# The Accuracy of MAFLD Fibrosis Score in non-Invasive Assessment of Liver Fibrosis and Fibrotic NASH in MAFLD Egyptian Patients

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**Background and study aim:** Fibrotic non-alcoholic steatohepatitis (NASH), a clinically significant form of metabolic dysfunction-associated fatty liver disease (MAFLD) characterized by liver fibrosis, requires prompt identification to enable early therapeutic intervention. Existing non-invasive scores show limited accuracy across populations, including the Fibrosis-4 index (FIB-4), the aspartate aminotransferase-to-platelet ratio index (APRI), and the non-alcoholic fatty liver disease (NAFLD) Fibrosis Score (NFS), compared to the MAFLD Fibrosis Score (MFS). This research sought to determine how well the MFS could diagnose significant fibrosis and fibrotic NASH. **Patients and Methods:** a total of 382 Egyptian patients diagnosed with NAFLD were included in this cross-sectional analysis. The fibroscan-aspartate aminotransferase (FAST) score and liver stiffness measurement (LSM) were used to categorize the participants; fibrotic NASH was regarded as having a threshold of ≥0.67. Clinical parameters and fibrosis scores were calculated and compared. **Results:** Significant fibrosis (≥F2) was found in 19.6% of cases, while fibrotic NASH was present in 6.02%. MFS had the greatest AUC (0.737) for significant fibrosis and at a threshold of >13.75 it predicted significant fibrosis with a sensitivity of 66.7% and a specificity of 67.75%. For fibrotic NASH, MFS showed a high specificity (94.4%) and positive predictive value (90.8%) at a cutoff of >15.98, ranking third after APRI and FIB-4.**Conclusion:** MFS is a promising non-intrusive model to detect significant fibrosis and fibrotic NASH and may enhance early diagnosis when combined with other markers.

## INTRODUCTION

After NAFLD was replaced in June 2023, metabolic-associated fatty liver disease (MAFLD) was officially created. The condition is defined by hepatic steatosis and at least two metabolic risk diseases, such as type 2 diabetes mellitus (T2DM), obesity, or average weight with two or more of metabolic risk problems. [1] The prevalence of MAFLD in Egypt is estimated at approximately 47.5%, with 56.7% showing fibrosis, as opposed to a frequency of over 30% worldwide. [2] European and American liver organizations -EASL and AASLD- advise against routine community screenings for MAFLD and diabetes, despite the fact that these conditions are on the rise and that MAFLD is quite common. [3,4].

Twenty percent of those with MAFLD will also develop non-alcoholic steatohepatitis (NASH), a severe inflammatory form of the disease that is associated with advanced cirrhosis, liver cancer, or fibrosis of the liver.

Histological classification defines stage F2 and F3 as significant and advanced fibrosis, while stage F4 indicates cirrhosis. Moreover, NASH patients are categorized as having early NASH (F0–F1 fibrosis), fibrotic NASH (≥F2 fibrosis), or NASH-cirrhosis (F4 fibrosis). [5]

Since most MAFLD patients are asymptomatic, the development of efficient diagnostic tools is crucial to detect fatty liver early, delay the course of the disease, and gain from prospective pharmaceutical therapies in the future. [6] Detection of hepatic steatosis should lead to further fibrosis assessment with non-intrusive techniques. Liver biopsy is still the most reliable method for determining fibrosis, however because of its intrusiveness and related risks, less invasive techniques have been explored. [7]

Three fibrosis scores—NFS, aspartate aminotransferase-to-platelet ratio index (APRI), and fibrosis-4 index (FIB-4)—have been the focus of many laboratory studies. They are still not very sensitive in identifying steatosis and early fibrosis (>F2) in asymptomatic people. Moreover, when it comes to people under 35, the FIB-4 and NFS show low performance. FIB-4 may lead to overdiagnosis or false negatives, especially in non-diabetics, while APRI has shown inconsistent performance in predicting advanced fibrosis in MASH cases. [8] Although more accurate models such as Hepascore and FibroTest exist, their dependence on specialized biomarkers limits their widespread use. [9]

The fibroScan-AST (FAST) score identified NASH patients with a NAFLD Activity Score (NAS) ≥4 and severe fibrosis (≥F2) with an area under the receiver operating characteristic curve (AUROC) ranging from 0.74 to 0.95. The score was first introduced by Newsome et al. in an English cohort of NAFLD patients and has now been validated in many international cohorts. [10]

Recently, a novel composite score, the MAFLD Fibrosis Score (MFS) was introduced, incorporating seven factors (age, gamma-glutamyl transferase (GGT), body mass index (BMI), international normalized ratio (INR), type 2 diabetes history, aspartate aminotransferase (AST), and platelet number). [11]

The focus of this study was to assess MFS's diagnostic effectiveness in identifying fibrotic NASH (FAST score ≥0.67) and significant liver fibrosis (≥F2 fibrosis) in Egyptian MAFLD patients in contrast to conventional fibrosis indices (APRI, FIB-4, and NFS).

## PATIENTS AND METHODS

### 1.2 Study Design and Patients Selection

382 Egyptian patients aged 18 years or older with NAFLD participated in this cross-sectional research, recruited from the outpatient clinic of Tropical Medicine Department, Tanta University, between March 2024 and March 2025. A flow diagram of patient recruitment and exclusions is provided in Figure 1.

The American Association of Clinical Endocrinology Guidelines (AACE 2022) were followed in the diagnosis of NAFLD: [1] When using fibroscan to calculate the liver stiffness (LSM) and controlled attenuation parameter (CAP) for the diagnosis of steatosis, an LSM value of 6.5 kPa omitted significant fibrosis (F≥2) with a NPV of 91%, while 12.1 kPa omitted cirrhosis (F4) with a NPV of 99%, all at an unchanged sensitivity. [2] Secondary factors such as corticosteroid or methotrexate use, a history of malignancy within two years, and other etiologies of chronic liver illnesses, including hepatitis C and B infections and autoimmune hepatitis, were excluded by performing hepatitis viral markers [hepatitis C virus antibody (HCV Ab), hepatitis B surface antigen (HBsAg), hepatitis B core antibody (HBcAb)], antinuclear antibody (ANA), and total immunoglobulin G (IgG); and [3] No more than 30 grams of alcohol per day for men and 20 grams per day for women. [12]

### 2.2 **Clinical and laboratory parameters**

Every patient had detailed medical history taking, assessment of co-morbidities, presence or absence of T2DM, hypertension and clinical examination. Anthropometric measurements were taken, in the form of BMI and waist circumference. Abdominal and pelvic ultrasounds were performed to assess liver status.

