International Journal of Research in Medical Sciences
*Agrawal A et al. Int J Res Med Sci. 2024 Nov;12(11):4115-4121*
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20243358

**Original Research Article**

# Assessing the relationship between FibroScan and laboratory parameters in evaluating hepatic fibrosis in patients with non-alcoholic fatty liver disease

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**Received:** 14 August 2024
**Revised:** 08 October 2024
**Accepted:** 10 October 2024

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## ABSTRACT

**Background:** The gold standard for diagnosing steatosis, grading fibrosis, and determining its severity is liver biopsy (LB). An alternate, less expensive, and trouble-free technique for accurately identifying and measuring steatosis is the controlled attenuation parameter (CAP) value acquired with FibroScan.[3] Hence, the present study was undertaken to correlate laboratory parameters (AST/ALT ratio, AST platelet ratio index, Fib-4 score) with FibroScan as a marker of hepatic fibrosis in non-alcoholic fatty liver disease (NAFLD) patients.
**Methods:** 68 patients diagnosed to have NAFLD as per imaging were subjected to FibroScan using transient elastography within 2 weeks. Blood sample was taken for various lab tests. Area under receiver operating curve was plotted for predicting advanced fibrosis. Sensitivity and specificity were calculated.
**Results:** Based on ROC curves of APRI, FIB-4, and AST/ALT ratio for the detection of F2 of liver fibrosis the best index to diagnose F2 from lower stages of liver fibrosis was APRI, with an AUROC curve of 0.747 (95% confidence interval [CI] 0.599–0.894). The optimal cut-off of APRI was 0.276 for this purpose, with a sensitivity of 70.83%, specificity of 79.55%, PPV of 65.38%, NPV of 83.3%, and DA of 76.47%.
**Conclusions:** To conclude, it was found that the fibrosis stages increased significantly with APRI scores and Fib-4 score. AST/ALT ratio decreased with increase in FibroScan grade, however, mean of AST/ALT ratio was more in F2 grade than F1 with statistically non- significant relation. This can eliminate the need for LB in patients without clear indication.

**Keywords:** APRI, FIB-4, AST/ALT ratio, Hepatic fibrosis, NAFLD

## INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) is one of the leading causes of chronic liver disease. The spectrum of NAFLD includes simple steatosis, nonalcoholic steatohepatitis (NASH), advanced fibrosis, and cirrhosis.[1] NAFLD is the most prevalent cause of fatty liver (steatosis) that represents infiltration of fat in the liver (more than 5-10% of hepatocytes) without excessive alcohol consumption and other causes of liver disease.[2] Many NAFLD patients go undetected, therefore it is critical to identify people who could be at risk.[3] In the diagnosis of NAFLD and related disorders, liver biopsies can be extremely helpful and its findings can range from triglyceride deposition as droplets in the hepatocyte to more extensive forms of NASH.

The gold standard for diagnosing steatosis, grading fibrosis, and determining its severity is LB. However, patients are typically not prepared for this procedure because it is invasive and difficult to replicate.[2] Moreover, although LB is the most specific method of detecting and staging fibrosis, it is an invasive method and has many limitations including high cost,

haemorrhage, bile leak, infection, sampling error as it represents 1/50000 of liver volume. Furthermore, variations of opinion between pathologists leading to inter and intra observer discrepancies.[4] An alternate, less expensive, and trouble-free technique for accurately identifying and measuring steatosis is CAP value acquired with FibroScan.[2] Hence, present study undertaken to correlate lab parameters (AST/ALT ratio, AST platelet ratio index, Fib-4 score) with FibroScan as marker of hepatic fibrosis in NAFLD patients.

