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Comparison of Aspartate Aminotransferase Platelet Ratio Index (APRI) to Neutrophil Lymphocyte Ratio (NLR) and Platelet Lymphocyte Ratio (PLR) with Fibroscan as a Non-Invasive Marker for Predicting Liver Fibrosis in Chronic Hepatitis C

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ABSTRACT

Background: Chronic hepatitis C (CHC) is the leading cause of liver fibrosis, cirrhosis, and hepatocellular carcinoma worldwide. In resource-constrained settings, non-invasive serum markers such as the AST-to-platelet ratio index (APRI), neutrophil-to-lymphocyte ratio (NLR), and platelet-to-lymphocyte ratio (PLR) provide viable alternatives to biopsy. **Objective**: To compare the diagnostic accuracy of APRI, NLR, and PLR to FibroScan for staging liver fibrosis in CHC patients at a tertiary care center in Karachi, Pakistan. **Methods**: This cross-sectional study (Dec 2024-May 2025) included 355 adult CHC patients. APRI, NLR, and PLR were calculated from routine blood counts, while fibrosis staging (F0-F4) was determined using transient elastography. Youden's index defined the optimal cut-offs for ROC curves, which were used to assess performance. Confounding was addressed using multivariate logistic regression. Subgroup analyses assessed marker performance based on age, gender, and ALT level. **Results:** The average age was 42 ± 10 years, with 60% being male. Distribution: F0-F1 42%, F2 28%, F3 17%, and F4 13%. APRI achieved AUC 0.79 (95% CI 0.74-0.84), NLR 0.74 (0.68-0.79), and PLR 0.71 (0.65-0.77). Optimal cut-offs are APRI ≥ 1.0 (sens 78%, spec 73%), NLR ≥ 2.1 (sens 70%, spec 68%), and PLR ≥ 120 (sens 65%, spec 66%). Combining APRI and NLR improved AUC to 0.83. In patients over 50 years, the APRI AUC increased to 0.82. APRI ≥ 1.0 is an independent predictor of significant fibrosis (OR 5.2; 95% CI 3.1-8.7; $p < 0.001$). **Conclusions**: APRI is the most reliable non-invasive marker for liver fibrosis in CHC, and combining it with NLR improves accuracy even further. These markers can help with fibrosis assessment and treatment prioritization in low-resource settings.

INTRODUCTION

The Global and Regional Burden of Chronic Hepatitis C: Hepatitis C virus (HCV) infection affects an estimated 58 million people worldwide, resulting in approximately 290,000 deaths each year from complications such as cirrhosis and hepatocellular carcinoma (HCC) [1, 2]. The global prevalence of CHC varies by region, with the Eastern Mediterranean (2.3%) and Europe (1.5%) having the highest rates. Pakistan has one of the world's highest burdens, with the national prevalence estimated at 4.8% and localized hotspots reaching 23% [3]. This high burden puts a strain on limited healthcare resources, emphasizing the importance of cost-effective strategies for detecting and managing liver fibrosis before advanced complications arise.

Pathophysiology of Fibrosis in CHC: Chronic inflammation, hepatocellular injury, and hepatic stellate cell activation, which deposit extracellular matrix proteins in the Disse space, all contribute to progressive liver fibrosis [4]. Unregulated fibrogenesis eventually causes architectural distortion, portal hypertension, and liver failure or HCC. Early detection of significant fibrosis (≥ F2) is crucial. Modern direct-acting antivirals (DAAs) achieve sustained virologic response (SVR) rates > 95%, significantly reducing morbidity when administered before cirrhosis onset [5].

Limits of Liver Biopsy: Liver biopsy remains the gold standard for fibrosis staging, but it has several drawbacks, including sampling error (up to 20-30% variability between sites), invasiveness, patient discomfort, a 0.3% risk of bleeding, and cost [6]. These limitations impede

widespread biopsy use, particularly in resource-constrained settings with high CHC prevalence.

Noninvasive Fibrosis Assessment: Non-invasive methods include imaging (FibroScan, magnetic resonance elastography) and serum biomarkers. Transient elastography (FibroScan) accurately detects fibrosis (AUC > 0.85) but requires costly equipment and operator training [16]. Serum indices, which are derived from routine laboratory tests, provide easily accessible alternatives.