Laboratory investigations were performed within two weeks of LSM and CAP measurements. These included alanine aminotransferase (ALT), AST, GGT, total bilirubin, albumin, complete blood count (CBC), and INR. Components of lipid profile including total cholesterol, triglycerides, high-density lipoprotein (HDL), low-density lipoprotein (LDL), and very low-density lipoprotein (VLDL), along with glycated

hemoglobin (HbA1c) levels, were also assessed.

**2.3Transient Elastography (Fibroscan)**

Skilled operators used fibroScan 502 (Echosens, France) to get LSM and CAP. Prior to the examination, patients fasted for no less than three hours. We permitted readings with an interquartile range (IQR) of under thirty percent of the median, a success rate exceeding 60%, and ≥10 valid observations. Ultrasound attenuation at 3•5 MHz is estimated on average by CAP, which is expressed in dB/m, and LSM, which is expressed in kPa. Based on the BMI thresholds, M and XL probes were employed (M probe: <30 kg/m2, XL probe: ≥30 kg/m2). [13]

According to several studies, the median CAP of 288 dB/m was used to characterize steatosis, which was then categorized as either missing (S0: < 248 dB/m), mild (S1: 248 to 268 dB/m), moderate (S2: 268 to 280 dB/m), or severe (S3: > 280 dB/m). LSM threshold of 7.6 Kpa was employed to designate significant fibrosis (F ≥ 2), while a threshold of 8.8 kPa indicated advanced fibrosis (F ≥ 3). [14]

2.4 .**Calculation of Score Values**

To determine the FAST score, the following equation was applied: [e (- 1.65+1.07 x In (LSM) + 2.66*10-8 x CAP3 -63.3 x AST-1)] / [1+e (-1.65 + 1.07 × In (LSM) + 2.66*10-8 x CAP3 -63.3 x AST-1)] as originally proposed by Newsome et al. [10] The NFS was determined using the equation: –1.675 + 0.037 – age (years) + 0.094 – BMI (kg/m2) + 1.13 × IFG/diabetes (yes = 1, no = 0) + 0.99 × AST/ALT ratio – 0.013 × platelet count (×109/l) – 0.66 × albumin (g/dl). [15] The subsequent equation was applied to estimate the FIB-4 score: Age (years)×AST(U/L)/ [platelet count (109/L) ×ALT1/2 (U/L)]. [16]

The APRI was determined as: [(AST level/ Upper Limit of Normal)/platelet count (109/l)] ×100. [17] MFS was computed as follows: 0.078 × Age(year)−0.007363 × Platelet count (109/L) +0.0146× AST(U/L) + 0.007618 × GG T(U/L)+ 6.673 × INR + 0.09833 × BMI(kg/m2) + 1.425 × T2DM (yes = 1, no = 0). [11]

**2.5 Patient Grouping**

Based on fibrosis staging by fibroscan, patients were grouped as follows (Table 2:(

Group Ia: included 307 (80.4 %) patients without significant fibrosis (F0-F1 .(

Group IIa: included 75 (19.6 %) patients with significant to advanced fibrosis (≥ F2 .(

According to the FAST score results our 382 patients were split into two groups (Table 4:(

Group Ib: involved 359 (93.98 %) patients without fibrotic NASH (FAST score <0.67 .(

Group IIb: included 23 (6.02%) patients with fibrotic NASH (FAST ≥ 0.67 .(

These groupings were used for subsequent statistical comparisons.

**2.6 Statistical analysis**

IBM-SPSS software, version 20.0 (Armonk, NY: IBM Corp.), was implemented to input and analyze all of the data that was gathered. Counts and percentages were used to represent the categorical variables. Using the Chi-square test, relationships between categorical variables were assessed. Normality was evaluated for continuous data using the Shapiro-Wilk and Kolmogorov-Smirnov tests. The mean ± standard deviation and median were used to display variables with a normal distribution, and the student's t-test was used to compare two groups. This was replaced by the Mann-Whitney U test for data that was not normally distributed.

The effectiveness of MFS, FIB-4, APRI, and NFS scores in identifying both significant fibrosis and fibrotic NASH (FAST score ≥0.67) was checked utilizing Receiver Operating Characteristic (ROC) curve analysis. The best cutoff point was selected based on the value yielding the highest sensitivity and specificity. To compare the differences between the studied scores' AUROC, the DeLong's test was used .

## RESULTS

**Features of the participants**

452 individuals were first assessed at the Tropical Medicine outpatient clinic of Tanta Faculty of Medicine. Out of these, 36 participants were excluded due to unreliable elastography readings and 34 due to missing diagnostic or scoring data. Ultimately, the analysis compromised 382 patients, recruited

between March 2024 and March 2025 (Figure 1 .(

Table 1 outlines demographic, laboratory, elastographic, and non-invasive fibrosis score characteristics for the study cohort. The average age was 48.6 ± 12 years, with 49% (188/382) of the participants were males and the mean BMI was 33.3 ± 5.65. T2DM and hypertension were found in 119 (31.15%) and 115 (30.1 %) patients respectively. Among our 382 patients, 307 (80.3 %) were F0-F1, 17 (4.5%) were F2, 28 (7.3%) were F3 and 30 (7.9 %) were F4.

Table 2 compares clinical and biochemical data across group Ia and group IIa. Among the MFS parameters, age, platelet count, AST, GGT, and INR, all differed significantly ($p < 0.05$) while T2DM ($p = 0.117$) and BMI ($p = 0.92$) did not. Nevertheless, waist circumference was substantially greater in group IIa ($p = 0.008$). Additionally, LSM, ALT, total bilirubin, and LDL were significantly elevated among patients with significant to advanced fibrosis (≥ F2) (group IIa). Conversely, albumin, and HDL showed a highly significant decrease among group IIa patients (P<0.001.(

Regarding the non-invasive fibrosis scores results, FIB4, FAST, and APRI score were significantly increased among patients with significant and advanced fibrosis with median (IQR) of 1.4 (1.14), 0.53 (0.4), and 0.38 (0.37) respectively (P<0.001). NFS and MFS scores mean of -0.55 ± 1.36 and 14.69 ± 1.76 respectively were also notably elevated in group IIa as opposed to group Ia (P<0.001) (Table 2 .(

Diagnostic performance for different scores to recognize patients with significant and advanced fibrosis (≥ F2) (Table 3 & FIG 2(

The MFS's diagnostic efficiency was investigated at a threshold value of >13.75, yielding in a 66.7% sensitivity, 67.75% specificity, 65.16% PPV, 69.2% NPV, and 67.07% accuracy in total. Among all evaluated scores, the MFS achieved the greatest area under ROC curve (AUROC), recorded at 0.737 [95% confidence interval (CI): 0.673–0.801.[