## METHODS

Hospital based analytical cross-sectional study was conducted among 68 patients from November 2022-July 2024 who underwent ultrasonography of abdomen with findings suggestive of NAFLD in the imaging were screened for inclusion into the study at department of general medicine, Pt. J. N. M. medical college, Raipur. Patients >18 years undergoing routine ultrasonography in the Department of Radiodiagnosis with findings suggestive of NAFLD were included in the study. Chronic alcoholics (as per DSM-5), patients on lipid lowering agents, anti- glycemic drugs pregnant women and acute and chronic viral hepatitis infected were excluded from the study. After obtaining approval from the institutional ethic committee and a written informed consent from the patients, present study was commenced. Patients were evaluated by history taking, examination and performing relevant investigations. Using criteria from the 2005 modified national cholesterol education program adult treatment panel III, patients were assessed for the presence of metabolic syndrome (MetS).[5]

Random blood glucose (RBG), fasting lipid profile that included total cholesterol, triglycerides (TG), high-density lipoprotein (HDL) cholesterol and low-density lipoprotein (LDL) cholesterol, glycated hemoglobin (HbA1c), complete blood count (CBC), liver biochemistry that included total bilirubin (TB), direct bilirubin (DB), alanine aminotransferase (ALT), aspartate aminotransferase (AST), were carried out under strict aseptic precautions. Patients diagnosed to have NAFLD as per imaging were subjected to FibroScan using Transient elastography within 2 weeks. Liver stiffness and CAP were measured using transient elastography and FibroScan®. In order to assess liver stiffness (LSM) and acquire CAP, the M probe was initially utilized. When the M probe failed, the XL probe designed for obese patients was utilized. For every subject, at least ten reliable measures were taken. When comparing the number of validated measures to the total number of measurements, a success rate of ≥60% and the ratio of liver stiffness's interquartile range (IQR) to the median (IQR/LSM) of ≤30% were deemed dependable and utilized in the final analysis.[6]

Data obtained was reported in MS excel and was checked for completeness and correctness. Categorical data was presented in number and percentage and quantitative data in mean±standard deviation. Area under receiver operating curve (AUROC) was plotted for visceral fat in predicting advanced fibrosis. Sensitivity and specificity were calculated. Data was analyzed using suitable statistical software.

## RESULTS

Table 1 shows age group-wise distribution of study subjects' results revealed that out of all 68 subjects 39 were female and 29 were male, 4 study participants belonged to 21-30 years, 5 subjects belonged to 31-40 years, 12 subjects belonged to 41-50 years, 23 subjects belonged to 51-60 years and 24 subjects belonged to >60 years of age group.

**Table 1: Age group-wise distribution of study subjects.**

| Age group (in years) | N | Percentage (%) |
|---|---|---|
| **21-30** | 4 | 5.9 |
| **31-40** | 5 | 7.4 |
| **41-50** | 12 | 17.6 |
| **51-60** | 23 | 33.8 |
| **>60** | 24 | 35.3 |
| **Total** | 68 | 100.0 |

Table 2 shows mean sugar-related parameters of study subjects results revealed that mean RBS was found 131.35 and mean Hb was found 12.25 gm% among study participants, mean TLC level was observed 5628.81 and mean platelet count was found 230.43, mean AST was found 28.47, mean ALT was 26.40, mean urea 22.63, mean creatinine 1.01, mean total bilirubin 0.62 md/dl, mean direct bilirubin 0.12 mg/dl, mean total cholesterol 252 mg/dl, mean triglyceride 162 mg/dl mean HDL 48.0 mg/dl, mean LDL 164.4 mg/dl and mean VLDL 32.6 were observed.