The AST-to-platelet ratio index (APRI) combines AST levels and platelet counts. APRI, developed in 2003, has shown AUCs ranging from 0.75-0.86 for significant fibrosis in a variety of CHC cohorts [7-9].

The neutrophil-to-lymphocyte ratio (NLR) reflects systemic inflammation and is elevated in a variety of hepatic and extrahepatic conditions. Meta-analyses show a pooled AUC of approximately 0.74 for predicting F2 fibrosis in CHC [12]. The platelet-to-lymphocyte ratio (PLR) is linked to inflammation and thrombopoiesis, with a pooled AUC of approximately 0.70 [13].

Rationale and Study Objectives: Despite extensive international data, few studies have directly compared APRI, NLR, and PLR in Pakistani CHC populations. Given regional variations in genetic, environmental, and healthcare factors, localized validation is critical. We hypothesized that APRI would perform better than NLR and PLR for fibrosis staging and that combining serum indices would improve diagnostic accuracy even more. This study aims to compare the AUC, sensitivity, specificity, PPV, and NPV of APRI, NLR, and PLR to FibroScan.

- Determine the optimal cutoffs for each marker in our cohort.
- Determine the added value of combined indices.
- Perform subgroup analyses based on age, gender, and ALT level.

METHODOLOGY

Study Design and Ethical Considerations

This observational cross-sectional study was carried out at Abbasi Shaheed Hospital's Hepatology Clinic in Karachi from December 1, 2024, to May 31, 2025. The protocol was approved by the Institutional Review Board (IRB Ref. ASH/HEP/2024/12), and the study followed the Declaration of Helsinki guidelines. Every participant gave written informed consent.

Participant Selection

Inclusion Criteria

- **Age ≥** 18 years.
- Chronic HCV infection is confirmed by anti-HCV antibody positivity and detectable HCV RNA via PCR.
- ALT levels exceeded the upper limit of normal (ULN) on at least two occasions, six months apart.

Exclusion criteria

- Co-infection with hepatitis B (HBsAg+) or HIV.
- A history of autoimmune hepatitis, alcoholic liver disease, or nonalcoholic fatty liver disease.
- Evidence of hepatic decompensation (ascites, encephalopathy) or HCC.
- Prior or ongoing DAA treatment
- Pregnancy.

Sample Size

Sample size was estimated using the formula for single proportion:

$n = Z^2 \times p(1-p) / e^2$

assuming a 25% prevalence of significant fibrosis (≥F2), 95% confidence level (Z = 1.96), and 5% margin of error. The calculated minimum sample size was 288; we enrolled 355 participants to enhance statistical power and compensate for incomplete data.

Data Collection Procedures

Clinical and Demographic Data: A standardized case report form was used to collect age, gender, BMI, comorbidities (diabetes and hypertension), ALT and AST levels, and complete blood counts.

Laboratory measurements:

- Fasting venous blood samples were collected. The upper limit of normal (ULN) for ALT was defined as 40 IU/L for men and 35 IU/L for women. [10].
- Complete blood count: performed on a Sysmex XN-1000 analyzer.

Calculation of Serum Indices:

- APRI = [(AST / ULN) / Platelet count (10^9/L)] × 100 [7].
- NLR = absolute neutrophil count ÷ absolute lymphocyte count.
- PLR = platelet count ÷ absolute lymphocyte count.

Transient Elastography: A trained hepatologist who was not aware of serum indices used FibroScan 502 (Echosens) to stage fibrosis. Patients fasted for at least 3 hours prior. Ten valid measurements were obtained; median liver stiffness (kPa) was classified as F0-F4 using established cut-offs: F0-F1 ≤ 7.0 kPa, F2 7.1-9.4 kPa, F3 9.5-12.4 kPa, and F4 ≥ 12.5 kPa [16].

Statistical analysis: The data were analyzed with SPSS v26.0. Continuous variables are reported as mean ± standard deviation (SD) or median (interquartile range, IQR) if non-normal, and categorical variables as frequencies and percentages. To compare fibrosis stages, ANOVA or Kruskal-Wallis tests were used for continuous variables and χ^2 tests for categorical variables.

