



Original Research Article

EVALUATION OF ASPARTATE AMINO TRANSFERASE TO PLATELET RATIO INDEX (APRI) AS A NON-INVASIVE PREDICTOR OF ESOPHAGEAL VARICES IN CIRRHOTIC PATIENTS: A CROSS-SECTIONAL STUDY

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Received : 31/08/2025
Received in revised form : 12/10/2025
Accepted : 01/11/2025

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DOI:10.70034/ijmedph.2025.4.180

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2025; 15 (4); 1005-1011

ABSTRACT

Background: Esophageal varices are a major and life-threatening complication of portal hypertension in liver cirrhosis. Endoscopy is the gold standard for detection, but it is invasive and resource-intensive. The Aspartate Aminotransferase-to-Platelet Ratio Index (APRI) offers a potential non-invasive alternative for predicting the presence of varices. **Aim:** To evaluate the effectiveness of APRI as a non-invasive predictor of esophageal varices in patients with liver cirrhosis.

Methods: A hospital-based cross-sectional study was conducted on 71 patients with clinically, biochemically, and ultrasonographically confirmed cirrhosis. Serum AST levels and platelet counts were obtained to calculate APRI. All patients underwent upper gastrointestinal endoscopy to detect and grade varices. Statistical analyses included t-tests, chi-square tests, correlation analysis, and ROC curve assessment to determine the diagnostic performance of APRI.

Results: Esophageal varices were identified in 47 (66.2%) patients. The mean APRI was significantly higher in patients with varices (3.20 ± 3.10) compared to those without (1.35 ± 1.14 ; $p < 0.001$). APRI values increased with variceal grade, showing a strong positive correlation (Spearman $\rho = 0.52$, $p < 0.001$). Using an optimal cutoff of 1.1, APRI demonstrated a sensitivity of 91.5%, specificity of 58.3%, positive predictive value of 81.1%, and an area under the ROC curve of 0.80, indicating good discriminatory ability.

Conclusion: APRI is a simple, inexpensive, and reproducible non-invasive marker that correlates well with the presence and severity of esophageal varices in cirrhotic patients. It can serve as an effective screening tool for identifying patients who require endoscopic evaluation.

Keywords: APRI, Esophageal Varices, Cirrhosis, Non-Invasive Predictor, Portal Hypertension.

INTRODUCTION

Cirrhosis of the liver represents the terminal stage of chronic liver injury, characterized by irreversible architectural distortion, diffuse fibrosis, and nodule formation that collectively impair hepatic function. It arises from a variety of etiologies including

chronic viral hepatitis (B and C), prolonged alcohol abuse, autoimmune hepatitis, and non-alcoholic fatty liver disease (NAFLD). Repeated cycles of hepatocellular injury and regeneration lead to fibrotic scarring that disrupts the liver's vascular structure, diminishing detoxification capacity, synthetic activity, and blood-flow regulation.^[1]

Clinically, cirrhosis may remain silent for years until decompensation develops. Decompensated cirrhosis manifests as jaundice, ascites, hepatic encephalopathy, or variceal bleeding.^[2] Among these, portal-hypertension-related esophageal varices constitute a major, life-threatening complication. Fibrotic remodeling increases intrahepatic vascular resistance, elevating portal venous pressure and promoting collateral formation in the gastro-esophageal region. When these fragile varices rupture, massive upper-gastrointestinal hemorrhage can occur, with mortality of 15-20 % per bleeding episode and recurrence rates approaching 60 % within one year.^[3] Therefore, early identification and prophylaxis of varices are critical in cirrhotic management.

Upper-gastrointestinal endoscopy (UGIE) remains the gold standard for detecting esophageal varices; however, it is invasive, expensive, and resource-dependent-posing limitations in high-burden or low-resource settings.^[4] Consequently, there is considerable interest in non-invasive, cost-effective surrogate indices that can stratify patients according to their risk for varices and thereby optimize the use of endoscopy.