**Table 2: Mean lab parameters of study subjects.**

| laboratory parameters | Mean | SD |
|---|---|---|
| **HB (gm/dl)** | 12.25 | 2.11 |
| **TLC (per microlt)** | 5628.81 | 1000.74 |
| **Platelet count (10×9/lt)** | 230.43 | 70.91 |
| **RBS (mg/dl)** | 131.35 | 35.78 |
| **AST (U/lt)** | 28.47 | 15.09 |
| **ALT (U/lt)** | 26.40 | 13.66 |
| **Total bilirubin (mg/dl)** | 0.62 | 0.31 |
| **Direct bilirubin (mg/dl)** | 0.12 | 0.02 |
| **Indirect bilirubin (mg/dl)** | 0.50 | 0.40 |
| **Serum albumin (gm/dl)** | 4.0 | 0.50 |
| **Urea (mg/dl)** | 22.63 | 9.24 |
| **Creatinine (mg/dl)** | 1.01 | 0.31 |
| **Total cholesterol (mg/dl)** | 252 | 20.52 |
| **Triglyceride (mg/dl)** | 162 | 12.80 |
| **HDL cholesterol (mg/dl)** | 48.0 | 5.62 |
| **LDL cholesterol (mg/dl)** | 164.4 | 16.52 |
| **VLDL (mg/dl)** | 32.6 | 4.86 |

**Table 3: Mean of FibroScan and laboratory parameters of study subjects.**

| FibroScan and laboratory parameters | Mean | SD |
|---|---|---|
| **Hepatic fibrosis score** | 6.55 | 2.97 |
| **Fib 4 score** | 1.55 | 1.18 |
| **AST:ALT** | 1.49 | 2.88 |
| **APRI** | 0.31 | 0.27 |

Table 3 shows mean of fibro scan-related parameters of study subjects results revealed that mean hepatic fibrosis score was found 6.55 and mean fib 4 score was observed 1.55, mean AST/ALT was found 1.49 and mean APRI was observed 0.31.

**Table 4: Distribution of subjects according to FibroScan grades in patients.**

| FibroScan grades | N | Percentage (%) |
|---|---|---|
| **F0 (1-6 kPa)** | 39 | 57.4 |
| **F1 (6.1-7 kPa)** | 5 | 7.4 |
| **F2 (7.1-9 kPa)** | 17 | 25.0 |
| **F3 (9.1-10.3 kPa)** | NA | NA |
| **F4 (≥10.4 kPa)** | 7 | 10.3 |

Table 4 shows distribution of subjects according to FibroScan grades in patients results revealed that F0 grade observed in 39 subjects and F1 grade was observed in 5 study subjects, F2 grade was observed among 17 subjects and F4 grade was found in 7 study subjects.

Table 6 and Figure 1 demonstrates the ROC curves of APRI, FIB-4, and AST/ALT ratio for the detection of F2 of liver fibrosis from the lower stages. Based on these curves, the best index to diagnose F2 from lower stages of liver fibrosis was APRI, with an AUROC curve of 0.747 (95% confidence interval [CI] 0.599-0.894). The optimal cut-off of APRI was 0.276 for this purpose, with a sensitivity of 70.83%, specificity of 79.55%, PPV of 65.38%, NPV of 83.3%, and DA of 76.47%.

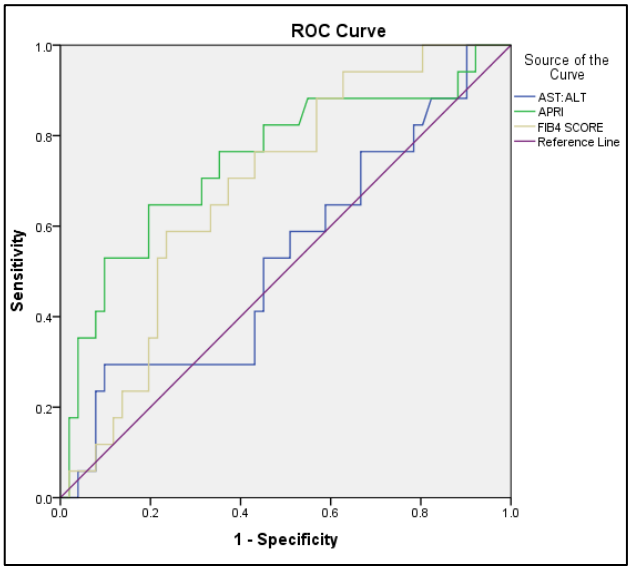


**Figure 1: ROC curves of APRI, FIB-4, and AST/ALT ratio for the detection of F2 of liver fibrosis from the lower stages.**