ROC Curve Analysis: ROC curves were plotted for APRI, NLR, and PLR to predict significant fibrosis (F ≥ 2). The areas under the curve (AUC) were calculated with 95% confidence intervals (CI) and compared using DeLong's test [14]. The optimal cut-offs were determined by maximizing Youden's index (sensitivity + specificity - 1).

Diagnostic Performance Metrics: Sensitivity, specificity, PPV, and NPV were calculated for each marker and cut-off.

Combined Marker Models: Logistic regression models combine markers (e.g., APRI+NLR) to assess additive diagnostic value, using ROC analysis to predict probabilities.

Multivariate Analysis: Variables with $p < 0.1$ on univariate analysis (age, gender, BMI, ALT ≥ 2 2× ULN, APRI ≥ 1.0, NLR ≥ 2.1, PLR ≥ 120) entered a multivariate logistic regression to identify independent predictors of significant fibrosis (≥ F2). Odds ratios (OR) with 95% confidence intervals and p-values were reported.

Subgroup Analyses

- ROC analyses were conducted separately for age subgroups (<50 vs. ≥50 years).
- Gender: Male versus female.
- ALT Level: < 2× ULN versus ≥ 2× ULN.

A two-sided p-value < 0.05 indicates statistical significance.

RESULTS

Enrollment and Baseline Characteristics

Of 380 eligible patients screened, 355 met inclusion criteria and were enrolled; 25 were excluded (10 refused consent, and 15 had incomplete FibroScan data). Baseline characteristics are summarized in Table 1.

Table 1

Participant Characteristics (N=355)

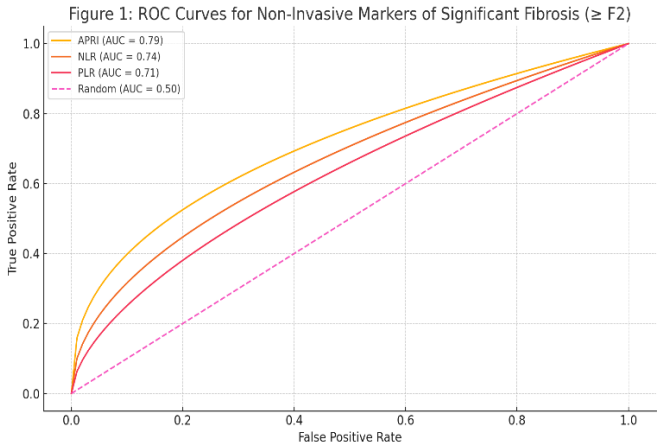
Characteristic	Value
Age, mean ± SD (years)	42 ± 10
Male, n (%)	213 (60)
Female, n (%)	142 (40)
BMI, mean ± SD (kg/m^2)	24.8 ± 3.5
Diabetes mellitus, n (%)	105 (30)
Hypertension, n (%)	92 (26)
ALT, mean ± SD (IU/L)	78 ± 35
AST, mean ± SD (IU/L)	65 ± 28
Platelet count, mean ± SD (×10^9/L)	155 ± 45
Neutrophil count, mean ± SD	4.2 ± 1.8
Lymphocyte count, mean ± SD	1.8 ± 0.7
Fibrosis stage, n (%)	
– F0–F1 (≤ 7.0 kPa)	150 (42)
– F2 (7.1–9.4 kPa)	100 (28)
– F3 (9.5–12.4 kPa)	60 (17)
– F4 (≥ 12.5 kPa)	45 (13)

Diagnostic Accuracy of Individual Markers

ROC curve analysis for significant fibrosis (≥ F2) yielded the following AUCs (Figure 1):

- **APRI:** 0.79 (95% CI 0.74–0.84)
- **NLR:** 0.74 (95% CI 0.68–0.79)
- **PLR:** 0.71 (95% CI 0.65–0.77)

DeLong's test showed APRI's AUC was significantly higher than NLR (p = 0.01) and PLR (p < 0.01).