After that, the FIB-4 score (AUROC: 0.678, 95% CI: 0.607–0.750), APRI (AUROC: 0.669, 95% CI: 0.604–0.740), and NFS (AUROC: 0.652, 95% CI: 0.599–0.739) came next. According to DeLong's statistical test, the AUROC of MFS was substantially greater than both FIB-4 ($p = 0.013$) and NFS ($p < 0.001$.(

A composite index that takes into account AST, CAP, and LSM levels is the FAST score. With a claimed specificity of 90%, a FAST score of ≥0.67 is regarded as suggestive of biopsy-confirmed fibrotic NASH, which is determined by a NAFLD activity score (NAS) ≥4 coupled with fibrosis stage ≥2. [10] Accordingly, our study sought to investigate how well various non-intrusive scores in identifying patients at this critical stage. Table 4 contrasts clinical, laboratory, fibroscan and non-invasive fibrosis scores results between group Ib and group IIb. Again, when considering the MFS parameters Platelet count, AST, GGT, INR, and history of type 2 diabetes all revealed statistically noteworthy variations between the two groups. ($p < 0.001$) with the exception of the age ($p = 0.80$) and BMI ($p = 0.447$ .(

Additionally, ALT, total bilirubin, LDL and HbA1c were significantly higher in group IIb. Conversely, hemoglobin, and albumin showed a highly significant decrease among group IIb patients (P<0.05.(

Moreover, the results revealed a highly significant increase in LSM and CAP among patients with fibrotic NASH (group IIb). The median (IQR) LSM, among fibrotic NASH group was 13.8 (11.4) kPa among them 21/23 (91.3%) patients with ≥ F2 compared to a median (IQR) of 5 (1.9) kPa among them 305/359 (85%) patients with F0-F1 in the other group (Table 4.(

In regard to the non-intrusive fibrosis scores results, FIB4 and NFS scores were significantly increased in patients with fibrotic NASH with mean of 2.22 ± 085 and 0.067 ± 1.33 in contrast to the other group (P<0.001). The APRI score median (IQR) of 0.75 (0.29) was also notably elevated in group IIb in comparison to group Ib (P<0.001). The MFS score shows significant increase in group IIb over the other group (P<0.001) with mean of 15.68 ± 1.72 (Table 4 .(

Diagnostic performance for different scores to discriminate patients with fibrotic NASH (Table 5 & FIG 3(

The MFS score's diagnostic efficacy had 60.87% sensitivity, 94.4% specificity, 90.8% PPV, 72.7% NPV, and 78.49% overall accuracy, when a cutoff point was set at >15.98.


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The greatest AUROC was 0.938 [95% CI, 0.910 – 0.965] for the APRI score, then 0.876 [95% CI, 0.799-0.952] for the FIB 4, 0.839 [95% CI, 0.753 – 0.924] for the MFS score, and 0.766 [95% CI, 0.653 – 0.880] for the NFS score. According to DeLong's test APRI's AUROC was notably greater than that of the MFS (p = 0.04.(

Logistic Regression and ROC Analysis for MFS, APRI, FIB-4, NFS, and Combined Models for detection of significant fibrosis and fibrotic NASH (Table 6(

Logistic regression analysis demonstrated that MFS was the best performing single score for detecting significant fibrosis, with the highest accuracy (82.5%) and AUC (0.737), along with relatively higher sensitivity (22.7%) while maintaining excellent specificity (97.1%). FIB-4 and APRI showed fair discriminatory power (AUC = 0.678 and 0.669, respectively) but had very low sensitivity despite high specificity (>99%). NFS achieved the weakest performance (AUC = 0.652) with negligible sensitivity. Notably, the combined MFS + NFS model improved discrimination (AUC = 0.759, $R^2$ = 0.222) and provided a more balanced sensitivity (21.3%) and specificity (96.7%) compared with either score alone.

In predicting fibrotic NASH, APRI achieved the best single-score performance with the highest AUC (0.938) and explanatory power ($R^2$ = 0.449), coupled with very high specificity (99.2%) but modest sensitivity (21.7%). FIB-4 showed good discrimination (AUC = 0.876) and slightly better sensitivity (26.1%), while MFS alone had only moderate accuracy (AUC = 0.839, sensitivity 13%). NFS was the weakest predictor (AUC = 0.766) with essentially no sensitivity. The combined MFS + APRI model yielded the strongest overall performance, with excellent discrimination (AUC = 0.955, $R^2$ = 0.519) and improved sensitivity (26.1%) without compromising specificity (98.6%).

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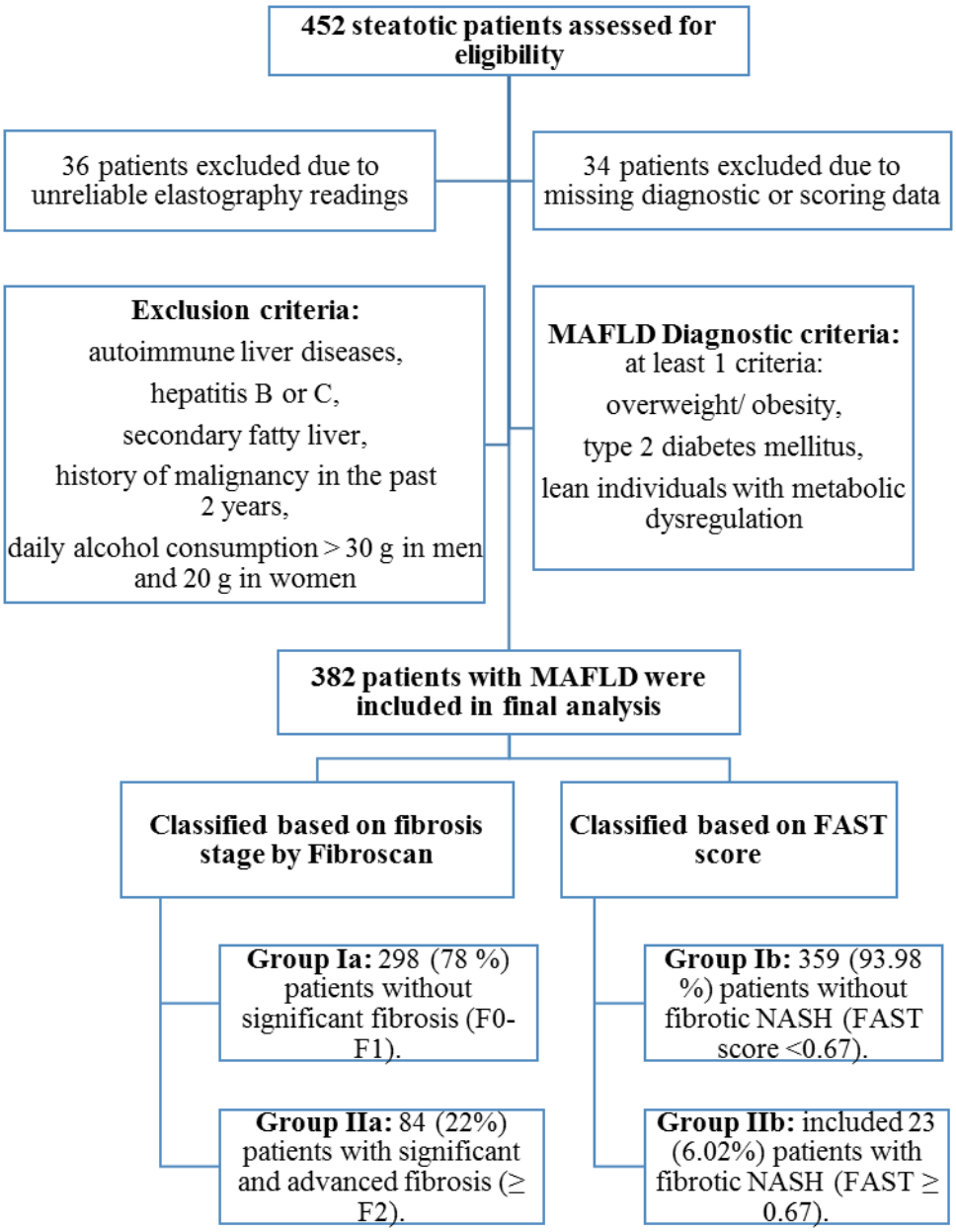