**Table 5: Comparison of distribution of subjects according to laboratory parameters with FibroScan grades.**

| Parameters | Grades | F0 | | F1 | | F2 | | F4 | | Chi-square value | P value |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | N | % | N | % | N | % | N | % | | |
| | Normal | 30 | 76.9 | 3 | 60 | 14 | 82.4 | 6 | 85.7 | | |
| **RBS** | Diabetic | 1 | 2.6 | 2 | 40 | 2 | 11.8 | 0 | 0 | 12.04 | 0.06 |
| | Impaired glucose | 12 | 30.8 | 0 | 0 | 5 | 29.4 | 1 | 14.3 | | |
| | Non-diabetic | 26 | 66.7 | 3 | 60 | 10 | 58.8 | 6 | 85.7 | | |
| **APRI** | <0.5 | 37 | 94.9 | 5 | 100 | 10 | 58.8 | 5 | 71.4 | 22.1 | <0.01 |
| | 0.5-1.5 | 2 | 5.1 | 0 | 0 | 7 | 41.2 | 1 | 14.3 | | |
| | >1.5 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 14.3 | | |
| **FIB-4 score** | <1.45 | 29 | 74.4 | 5 | 100 | 7 | 41.2 | 2 | 28.6 | 15.58 | 0.01 |
| | 1.45-3.39 | 8 | 20.5 | 0 | 0 | 9 | 52.9 | 3 | 42.9 | | |
| | ≥3.4 | 2 | 5.1 | 0 | 0 | 1 | 5.9 | 2 | 28.6 | | |
| **AST: ALT** | <0.8 | 6 | 15.4 | 3 | 60 | 2 | 11.8 | 0 | 0 | 8.69 | 0.033 |
| | ≥0.8 | 33 | 84.6 | 2 | 40 | 15 | 88.2 | 7 | 100 | | |

**Table 6: Diagnostic performance of the indices for the differentiation of F2 from lower stages.**

| Indices | AUC | Asymptotic 95% confidence interval | | P value | Optimal cut off | Sensitivity | Specificity | PPV | NPV | DA |
|---|---|---|---|---|---|---|---|---|---|---|
| | | Lower bound | Upper bound | | | | | | | |
| **APRI** | 0.747 | 0.599 | 0.894 | 0.002 | 0.276 | 70.83 | 79.55 | 65.38 | 83.3 | 76.47 |
| **AST:ALT** | 0.531 | 0.369 | 0.693 | 0.702 | 0.854 | 66.67 | 29.55 | 34.04 | 61.90 | 42.65 |
| **FIB4** | 0.686 | 0.552 | 0.820 | 0.022 | 0.969 | 87.5 | 47.73 | 47.73 | 87.5 | 61.76 |

## DISCUSSION

The present study was undertaken to correlate laboratory parameters (AST/ALT ratio, AST platelet ratio index, Fib-4 score) with FibroScan as a marker of hepatic fibrosis in NAFLD patients and the findings confirmed the significant correlation observed between the FibroScan results and the FIB-4 scores, AST/ALT ratios, and APRI ratios.

The maximum number of patients (33.8%) belonged to age group 51-60 years followed by 35.3% above 60 years, 17.6% belonged to 41-50 years, 7.4% belonged to 31-40 years and 5.9% in 21-30 years age group with 57.4% as females and 42.6% as male patients. The trend found was upsurge of cases with increase in age. Fallatah et al reported a mean age of 50.2 years and 53.3% were male patients.[7] Lin et al discovered the prevalence of NAFLD increased progressively with age, with rates of 11.7%, 15.9%, 21.5%, and 22.8% in the 20-34, 35-49, 50-64, and over-65-year-old age groups, respectively ($p<0.001$). In men, the highest occurrence of NAFLD was seen in the age range of 35-49 years, with a prevalence rate of 23.5%. Among women, the highest incidence of NAFLD was found in the age group of over 65 years, with a prevalence rate of 23.4%.

