invasive fibrosis scores (FibroMeter, BARD, FIB-4, and NFS).

		FibroScan	FibroMeter	BARD	fib 4	NFS
FibroScan	Correlation Coefficient	-	0.191	0.213(*)	0.194	0.258(**)
	P value	-	0.056	0.032	0.052	0.009
FibroMeter	Correlation Coefficient	0.191	-	0.373(**)	0.559(**)	0.649(**)
	P value	0.056	-	0.0001	0.0001	0.0001
BARD	Correlation Coefficient	0.213(*)	0.373(**)	-	0.355(**)	0.454(**)
	P value	0.032	0.0001	-	0.0001	0.0001
fib 4	Correlation Coefficient	0.194	0.559(**)	0.355(**)	-	0.743(**)
	P value	0.052	0.0001	0.0001	-	0.0001
NFS	Correlation Coefficient	0.258(**)	0.649(**)	0.454(**)	0.743(**)	-
	P value	0.009	0.0001	0.0001	0.0001	-

* Correlation is significant at the 0.05 level (2-tailed).

** Correlation is significant at the 0.01 level (2-tailed).

DISCUSSION

In this study, we compared metabolic characteristics, biochemical parameters, and non-invasive fibrosis markers in patients with MASLD and healthy individuals, and examined how commonly used fibrosis assessment tools relate to one another.

We have observed that patients with MASLD were significantly older than the control group, which is in agreement with previous studies showing that both the prevalence and severity of MASLD increase with age.^[17,18] Age plays an important role in progression of fibrosis and is included in several non-invasive fibrosis indices, such as FIB-4 and the NAFLD Fibrosis Score (NFS).^[19] As a result, the differences in age between the groups could partially account for the higher fibrosis-related scores seen in patients with NAFLD.

Measurements such as body weight, BMI, waist and hip circumference, and mid-arm circumference were markedly higher among individuals in the MASLD group. This further highlights the important role of obesity, especially the buildup of visceral fat, in the development of MASLD. Excess adipose tissue contributes to hepatic steatosis by increasing the release of free fatty acids from enhanced lipolysis, promoting systemic low-grade inflammation, and disrupting adipokine balance.^[20,21]

In our study, patients with MASLD also showed significant elevated levels of fasting insulin, fasting glucose, and HbA1c, consistent with the well documented link between insulin resistance and MASLD.^[22] Insulin resistance has been reported to cause hepatic fat accumulation and fibrosis by means of mechanisms such as oxidative stress, lipotoxicity, and activation of hepatic stellate cells.^[23]

Elevated levels of triglycerides, total cholesterol, LDL, and VLDL, along with reduced HDL was observed in MASLD patients. This lipid abnormality, commonly documented as characteristic of MASLD-associated dyslipidemia.^[8] Such abnormal amount of lipids in the blood, increases cardiovascular risk, which remains the leading cause of death in MASLD and may also contribute in the advancement of hepatic injury and disease progression.^[24]

MASLD patients in this study showed increased levels of ALT and ferritin, which may indicate underlying hepatocellular injury and iron-related

oxidative stress, both of which have been reported to be associated with fibrosis progression.^[25,26] In contrast, there is no significant difference in AST, GGT, SAP, albumin, and coagulation parameters between MASLD and control groups, suggesting that most patients were in early to intermediate stages of disease without severe fibrosis or cirrhosis.

NAFLD cases in our study have shown elevated platelet counts and reduced mean platelet volume. Similar hematological pattern has already been reported, which is considered to be indicative of ongoing low-grade systemic inflammation rather than portal hypertension, which is more commonly associated with low platelet counts in advanced liver fibrosis.^[27]

Non-invasive fibrosis assessment methods (FibroScan, FibroMeter, and NFS scores) showed significantly higher liver stiffness values in MASLD patients compared with controls and can be used in cases at increased risk of fibrosis. In contrast, the BARD score and FIB-4 index did not differ significantly between the two groups, likely because they are less sensitive in detecting early fibrosis and depends on fewer or less specific clinical variables.^[16,28]

We have observed that FibroScan has weak and statistically non-significant correlations with FibroMeter and FIB-4. FibroScan showed a moderate but significant correlation with the BARD score. The strongest correlation was with NFS, suggesting that the NFS score may better reflect fibrosis-related changes seen in liver stiffness. This finding is consistent with previous study, which reported that moderate association between blood-based fibrosis scores and transient elastography, especially in the early stages of the disease.^[29]

Highly significant correlations were found among the serum-based fibrosis markers. FibroMeter was strongly related to both FIB-4 and NFS, and the strongest correlation was between FIB-4 and NFS. The possible reason could be that these scoring systems are based on similar variables, including age, liver enzyme levels, and platelet count. Therefore, they may reflect related biological processes rather than providing entirely independent assessments of fibrosis.^[12] Although this consistency confirms their reliability, it also indicates some redundancy among the scores and limits their effectiveness as a standalone replacement for imaging techniques.

CONCLUSION

In conclusion, our findings indicate that serum-based fibrosis scores are useful as initial screening tools, but showed limited agreement with FibroScan. A step-by-step approach using serum markers for early risk assessment followed by detailed evaluation by FibroScan may provide an optimal balance of accuracy and convenience while minimizing the need for invasive liver biopsy. Longitudinal studies are needed to clarify how well these tools predict fibrosis progression and long-term clinical outcomes in patients with MASLD.

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