**Figure (1): Flowchart of patient selection and categorization based on Fibroscan and FAST score results.**
FAST: Fibroscan-AST, MAFLD: metabolic-associated fatty liver disease, NASH: non-alcoholic steatohepatitis.

**Table (1): Characteristics of MAFLD patients included in the study:**

| Parameter | All Cases (n = 382) |
|---|---|
| **Age (years)** | 48.62 ± 11.99 |
| **Sex, n (%)**<br>Male | <br>188 (49.21) |
| **T2DM, n (%)** | 119 (31.15) |
| **HTN, n (%)** | 115 (30.1) |
| **BMI (kg/m$^2$)** | 33.27 ± 5.65 |
| **Mid arm circumference (cm)** | 34.95 ± 3.03 |
| **Waist Circumference (cm)** | 112.24 ± 12.04 |
| **Hip Circumference (cm)** | 120.55 ± 12.14 |
| **LSM (kPa)** | 6.56 ± 4.38 |
| **Fibrosis Stage, n (%)**<br>0<br>1<br>2<br>3<br>4 | <br>258 (67.5)<br>49 (12.8)<br>17 (4.5)<br>28 (7.3)<br>30 (7.9) |
| **CAP (dB/m$^2$)** | 301.63 ± 4.84 |
| **Steatosis stage, n (%)**<br>1<br>2<br>3 | <br>118 (30.9)<br>29 (7.6)<br>235 (61.5) |
| **Hemoglobin (g/dl)** | 12.95 ± 1.54 |
| **Platelets × 10$^3$ /(mm$^3$)** | 222.97 ± 51.53 |
| **WBCs × 10$^3$ /(mm$^3$)** | 6.99 ± 1.99 |
| **T. Bilirubin (mg/dl)** | 0.8 ± 0.22 |
| **Albumin (gm/dl)** | 4.17 ± 0.37 |
| **ALT (U/L)** | 38.12 ± 24.01 |
| **AST (U/L)** | 32.78 ± 21.25 |
| **GGT (U/L)** | 42.07 ± 27.65 |
| **INR** | 1.02 ± 0.06 |
| **Cholesterol (mg/dl)** | 204.02 ± 37.58 |
| **Triglycerides (mg/dl)** | 152.29 ± 48.22 |
| **LDL (mg/dl)** | 121.15 ± 22.08 |
| **HDL (mg/dl)** | 46.41 ± 9.18 |
| **VLDL (mg/dl)** | 31.14 ± 13.38 |
| **Creatinine (mg/dl)** | 0.81 ± 0.16 |
| **HbA1c (%)** | 6.02 ± 1.22 |
| **FAST score** | 0.25 ± 0.21 |
| **NAFLD fibrosis score** | -1.17 ± 1.33 |
| **FIB4** | 1.2 ± 0.61 |
| **APRI score** | 0.35 ± 0.23 |
| **MAFLD fibrosis score** | 13.48 ± 1.69 |

Values were expressed as n (%), mean ± SD, or median (interquartile range). T2DM: type 2 diabetes, HTN: hypertension, BMI: body mass index, LSM: liver stiffness measurements, CAP: controlled attenuated parameter, WBC: white blood cells, ALT: alanine aminotransferase, AST: aspartate aminotransferase, GGT: gamma-glutamyl transferase, INR: international normalized ratio, LDL: low density lipoprotein, HDL: high density lipoprotein, VLDL: very low density lipoprotein, HbA1c: hemoglobin A1c, FAST: Fibroscan-AST, Fib-4: fibrosis-4, APRI: aspartate aminotransferase-to-platelet ratio index.

**Table (2): Comparison between the two studied groups (Ia & IIa) according to clinical, anthropometric, laboratory and non-invasive fibrosis scores**