The laboratory values in the present study reported ALT and AST levels within normal range. Subjects with all stages of NAFLD have shown normal levels of liver enzymes, indicating that ALT and AST are not effective in predicting NAFLD.[8] The current standard range of liver transaminases found in patients with NAFLD may, hence, fail to accurately detect the existence of NAFLD. In the study conducted by Sanyal et al it was found that only 31% of individuals with NAFLD had alanine aminotransferase (ALT) levels above the normal laboratory limit of >40 U/L.[9] Additionally, 57% of individuals with NAFLD had ALT levels between 25 and 40 U/L. On the other hand, 67% of individuals without NAFLD had ALT levels below 25 U/L, but 30% of non-NAFLD individuals had ALT levels between 25 and 40 U/L. Fallatah et al also found the serum ALT levels were normal among NAFLD patients.[7] NAFLD patients have fluctuations in their liver enzyme levels. When their levels are raised, the rise is typically slight and usually limited to one or both of AST and ALT. However, it is crucial to highlight that while increased ALT levels are typically linked to histological NAFLD, a significant proportion of NAFLD patients have normal or almost normal liver enzyme levels. Thus, the ALT level alone is insufficient to exclude the presence of severe liver disease in patients who are suspected of having NAFLD.[3]

The mean values of total cholesterol and triglycerides were found to be elevated in the present study which was in concordance with reported by Tous et al, Lee et al and Kuipers et al.[10-12] Moreover, a study by Cheng et al reported that fatty liver can be considered as the hepatic consequence of metabolic syndrome.[13]

There is a high prevalence of metabolic syndrome and fatty liver among the elderly population. Metabolic disorders are closely related to fatty liver; moreover, fatty liver appears to be a good predictor for the clustering of risk factors for metabolic syndrome. These studies have reported high levels of triglycerides in NAFLD patients. The development of NAFLD is explained by a "two-hit hypothesis". The initial impact involves the buildup of triglycerides in liver cells, leading to a condition called simple steatosis. This is mostly caused by insulin resistance linked to obesity, and it heightens the liver's susceptibility to additional damage. The second impact is mostly lipotoxicity, which is induced by oxidative stress resulting from elevated lipid peroxidation, heightened formation of reactive oxygen species inside liver cells, mitochondrial malfunction, and inflammation. The activation of hepatic stellate cells can lead to the progression of steatohepatitis and fibrosis, as a result of these biological processes. Several biological substrates and circumstances that contribute to the development of NAFLD, such as insulin resistance and proinflammatory cytokines, have similar effects on arteries, leading to the occurrence of atherosclerosis. This may elucidate the correlation between NAFLD and cardiovascular disease (CVD). Triglycerides do not accumulate in the artery wall, despite their accumulation in hepatocytes.[14] Moreover, recent data indicates that disrupted regulation of cholesterol levels in the liver and the buildup of free cholesterol (FC) may be contributing factors in the development of NAFLD/NASH. Hepatic FC accumulation in NAFLD is caused by changes in intracellular cholesterol transport and imbalanced cellular cholesterol homeostasis. This is characterized by the activation of cholesterol biosynthetic pathways, increased cholesterol de-esterification, and reduced cholesterol export and bile acid synthesis pathways. The buildup of FC causes liver injury by activating intracellular signaling pathways in Kupffer cells (KCs), Stellate cells (HSCs), and hepatocytes. The stimulation of KCs and hepatic stellate cells (HSCs) induces an inflammatory response and the formation of fibrous tissue. Furthermore, the buildup of FC in liver mitochondria leads to impaired mitochondrial activity, leading to an elevated generation of reactive oxygen species. This, in turn, activates the unfolded protein response in the endoplasmic reticulum (ER), resulting in ER stress and apoptosis. These occurrences form a harmful cycle that adds to the persistence of steatosis and encourages continuous death of liver cells and damage to the liver, which in turn can lead to the advancement of the disease.[15]