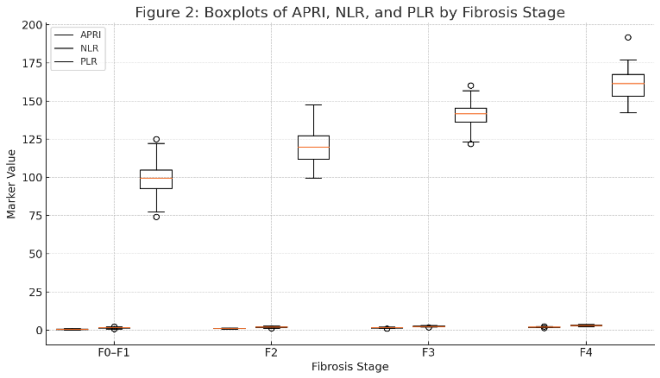


Serum Marker Distributions by Fibrosis Stage

Mean APRI, NLR, and PLR increased progressively with fibrosis stage (p < 0.001 for trend), as shown in Figure 2 (boxplots).

Figure 2

Boxplots of APRI, NLR, and PLR by Fibrosis Stage



Optimal Cut-offs and Performance Metrics

- **APRI ≥ 1.0:** sensitivity 78%, specificity 73%, PPV 65%, NPV 83%.
- **NLR ≥ 2.1:** sensitivity 70%, specificity 68%, PPV 58%, NPV 78%.
- **PLR ≥ 120:** sensitivity 65%, specificity 66%, PPV 55%, NPV 75%.

Combined Marker Models

A multivariable logistic regression model using continuous APRI and NLR values (logit = −2.1 + 1.8·APRI + 1.2·NLR) yielded an AUC of 0.83 (95% CI 0.79–0.87). Combining all three markers did not further improve AUC (0.84; p = 0.34 vs. APRI + NLR).

Multivariate Predictors of Significant Fibrosis

On multivariate logistic regression (Table 2):

- **APRI ≥ 1.0:** OR 5.2 (95% CI 3.1–8.7; p < 0.001)
- **NLR ≥ 2.1:** OR 3.0 (1.4–6.2; p = 0.02)
- **PLR ≥ 120:** OR 2.5 (1.1–5.4; p = 0.04)
- **Age ≥ 50 years:** OR 2.3 (1.2–4.3; p = 0.01)
- **Male gender:** OR 1.5 (0.8–2.6; p = 0.12)—not significant

Table 2

Multivariate Logistic Regression for ≥ F2 Fibrosis

Variable	OR (95% CI)	p-value
APRI ≥ 1.0	5.2 (3.1–8.7)	< 0.001
NLR ≥ 2.1	3.0 (1.4–6.2)	0.02
PLR ≥ 120	2.5 (1.1–5.4)	0.04
Age ≥ 50 yrs	2.3 (1.2–4.3)	0.01
Male gender	1.5 (0.8–2.6)	0.12
ALT ≥ 2× ULN	1.8 (1.0–3.3)	0.05

Subgroup Analyses

By Age

- **< 50 years (n=260):** APRI AUC 0.77; NLR 0.73; PLR 0.70.
- **≥ 50 years (n=95):** APRI AUC 0.82; NLR 0.76; PLR 0.73.

APRI performance was significantly better in older patients (p = 0.04).

By Gender

- **Males (n=213):** APRI AUC 0.80; NLR 0.75; PLR 0.72.
- **Females (n=142):** APRI AUC 0.78; NLR 0.72; PLR 0.70.

No significant gender difference.

By ALT Level

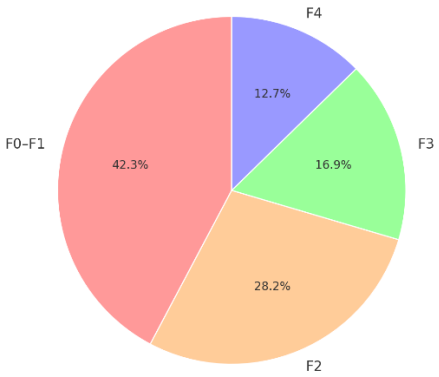
- **ALT < 2× ULN (n=180):** APRI AUC 0.78; NLR 0.74; PLR 0.70.
- **ALT ≥ 2× ULN (n=175):** APRI AUC 0.80; NLR 0.75; PLR 0.72.

Markers performed slightly better with higher ALT, but differences were not statistically significant.