| Parameter | Fibrosis stage | | Test of sig. | P-value |
|---|---|---|---|---|
| | Group Ia (F0-F1) N=307 (80.4%) | Group IIa (F2-F3-F4) N=75 (19.6%) | | |
| **Age (years)** | 47 (18) | 53 (20) | U = -2.641 | 0.008* |
| **Sex, n (%)** Male | 147 (47.9) | 41 (54.7) | $\chi2$ = 1.11 | 0.292 |
| **T2DM, n (%)** | 90 (29.3) | 29 (38.7) | $\chi2$ = 2.457 | 0.117 |
| **HTN, n (%)** | 81 (26.4) | 34 (45.3) | $\chi2$ = 10.285 | 0.001* |
| **BMI (kg/m$^2$)** | 33.3 (7.3) | 32.8 (6.4) | U = -0.100 | 0.92 |
| **Mid arm circumference (cm)** | 35 (3) | 35 (3) | U = -0.646 | 0.518 |
| **Waist Circumference (cm)** | 111 (15) | 113 (11) | U = -2.648 | 0.008* |
| **Hip Circumference (cm)** | 120.62 ± 12.73 | 120.27 ± 9.43 | T = -0.223 | 0.824 |
| **LSM (kPa)** | 4.8 (1.5) | 11.5 (6) | U = -13.433 | <0.001* |
| **CAP (dB/m$^2$)** | 293 (61) | 318 (86) | U = -1.074 | 0.283 |
| **Steatosis stage, n (%)**<br>1<br>2<br>3 | <br>91<br>22<br>194 | <br>27<br>7<br>41 | $\chi2$ = 1.874 | 0.392 |
| **Hemoglobin (g/dl)** | 12.8 (2.4) | 12.6 (2.2) | U = -1.742 | 0.082 |
| **Platelets × 10$^3$ /(mm$^3$)** | 220 (73) | 184 (96) | U = -3.629 | <0.001* |
| **WBCs × 10$^3$ /(mm$^3$)** | 7 (2.6) | 6.3 (3) | U = -1.471 | 0.141 |
| **T. Bilirubin (mg/dl)** | 0.8 (0.2) | 0.9 (0.3) | U = -4.404 | <0.001* |
| **Albumin (gm/dl)** | 4.2 (0.6) | 3.8 (0.7) | U = -5.074 | <0.001* |
| **ALT (U/L)** | 28 (19.5) | 36 (28) | U = -3.344 | 0.001* |
| **AST (U/L)** | 26 (17) | 38 (23) | U = -3.889 | <0.001* |
| **GGT (U/L)** | 28 (25) | 54 (37) | U = -7.581 | <0.001* |
| **INR** | 1 (0) | 1.1 (0.12) | U = -15.736 | <0.001* |
| **Cholesterol (mg/dl)** | 198 (47) | 203 (45) | U = -1.076 | 0.282 |
| **Triglycerides (mg/dl)** | 146 (41) | 146 (54.8) | U = -0.712 | 0.476 |
| **LDL (mg/dl)** | 120 (33) | 126 (29) | U = -2.898 | 0.004* |
| **HDL (mg/dl)** | 46.88 ± 9.54 | 44.48 ± 7.29 | T = 2.034 | 0.043* |
| **VLDL (mg/dl)** | 28 (13) | 27 (15) | U = -0.884 | 0.377 |
| **Creatinine (mg/dl)** | 0.8 (0.2) | 0.8 (0.2) | U = -1.213 | 0.225 |
| **HbA1c (%)** | 5.5 (1.2) | 5.6 (1.6) | U = -1.452 | 0.146 |
| **FIB4** | 1.02 (0.65) | 1.4 (1.14) | U = -4.793 | <0.001* |
| **FAST score** | 0.14 (0.23) | 0.53 (0.4) | U = -8.180 | <0.001* |
| **NAFLD fibrosis score** | -1.32 ± 1.28 | -0.55 ± 1.36 | T = -4.658 | <0.001* |
| **APRI score** | 0.27 (0.21) | 0.38 (0.37) | U = -4.547 | <0.001* |
| **MAFLD fibrosis score** | 13.18 ± 1.53 | 14.69 ± 1.76 | T = -6.839 | <0.001* |

Values were expressed as n (%), mean ± SD, or median (interquartile range). U: Mann-Whitney test, $\chi^2$: chi square test, T: student t-test. P value considered significant > 0.05. T2DM: type 2 diabetes, HTN: hypertension, BMI: body mass index, LSM: liver stiffness measurements, CAP: controlled attenuated parameter, WBC: white blood cells, ALT: alanine aminotransferase, AST: aspartate aminotransferase, GGT: gamma-glutamyl transferase, INR: international normalized ratio, LDL: low density lipoprotein, HDL: high density lipoprotein, VLDL: very low density lipoprotein, HbA1c: hemoglobin A1c, FAST: Fibroscan-AST, Fib-4: fibrosis-4, APRI: aspartate aminotransferase-to-platelet ratio index.

**Table (3): Diagnostic performance for different scores to discriminate patients with significant and advanced fibrosis (≥ F2) (n = 75) from patients without significant fibrosis (F0-F1) (n = 307) according to fibroscan:**

| ROC curve between High (F2-F3-F4) and Low (F0-F1) | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | **AUC** | **95% CI** | **Cutoff** | **Sensitivity** | **Specificity** | **PPV** | **NPV** | **Accuracy** |
| **MFS score** | 0.737 | 0.673 – 0.801 | >13.75 | 66.67 | 67.75 | 65.16 | 69.2 | 67.07 |
| **FIB-4** | 0.678 | 0.607 – 0.750 | >1.11 | 73.33 | 56.68 | 60.5 | 70.1 | 64.42 |
| **APRI score** | 0.669 | 0.604 – 0.740 | >0.46 | 48 | 84.69 | 73.9 | 64.3 | 67.26 |
| **NFS score** | 0.652 | 0.599 – 0.739 | >-1.7 | 80 | 43.97 | 56.4 | 70.8 | 60.74 |

ROC: receiver operator characteristic, AUC: area under the curve, CI: confidence interval, PPV: positive predictive value, NPV: negative predictive value, MFS: MAFLD fibrosis score, APRI: aspartate aminotransferase-to-platelet ratio index, FIB-4: fibrosis-4, NFS: NAFLD fibrosis score.

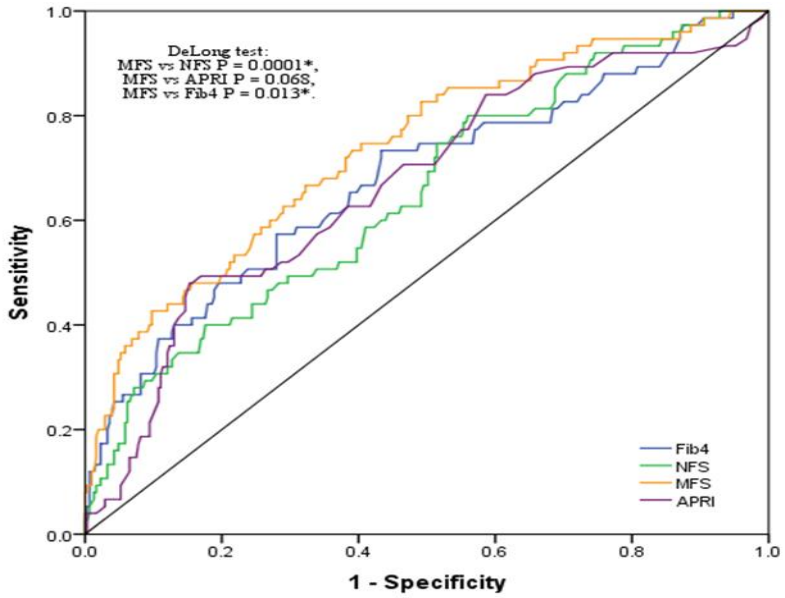


**Figure (2): ROC curve for different markers to discriminate patients with significant fibrosis (n = 75) from patients without significant fibrosis (n = 307).**