The fibro scan-related parameters of study subjects' results revealed that mean hepatic fibrosis score was found 6.55±2.97 and mean fib 4 score was observed 1.55±1.18, mean AST/ALT was found 1.49±2.88 and mean APRI was observed 0.31±0.27. A significant inverse correlation of AST/ALT ($r=-0.091$) with hepatic fibrosis score whereas Fib-4, and APRI scores were elevated with increase in hepatic fibrosis score as found

in the present study. Our results are in concordance with Williams et al who reported that the ratio is usually greater than 2.0 in alcoholic liver disease and less than 1.0 in patients with nonalcoholic chronic liver disease (chronic hepatitis and chronic cholestatic syndromes).[16] In recent decades, there has been scientific investigation into the utilization of the AST:ALT ratio for distinguishing alcoholic liver disease (ALD) from other types of liver disease, specifically the NAFLD spectrum. Both AST and ALT enzymes rely on pyridoxal-5'-phosphate (vitamin B6) for optimal functioning. The lack of this substance in heavy drinkers who have poor nutrition has a more significant impact on the generation of ALT compared to AST, resulting in an increase in the AST:ALT ratio. An optimal AST:ALT ratio should be less than 1. Among individuals diagnosed with alcoholic liver disease, 92% exhibit an AST:ALT ratio more than 1, whereas 70% have a ratio exceeding 2. AST:ALT ratios greater than 2 are highly indicative of alcoholic liver disease, while ratios less than 1 are more suggestive of NAFLD. [17]

In our study maximum cases (57.4%) on FibroScan reported F0 (1-6 kPa) grade followed by 25% F2 (7.1-9 kPa), 10.3% had F4 (≥10.4 kPa) and 7.4% reported F1 (6.1-7 kPa) grade. The results confirmed that the fibrosis stages increased significantly with APRI scores and Fib-4 score. AST/ALT ratio decreased with increase in FibroScan grade, however, mean of AST/ALT ratio was more in F2 grade than F1 with statistically non-significant relation. A similar study by Fallatah et al found the mean stiffness score was 12.02 (SD: 12.7) kPa.[7] Forty-four patients (36%) had advanced fibrosis and there was a significant correlation between the FibroScan results and the AST/ALT ratios, the APRI scores, and the FIB-4 results which corresponds to our study. The results confirmed that the fibrosis stages increased significantly with elevated AST/ALT ratio (≥0.8), Fib-4 and APRI scores. A likely, Danaf et al reported high positive relation between AST/ALT ratio, APRI and Fib-4 scores with fibrosis stages in NAFLD patients and suggested that they could be used clinically in combination with FibroScan to predict significant fibrosis and cirrhosis and to avoid LB.[3]

The ROC curves of APRI, FIB-4, and AST/ALT ratio for the detection of F2 of liver fibrosis from the lower stages reported that based on these curves, the best index to diagnose F2 from lower stages of liver fibrosis was APRI, with an AUROC curve of 0.747 (95% confidence interval [CI] 0.599-0.894). The optimal cut-off of APRI was 0.276 for this purpose, with a sensitivity of 70.83%, specificity of 79.55%, PPV of 65.38%, NPV of 83.3%, and DA of 76.47%. In similar to our study, Moosavy et al found a noteworthy correlation between FibroScan outcomes and the three indicators of AST/ALT ratio, APRI, and FIB-4 ($p<0.001$), with the most significant correlation observed between FibroScan findings and APRI ($r=0.682$).[18] The diagnostic accuracy for the diagnosis of any grade of liver fibrosis was highest (77.15%) when using APRI ≥0.527, as indicated by an area under the receiver operating characteristic (AUROC) curve of 0.852 (95% confidence interval [CI] 0.807; 0.897, $p<0.001$). While the AUROC curve of APRI and FIB-4 showed similarity (0.864) in differentiating between F3/F4 and F0-F2 stages of liver fibrosis, FIB-4 exhibited the highest diagnosis accuracy (82.02%). Our results are also in concordance with the study carried by Amernia et al in which APRI achieved an AUROC curve of 0.923 at a cut-off value of 0.702.[19] It demonstrated a sensitivity of 84.1%, specificity of 88.2%, positive predictive value (PPV) of 66.1%, negative predictive value (NPV) of 95.3%, and diagnostic accuracy (DA) of 87.3% for this purpose. Another study by Sha FR et al[20] asserted that APRI had the highest level of accuracy among non-invasive indices in predicting F2/F3 liver fibrosis, surpassing FIB-4, AST/ALT ratio, and AST/ALT/platelet ratio. Furthermore, Yue et al found that using an APRI cut-off value of 0.8 was effective in identifying cases of bridging fibrosis (F ≥ 3).[21] The world health organization (WHO) has recommended the use of the APRI index to evaluate hepatic fibrosis in patients with chronic hepatitis B (CHB).[22] The threshold for severe fibrosis is set between 0.5 and 1.5.