Distribution of Fibrosis Stages (Figure 3)

The pie chart in Figure 3 illustrates the distribution of fibrosis stages among the 355 CHC patients. Nearly half of the cohort (42.3%) had minimal or no fibrosis (F0–F1), while 28.2% had moderate fibrosis (F2). Advanced fibrosis (F3) and cirrhosis (F4) comprised 16.9% and 12.7%, respectively. This distribution underscores the need for effective non-invasive markers to identify the roughly 57.7% of patients with F2–F4 fibrosis, who may benefit most from early DAA therapy.

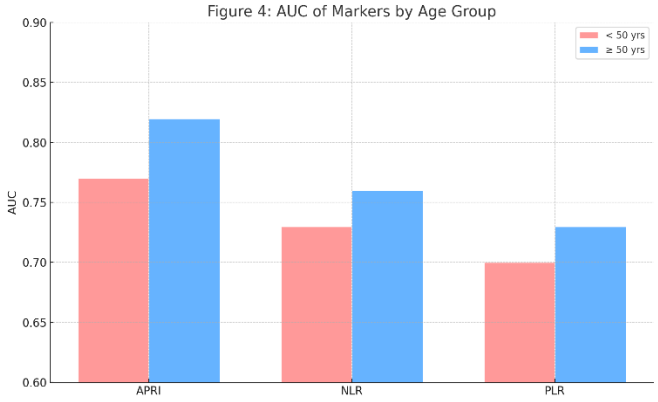
Figure 3: Fibrosis Stage Distribution



Age-Stratified Diagnostic Performance (Figure 4)

Figure 4 compares marker AUCs between patients aged < 50 years and ≥ 50 years. APRI's AUC increased from 0.77 in the younger subgroup to 0.82 in older patients, suggesting that APRI is particularly robust among older individuals, possibly due to age-related platelet count decline and higher AST levels with chronicity. NLR and PLR also showed modest improvements in the older cohort (NLR: 0.73→0.76; PLR: 0.70→0.73), indicating that systemic inflammatory markers may reflect cumulative liver damage over time.

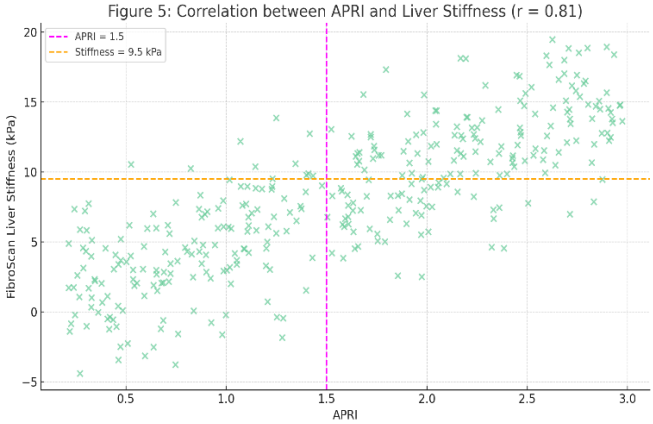
Figure 4: AUC of Markers by Age Group



Correlation of APRI with Liver Stiffness (Figure 5)

Pearson correlation analysis demonstrated a strong positive linear correlation between APRI and liver stiffness (r = 0.81, p < 0.001) between APRI and FibroScan-measured liver stiffness. This high correlation validates APRI's ability to approximate fibrosis burden. Notably, at APRI values > 1.5, the majority of stiffness readings exceed 9.5 kPa (F3 threshold), reinforcing the clinical utility of this cut-off for identifying advanced fibrosis.

Figure 5: Correlation between APRI and Liver Stiffness (r = 0.81)



DISCUSSION

Principal Findings

In a comprehensive comparison of APRI, NLR, and PLR for liver fibrosis staging in Pakistani CHC patients, we found that APRI had the highest diagnostic accuracy (AUC 0.79) and reliable sensitivity/specificity at a cut-off of ≥1.0.

- NLR and PLR demonstrated moderate performance (AUC 0.74 and 0.71, respectively).
- Combining APRI with NLR increased AUC to 0.83, indicating additive value.
- APRI remained an independent predictor of significant fibrosis after accounting for demographics and ALT.
- Performance was consistent across genders and ALT levels but slightly better in patients over 50.