**Table (4): Comparison between the two studied groups (Ib & IIb) according to clinical, anthropometric, laboratory and non-invasive fibrosis scores**

| Parameter | FAST score | | Test of sig. | P-value |
|---|---|---|---|---|
| | Group Ib (Non-Fibrotic NASH) n= (359) (93.98 %) | Group IIb (Fibrotic NASH) n= (23) (6.02%) | | |
| **Age (years)** | 48.65 ± 11.86 | 48 ± 14.15 | 0.253 | 0.800 |
| **Sex, n (%)** Male | 175 (48.7) | 13 (56.5) | $\chi2$ = 0.523 | 0.47 |
| **T2DM, n (%)** | 106 (29.53) | 13 (56.52) | $\chi2$ =7.344 | 0.01* |
| **HTN, n (%)** | 104 (28.97) | 11 (47.83) | $\chi2$ = 3.653 | 0.063 |
| **BMI (kg/m$^2$)** | 33.33 ± 5.72 | 32.4 ± 4.51 | 0.762 | 0.447 |
| **Mid arm circumference (cm)** | 34.93 ± 3.08 | 35.33 ± 2.25 | -0.606 | 0.545 |
| **Waist Circumference (cm)** | 112.07 ± 12.29 | 114.83 ± 6.76 | -1.773 | 0.086 |
| **Hip Circumference (cm)** | 120.58 ± 12.36 | 119.96 ± 8.23 | U = -0.334 | 0.739 |
| **LSM (kPa)** | 5 (1.9) | 13.8 (11.4) | U = -7.272 | <0.001* |
| **Fibrosis Stage, n (%)**<br>0<br>1<br>2<br>3<br>4 | <br>257 (71.6)<br>48 (13.4)<br>16 (4.5)<br>24 (6.7)<br>14 (3.9) | <br>1 (4.35)<br>1 (4.35)<br>1 (4.35)<br>4 (17.39)<br>16 (69.6) | $\chi2$ = 137.901 | <0.001* |
| **CAP (dB/m$^2$)** | 292 (65) | 346 (95) | U = -3.331 | <0.001* |
| **Steatosis stage, n (%)**<br>1<br>2<br>3 | <br>114 (31.8)<br>27 (7.5)<br>218 (60.7) | <br>4 (17.4)<br>2 (8.7)<br>17 (73.9) | $\chi2$ = 2.094 | 0.351 |
| **Hemoglobin (g/dl)** | 12.8 (2.4) | 11.8 (1.4) | U = -2.059 | 0.04* |
| **Platelets × 10$^3$ /(mm$^3$)** | 218 (74) | 158 (41) | U = -4.954 | <0.001* |
| **WBCs × 10$^3$ /(mm$^3$)** | 6.8 (2.6) | 6.3 (4.2) | U = -1.280 | 0.200 |
| **T. Bilirubin (mg/dl)** | 0.79 ± 0.22 | 0.95 ± 0.21 | T = -3.47 | 0.001* |
| **Albumin (gm/dl)** | 4.19 ± 0.36 | 3.72 ± 0.36 | T = 6.178 | <0.001* |
| **ALT (U/L)** | 29 (19) | 51 (32) | U = -4.924 | <0.001* |
| **AST (U/L)** | 26 (18) | 56 (33) | U = -6.298 | <0.001* |
| **GGT (U/L)** | 32 (26) | 76 (41) | U = -6.343 | <0.001* |
| **INR** | 1.01 ± 0.04 | 1.11 ± 0.08 | T = -6.198 | <0.001* |
| **Cholesterol (mg/dl)** | 203.48 ± 36.98 | 212.39 ± 46.06 | T = -1.102 | 0.271 |
| **Triglycerides (mg/dl)** | 152.34 ± 48.87 | 151.57 ± 37.34 | T = -0.073 | 0.941 |
| **LDL (mg/dl)** | 120.49 ± 21.99 | 131.43 ± 21.22 | T = -2.318 | 0.021* |
| **HDL (mg/dl)** | 46.59 ± 9.23 | 43.43 ± 7.99 | T = 1.604 | 0.11 |
| **VLDL (mg/dl)** | 31 ± 13.45 | 33.22 ± 12.36 | T = -0.769 | 0.443 |
| **Creatinine (mg/dl)** | 0.8 (0.2) | 0.7 (0.2) | U = -1.221 | 0.222 |
| **HbA1c (%)** | 5.5 (1.3) | 6.5 (2.1) | U = -2.394 | 0.017* |
| **FIB4** | 1.14 ± 0.53 | 2.22 ± 085 | T = -6.070 | <0.001* |
| **NAFLD fibrosis score** | -1.25 ± 1.29 | 0.067 ± 1.33 | T = -4.739 | <0.001* |
| **APRI score** | 0.27 (0.2) | 0.75 (0.29) | U = -7.039 | <0.001* |
| **MAFLD fibrosis score** | 13.34 ± 1.58 | 15.68 ± 1.72 | T = -6.840 | <0.001* |

Values were expressed as n (%), mean ± SD, or median (interquartile range). U: Mann-Whitney test, $\chi^2$: chi square test, T: student t-test, P value considered significant > 0.05. T2DM: type 2 diabetes, HTN: hypertension, BMI: body mass index, LSM: liver stiffness measurements, CAP: controlled attenuated parameter, WBC: white blood cells, ALT: alanine aminotransferase, AST: aspartate aminotransferase, GGT: gamma-glutamyl transferase, INR: international normalized ratio, LDL: low density lipoprotein, HDL: high density lipoprotein, VLDL: very low density lipoprotein, HbA1c: hemoglobin A1c, FAST: Fibroscan-AST, Fib-4: fibrosis-4, APRI: aspartate aminotransferase-to-platelet ratio index.

**Table (5): Diagnostic performance for different scores to discriminate patients with Fibrotic NASH (n = 23) from patients without Fibrotic NASH (n = 359) according to FAST score:**

| ROC curve between Fibrotic NASH and Non-Fibrotic NASH | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | **AUC** | **95% CI** | **Cutoff** | **Sensitivity** | **Specificity** | **PPV** | **NPV** | **Accuracy** |
| **APRI score** | 0.938 | 0.910 – 0.965 | > 0.44 | 100 | 81.6 | 83.1 | 100 | 89.32 |
| **FIB-4** | 0.876 | 0.799 – 0.952 | > 1.45 | 91.3 | 78.3 | 79.18 | 90.87 | 84.46 |
| **MFS score** | 0.839 | 0.753 – 0.924 | > 15.98 | 60.87 | 94.4 | 90.8 | 72.7 | 78.49 |
| **NFS score** | 0.766 | 0.653 – 0.880 | > -0.06 | 65.2 | 80.8 | 75.43 | 71.96 | 73.39 |

ROC: receiver operator characteristic, AUC: area under the curve, CI: confidence interval, PPV: positive predictive value, NPV: negative predictive value, MFS: MAFLD fibrosis score, APRI: aspartate aminotransferase-to-platelet ratio index, FIB-4: fibrosis-4, NFS: NAFLD fibrosis score.