It should be noted that FibroScan results may be less precise in individuals who are obese or have ascites, as the presence of excessive tissue or fluid can hinder the accuracy of the test.[23]

Furthermore, it should be noted that FibroScan results may exhibit variations among different devices, which might consequently impact the accuracy of diagnosis and subsequent treatment decisions.[24] Moreover, the precision of FibroScan outcomes may be contingent upon the operator's expertise and methodology, as the correct placement of the instrument on the skin is crucial for precise results.[25] Nevertheless, we attempted to resolve these concerns by employing a uniform device for all our patients, and all assessments were conducted by the same operator to mitigate inter-observer variability. In addition, inflammation might result in inaccurate FibroScan results, causing an overestimation of the severity of liver fibrosis.

To date, the histological assessment of fibrosis with LB is the gold standard, but obviously, invasiveness is the greater threshold. In addition, rare but potentially life-threatening complications, poor acceptability, sampling variability and cost maybe restrict its use. Furthermore, due to the epidemic of NAFLD worldwide and several limitations of LB evaluation, noninvasive assessment tools to detect fibrosis in NAFLD patients are needed.[81] Furthermore, the diagnosis of NAFLD is typically made in clinical settings through the use of ultrasound imaging, as doing a LB is not logistically feasible.[62] The findings of the present study encourages the use of readily available biomarkers and FibroScan plus abdominal US in the assessment of NAFLD instead of LB. FibroScan is a helpful instrument that was recently developed to assess

transient liver elasticity and expresses liver stiffness in KPa.

### *Limitations*

The study's sample size was limited, which may affect the generalizability of the results. The study's cross-sectional design precludes causal inferences and longitudinal assessments. Although non-invasive methods were used, LB was not performed for confirmation, potentially affecting accuracy.

## CONCLUSION

To conclude, it was found that the fibrosis stages increased significantly with APRI scores and Fib-4 score. AST/ALT ratio decreased with increase in FibroScan grade, however, mean of AST/ALT ratio was more in F2 grade than F1 with statistically non- significant relation.

The APRI test was used to diagnose F2 from lower stages of liver fibrosis, with an AUROC curve of 0.747 (95% CI 0.599-0.894). The most effective threshold value for APRI in assessing fibrosis phases was determined to be 0.276. This threshold yielded a sensitivity of 70.83%, specificity of 79.55%, positive predictive value (PPV) of 65.38%, negative predictive value (NPV) of 83.3%, and diagnostic accuracy (DA) of 76.47%.

This study has shown that the combination of FibroScan and AST/ALT, APRI, and FIB-4 methods provides a valuable approach for assessing liver fibrosis in NAFLD patients. This can eliminate the need for LB in patients without clear indication.

*Funding: No funding sources*
*Conflict of interest: None declared*
*Ethical approval: The study was approved by the Institutional Ethics Committee*

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**Cite this article as:** Agrawal A, Malhotra Y, Patil M, Lakra DP, Toppo A. Assessing the relationship between FibroScan and laboratory parameters in evaluating hepatic fibrosis in patients with non-alcoholic fatty liver disease. Int J Res Med Sci 2024;12:4115-21.