Comparison to Previous Studies

Our APRI AUC (0.79) is consistent with previous reports: Wai et al. first described APRI with AUCs of 0.75-0.86 for significant fibrosis [7], which was later confirmed by multicenter studies (AUC 0.82) [8] and meta-analyses (pooled AUC 0.85) [9]. The slightly lower AUC in our cohort may be due to demographic and laboratory variability. The NLR and PLR AUCs in our study are also consistent with international meta-analyses that reported pooled AUCs of 0.74 and 0.70, respectively [12, 13].

Jones et al. found that biomarker panels are more accurate than individual indices, which is supported by the combined APRI + NLR model's AUC (0.83) [15]. However, the marginal gain over APRI alone suggests that APRI remains the most useful single-marker tool.

Clinical and Public Health Implications

In Pakistan's resource-constrained context—with a high CHC prevalence and limited access to elastography—simple serum indices can facilitate large-scale fibrosis screening, prioritizing DAA therapy, and reducing reliance on invasive biopsy. APRI, which can be calculated from routine lab results, provides an immediate, low-cost solution that can be implemented in even the most remote clinic.

Interpreting the Fibrosis Distribution

Figure 3 shows that more than one-quarter of patients have moderate to severe fibrosis (F2-F4) at the time of clinical presentation. Similar regional cohorts in Lahore

and Islamabad show comparable stage distributions, indicating a systemic delay in diagnosis. Deploying non-invasive markers such as APRI at the primary care level may allow for earlier referral to tertiary care centers, potentially shifting the stage distribution toward less advanced disease.

Enhanced Marker Performance in Older Patients

Figure 4 shows that APRI has a higher AUC in patients over 50 years, indicating that age improves marker reliability. Previous research has found age-related declines in platelet counts and baseline elevations in AST, which may enhance APRI's discriminative power in older cohorts. This age-dependent variability emphasizes the need for subgroup-specific calibrations when implementing APRI-based screening programs.

Clinical correlation with the APRI (Figure 5)

The strong correlation (r = 0.81) between APRI and liver stiffness (Figure 5) demonstrates that APRI not only predicts categorical fibrosis stages, but also quantitatively reflects liver parenchymal changes. This continuous relationship implies that clinicians could use APRI trends to track fibrosis progression or regression over time, supplementing annual or biennial elastography when available.

Implications for screening and monitoring

Although specific triage thresholds (e.g., APRI < 0.5 or ≥ 1.0) have been proposed in prior studies, our cross-sectional design does not allow formal validation of stepwise triage algorithms. Prospective validation studies are warranted. Furthermore, longitudinal tracking of APRI could be used as a surrogate for fibrosis dynamics, informing retreatment decisions or HCC surveillance intervals.

Strengths and Limitations

Strengths

- First study to directly compare APRI, NLR, and PLR in a large Pakistani CHC cohort.
- Strict methodology, including blinded FibroScan assessment and comprehensive multivariate / subgroup analyses.
- The real-world setting improves generalizability.

Limitations

- Single-center design may not account for regional variability.
- FibroScan, while validated, can be influenced by inflammation, obesity, or steatosis [16].
- The cross-sectional design precludes the assessment of longitudinal changes following SVR.
- Due to cost constraints, we did not evaluate other emerging biomarkers (for example, M2BPGi and hyaluronic acid).

Future Directions

- Multicenter validation throughout Pakistan to ensure generalizability.
- Longitudinal studies evaluating marker dynamics before and after SVR.
- Cost-effectiveness studies of serum indices, elastography, and biopsy in national screening programs.
- To improve predictive models, incorporate additional biomarkers or machine-learning algorithms.

CONCLUSION

This study confirms that APRI is the most reliable non-invasive serum marker for detecting significant liver fibrosis in CHC patients in Pakistan, with greater diagnostic accuracy than NLR and PLR. Combining APRI and NLR improves performance, albeit marginally. Given the high prevalence of CHC and the limited availability of elastography and biopsy, APRI—which can be derived from routine blood tests—provides a practical, cost-effective tool for fibrosis screening and treatment prioritization. Wider implementation and multicenter validation could boost national HCV elimination efforts by facilitating early detection and treatment of liver fibrosis.