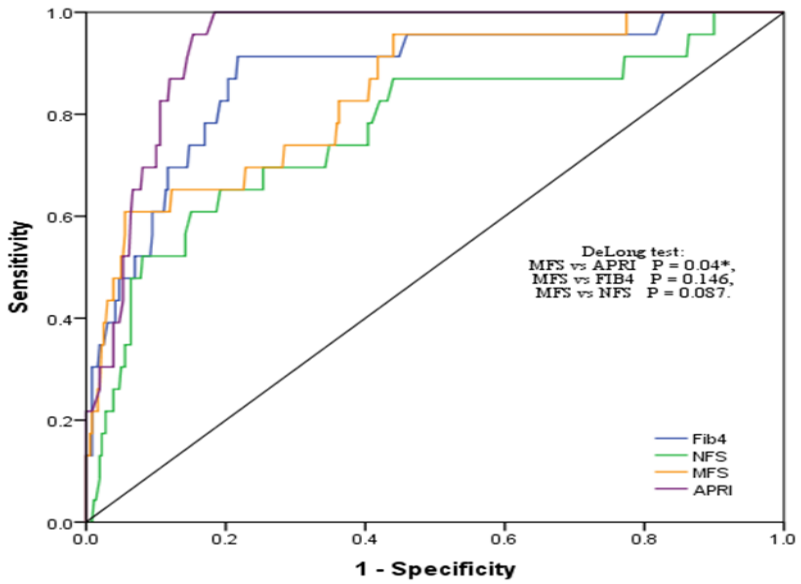


**Figure (3): ROC curve for different markers to discriminate patients with Fibrotic NASH (n = 23) from patients without Fibrotic NASH (n = 359) according to FAST score.**

**Table (6): Logistic Regression and ROC Analysis for MFS, APRI, FIB-4, NFS, and Combined Models for detection of significant fibrosis and fibrotic NASH.**

| Model / Predictor | B (SE) | p-value | OR (95% CI) | -2 Log Likelihood | Nagelkerke $R^2$ | Accuracy (%) | Sensitivity (%) | Specificity (%) | AUC (95% CI) |
|---|---|---|---|---|---|---|---|---|---|
| **Significant Fibrosis** | | | | | | | | | |
| **Combined (MFS + NFS)** | 0.944 (0.170) | <0.001* | 2.569 (1.84 – 3.587) | 321.075 | 0.222 | 81.9 | 21.3 | 96.7 | 0.759 (0.695 – 0.824) |
| | -0.534 (0.202) | 0.008* | 0.586 (0.394 – 0.872) | | | | | | |
| **MFS** | 0.577 (0.089) | <0.001* | 1.78 (1.495 –2.119) | 328.358 | 0.195 | 82.5 | 22.7 | 97.1 | 0.737 (0.673– 0.801) |
| **FIB-4** | 1.172 (0.215) | <0.001* | 3.229 (2.118 – 4.923) | 345.019 | 0.133 | 81.9 | 12 | 99 | 0.678 (0.607 – 0.750) |
| **APRI** | 2.204 (0.537) | <0.001* | 9.065 (3.164 –25.968) | 360.594 | 0.072 | 80.6 | 2.7 | 99.7 | 0.669 (0.604 – 0.740) |
| **NFS** | 0.456 (0.103) | <0.001* | 1.577 (1.288 – 1.932) | 357.505 | 0.085 | 80.9 | 2.7 | 100 | 0.652 (0.599 – 0.739) |
| **Fibrotic NASH** | | | | | | | | | |
| **Combined (MFS + APRI)** | 0.599 (0.185) | ٠,٠٠١* | 1.821 (1.267 – 2.617) | 93.451 | 0.519 | 94.2 | 26.1 | 98.6 | 0.955 (0.934 – 0.977) |
| | 6.592 (1.31) | <0.001* | 729.234 (55.934 – 9507.324) | | | | | | |
| **APRI** | 7.334 (1.166) | <0.001* | 1530.٩٥٥ (155.9 – 15033.728) | 105.399 | 0.449 | 94.5 | 21.7 | 99.2 | 0.938 (0.910 – 0.965) |
| **FIB-4** | 2.144 (0.358) | <0.001* | 8.533 (4.23 – 17.215) | 123.978 | 0.335 | 94.8 | 26.1 | 99.2 | 0.876 (0.799 – 0.952) |
| **Combined (MFS + FIB-4)** | 0.418 (0.21) | 0.047* | 1.52 (1.006 – 2.295) | 120.019 | 0.359 | 95.3 | 30.4 | 99.4 | 0.874 (0.798 – 0.951) |
| | 1.521 (0.462) | 0.001* | 4.577 (1.849 – 11.331) | | | | | | |
| **MFS** | 0.882 (0.158) | <0.001* | 2.417 (1.773 – 3.294) | 132.12 | 0.283 | 94.5 | 13 | 99.7 | 0.839 (0.753 – 0.924) |
| **NFS** | 0.786 (0.182) | <0.001* | 2.194 (1.537 – 3.133) | 152.28 | 0.150 | 94 | 0 | 100 | 0.766 (0.653 – 0.880) |

ROC: receiver operator characteristic, AUC: area under the curve, CI: confidence interval, OR: odds ratio, MFS: MAFLD fibrosis score, APRI: aspartate aminotransferase-to-platelet ratio index, FIB-4: fibrosis-4, NFS: NAFLD fibrosis score.

## DISCUSSION

Steatosis, ballooning of hepatocytes, lobular inflammation, and fibrosis represent the main histological traits of NASH. [18] In chronic liver illness, significant hepatic fibrosis (≥ F2) is a key prognostic factor that indicates a greater risk of the complications related to the liver, like cirrhosis and hepatocellular cancer. Patients with fibrotic NASH are the primary candidates for antifibrotic pharmacological interventions. [19,20]

As a result, the precise staging of liver fibrosis is essential, preferably through non-intrusive approaches. The FAST score performs well for noninvasively diagnosing fibrotic NASH (significant fibrosis ≥F2 and NAS score ≥4).

[21,22] A recently developed noninvasive score for predicting advanced fibrosis in MAFLD patients is the MFS score. [11] Our study aimed to compare MFS with some fibrosis indices (NFS, FIB-4, APRI) in Egyptian patients to discover those with significant fibrosis (≥F2) and fibrotic NASH.