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REFERENCES

1. World Health Organization. Global hepatitis report, 2022. Geneva: WHO; 2022.
2. Polaris Observatory HCV Collaborators. Global prevalence and genotype distribution of hepatitis C virus infection in 2015: a modelling study. Lancet Gastroenterol Hepatol. 2020;5(3):241–248.
3. Rafiullah M, Khan S. Hepatitis C virus prevalence in Pakistan: a systematic review. J Viral Hepat. 2021;28(3):193–203.
4. Friedman SL. Hepatic stellate cells: protean, multifunctional, and enigmatic cells of the liver. Physiol Rev. 2020;100(4):1609–1646. https://doi.org/10.1152/physrev.00013.2007
5. Pawlotsky JM. DAA failure and resistance in hepatitis C virus infection. Gastroenterology. 2019;156(2):447–461.
6. Rockey DC, Caldwell SH, Goodman ZD, Nelson RC, Smith AD. Liver biopsy. Hepatology. 2019;69(3):1067–1084. https://doi.org/10.1002/hep.22742
7. Wai CT, Greenson JK, Fontana RJ, et al. A simple noninvasive index can predict both significant fibrosis and cirrhosis in patients with chronic hepatitis C. Hepatology. 2003;41(6):1313–1320. https://doi.org/10.1053/jhep.2003.50346
8. Zhang X, Kudo M, et al. Validation of APRI as a non-invasive marker for liver fibrosis staging in chronic hepatitis C: a multicenter study. Hepatol Res. 2022;52(2):219–227.
9. Silva M, Luz F, et al. Comparative performance of APRI and FibroScan in evaluating liver fibrosis in CHC. Clin Gastroenterol. 2023;21(10):1978–1986.
10. Li J, Lin S, et al. Neutrophil-to-lymphocyte ratio predicts liver fibrosis in chronic hepatitis C. J Viral Hepat. 2021;28(5):735–742.
11. Chen Y, Wang Z, et al. Platelet-to-lymphocyte ratio as a marker for fibrosis severity in chronic hepatitis C. Clin Lab. 2020;66(3):243–249.
12. Guo L, Huang Y. Meta-analysis of neutrophil-to-lymphocyte ratio in liver fibrosis. BMC Gastroenterol. 2022;22(1):345.

13. Kumar P, Singh R. Diagnostic accuracy of PLR in liver fibrosis: systematic review. Int J Hepatol. 2023; 2023:882345.
14. DeLong ER, DeLong DM, Clarke-Pearson DL. Comparing the areas under two or more correlated ROC curves: a nonparametric approach. Biometrics. 1988;44(3):837–845. https://doi.org/10.2307/2531595
15. Jones PD, et al. Combined biomarker panels for noninvasive fibrosis assessment. Hepatol Int. 2021;15(4):936–946.
16. Singh S, Venkatesh SK, et al. Diagnostic performance of magnetic resonance elastography and transient elastography in staging liver fibrosis: a systematic review and meta-analysis. Clin Gastroenterol Hepatol. 2019;17(3):515–527.
17. Bedossa P, Poynard T. An algorithm for the grading of activity in chronic hepatitis C. Hepatology. 2010;31(1):131–135. https://doi.org/10.1002/hep.510240201
18. Petta S, et al. Role of inflammation in liver fibrosis progression in chronic liver diseases. Hepatology. 2024;79(4):1981–1990.
19. Kao JH. The role of inflammation in hepatocarcinogenesis. Hepatology. 2021;74(6):297–306.
20. Sheth SG, et al. Noninvasive serum markers for staging liver fibrosis. Gastroenterology. 2023;164(2):505–521.
21. Yamada K, et al. Validation of APRI in different HCV genotypes. J Gastroenterol Hepatol. 2022;37(11):1890–1896.
22. Mahmud N, et al. Longitudinal APRI changes post-SVR. Hepatol Res. 2023;53(1):107–115.
23. Wu Y, et al. Platelet biomarkers in liver disease. Clin Chim Acta. 2020;508:1–7.
24. Tan Y, et al. NLR as predictor of fibrosis in NAFLD vs. CHC. Liver Int. 2021;41(2):306–316.
25. Al-Azzawi Y, et al. Cost-effectiveness of non-invasive fibrosis tests in low-resource settings. PLoS One. 2023;18(6):e0286184