Our results reinforce that although metabolic syndrome components define MAFLD, they do not contribute equally to fibrotic progression. For instance, circumference of the waist, a surrogate of visceral adiposity, was significantly elevated among patients with significant fibrosis (p = 0.008), while BMI was not significant in either fibrosis (p = 0.92) or fibrotic NASH (p = 0.447). This highlights the stronger role of central obesity over general obesity in driving hepatic fibrosis through lipotoxicity and stellate cell activation. [6] These results align with an Egyptian study showing visceral fat indices outperform BMI in predicting NAFLD [23], while contrasting with an Iraqi study linking BMI to NAFLD prevalence, likely due to differences in fat distribution or population characteristics or the inability of BMI to distinguish between subcutaneous and visceral fat. [24]

Hypertension and fibrosis were associated (p = 0.001), possibly linked to systemic inflammation and endothelial dysfunction. [7] Conversely, type 2 diabetes was not significantly linked with fibrosis (≥F2) (p = 0.117), but was substantially more common in patients with fibrotic NASH (p = 0.01). These results imply that diabetes may exert a stronger impact on the inflammatory and fibrogenic components of NASH than on fibrosis alone. Older age was also substantially linked to fibrosis (p = 0.008), likely due to the cumulative effect of metabolic damage over time. [12]

In our study, several biochemical markers (aminotransferases, GGT, INR, platelets) showed significant associations with fibrosis progression (≥ F2) and fibrotic NASH, reflecting the complex interplay of inflammatory damage, impaired hepatic function, and oxidative stress that underpins the progression of liver fibrosis and fibrotic NASH. In research by Newsome et al., elevated aminotransferases along with decreased platelet counts and increased INR were robustly linked to progressive fibrosis in NAFLD patients. [10] Barb et al. also revealed that, increased GGT was strongly related to fibrotic NASH, further substantiating its role as an indicator of oxidative stress and metabolic dysregulation. [25]

In our study, patients with fibrotic NASH (FAST score ≥ 0.67), both liver LSM and CAP values were markedly higher, with median LSM 13.8 kPa and CAP 346 dB/m, versus 5 kPa and 292 dB/m in the non-fibrotic group. About 91.3% of fibrotic NASH patients had significant fibrosis (≥F2), affirming the link between elevated fibroscan readings and fibrotic NASH. These observations agree with prior reports confirming LSM and CAP as reliable non-invasive markers for identifying at-risk NASH, especially when integrated into composite diagnostic models. [15,26] Currently, the EASL and AASLD both advice the use of subsequent testing, using FIB-4 as the first test and LSM (using the 8 and 12 kPa cut-offs) as the second test, for the non-invasive diagnosis of liver fibrosis in NAFLD patients. [3,4]

In our research, non-intrusive scores (FIB-4, APRI, NFS, and MFS) were notably elevated in significant or advanced fibrosis patients, and in those with fibrotic NASH. MFS showed a higher AUC (0.737) for identifying significant fibrosis than FIB-4, APRI, and NFS (0.678, 0.669, and 0.652, respectively). Our results were aligned with Cheung et al. who reported that MFS showed significantly higher AUC of 0.802 outperforming FIB-4 (0.735), APRI (0.680), and NFS (0.729) in both training and validation datasets. When it came to predicting significant fibrosis (≥F2), MFS performed better than traditional scoring. [11]

With 66.67% sensitivity, 67.75% specificity, 65.16% PPV, 69.2% NPV, and 67.07% accuracy, we established the MFS score cutoff value at 13.75. These results closely resemble Cheung et al.'s suggested cutoffs of 14 and 15 for ruling out and in advanced fibrosis, with a gray area in between that necessitates more fibrosis testing or a liver biopsy. [11]

MFS ranked third for detecting fibrotic NASH (AUC 0.839) following APRI score (0.938) and FIB-4 (0.876). Although MFS did not outperform APRI or FIB-4 in this context, it still demonstrated a high level of diagnostic performance, with excellent specificity (94.4%) and positive predictive value (90.8%) at a cutoff of 15.98. Previous studies have shown more modest diagnostic performances for APRI, FIB-

4, and NFS in detecting fibrotic NASH. For example, Woreta et al., validated FAST score in a North American cohort and reported APRI with an AUC of 0.739, FIB-4 reached 0.730, and NFS scored 0.668 for predicting fibrotic NASH. [27] Similarly, Lee et al. observed that NFS and FIB-4 both underperformed in distinguishing at-risk NASH, with AUCs below 0.75 and emphasized that combining scores could enhance a diagnostic value. [28]

The better diagnostic performance of APRI in our study may be attributed to elevated AST levels in fibrotic NASH patients, which directly influences APRI calculations. However, APRI and FIB-4 have demonstrated variable diagnostic power across populations, with APRI being sensitive to transaminase fluctuations and platelet count, and FIB-4 showing limited accuracy in young individuals or those without diabetes. [8,17]

Our study limitations include small sample size with significant fibrosis, advanced fibrosis, and fibrotic NASH, alongside with lack of liver biopsy references. The validation and generalization of our findings would benefit from a comparable investigation with larger fibrotic NASH cohorts.

## CONCLUSION

In conclusion, our study results reveal that MFS surpasses FIB-4, NFS, and APRI scores as a promising non-intrusive indicator of significant fibrosis (≥ F2) in Egyptian MAFLD patients. To our knowledge, this is the first evaluation of MFS score in predicting fibrotic NASH (FAST score ≥ 0.67), our results highlight MFS as a valuable tool for ruling in fibrotic NASH specially when used in conjunction with other non-invasive markers. Further investigation is recommended to validate these results.

**Author contribution:** We declare that all listed authors have made substantial contributions to all of the following three parts of the manuscript:

-Research design, or acquisition, analysis or interpretation of data؛

-drafting the paper or revising it critically؛

-approving the submitted version.

We also declare that no-one who qualifies for authorship has been excluded from the list of authors.

**Ethical consideration:** Tanta University's Research Ethics Committee granted ethical approval for the study (No. 36264PR614/3/24) and it was filed on clinicaltrials.gov (NCT06492369). Every patient received a unique code number and gave their written, informed permission. The study was conducted in accordance with the principles of the Declaration of Helsinki.

**Declaration of Interest:** The writers disclose that they have no competing interests.

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## HIGHLIGHTS

- This article highlights that the MAFLD Fibrosis Score (MFS) outperforms FIB-4, APRI, and NFS in detecting significant fibrosis (≥F2) in Egyptian patients.
- The study highlights that at cutoff >13.75, MFS achieved AUROC 0.737 with balanced sensitivity and specificity for significant fibrosis.
- This article shows that for fibrotic NASH (FAST ≥0.67), MFS demonstrated excellent specificity (94.4%) and PPV (90.8%).
- The study highlights the complementary role of MFS alongside APRI and FIB-4 in diagnosing fibrotic NASH.

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