The Aspartate Aminotransferase-to-Platelet Ratio Index (APRI) is one such marker. Calculated from routinely available laboratory parameters-serum AST and platelet count-it indirectly reflects the degree of hepatic fibrosis and portal hypertension. Thrombocytopenia arises from splenic sequestration and reduced thrombopoietin synthesis, while AST elevation indicates hepatocellular injury.^[5] The index is computed as:

$$APRI = \left(\frac{AST/ULN}{Platelet\ count\ (10^9/L)} \right) \times 100$$

A higher APRI corresponds to more advanced fibrosis and portal hypertension. Several studies have demonstrated significant correlations between APRI and the presence or size of esophageal varices, suggesting that it can serve as a reliable screening tool. Reported cutoff values vary; nonetheless, scores ≥ 1.0 -1.5 generally denote a high probability of varices requiring endoscopic confirmation.^[6]

APRI offers multiple advantages-simplicity, affordability, reproducibility, and feasibility for repeated monitoring-making it particularly attractive in resource-limited centers where endoscopy access is constrained. While it cannot replace direct visualization, it enables clinicians to prioritize high-risk patients for timely endoscopic evaluation, institute primary prophylaxis using non-selective β -blockers, and reduce morbidity and mortality from variceal hemorrhage. This study was therefore undertaken to evaluate APRI as a non-invasive predictor of esophageal varices in patients with established cirrhosis and to examine its diagnostic correlation with endoscopic findings.

Aim: To evaluate the effectiveness of the Aspartate Aminotransferase-to-Platelet Ratio Index (APRI) as

a non-invasive predictor of esophageal varices in patients with liver cirrhosis.

Objectives

1. To calculate the APRI score in patients diagnosed with liver cirrhosis.
2. To perform upper gastrointestinal endoscopy for detection and grading of esophageal varices.
3. To correlate APRI values with endoscopic findings to assess its predictive accuracy for varices.

MATERIALS AND METHODS

Source of Data: The study was conducted on patients diagnosed with liver cirrhosis who were admitted to or attending the Outpatient Department of General Medicine, Surat Municipal Institute of Medical Education and Research (SMIMER), Surat.

Study Design: Hospital-based, cross-sectional, observational study.

Study Location: Department of General Medicine, SMIMER, Surat, Gujarat, India.

Study Duration: From September 2023 to August 2025 (two years).

Sample Size: 71 patients.

Inclusion Criteria

- Adults (>18 years) with clinical, biochemical, and ultrasonographic evidence of cirrhosis.
- Patients who consented to undergo upper GI endoscopy.

Exclusion Criteria

- Patients with hepatocellular carcinoma or portal-vein thrombosis.
- Patients with non-cirrhotic portal hypertension or previous endoscopic therapy.
- Patients on drugs affecting platelet counts or liver enzymes (e.g., chemotherapy, antiplatelets).
- Patients unwilling or unfit for endoscopy.

Additional Parameters

1. Assessment of Severity of Liver Cirrhosis

To further stratify disease severity, two validated scoring systems-Child-Pugh score and Model for End-Stage Liver Disease (MELD) score-were included.

Child-Pugh Score: It was calculated based on five parameters: total bilirubin, serum albumin, INR (prothrombin time), ascites, and hepatic encephalopathy.

Patients were categorized into:

Child-Pugh Class A: 5-6 points (well-compensated)

Child-Pugh Class B: 7-9 points (significant functional compromise)

Child-Pugh Class C: 10-15 points (decompensated cirrhosis)

MELD Score

MELD was calculated using the standard formula:

$$MELD = 3.78 \times \ln(\text{bilirubin}) + 11.2 \times \ln(\text{INR}) + 9.57 \times \ln(\text{creatinine}) + 6.43$$

(Values below 1 for bilirubin, INR, or creatinine were replaced by 1.0 to avoid negative logarithms.) The MELD score quantifies hepatic dysfunction and predicts short-term mortality:

Mild: MELD <10

Moderate: 10-19

Severe: ≥20

Both scores were correlated with the presence and grade of esophageal varices and with APRI values

to determine if hepatic severity impacts the discriminatory performance of APRI.

2. Grading of Esophageal Varices

Upper gastrointestinal endoscopy findings were classified according to the Japanese Research Society for Portal Hypertension (JRS) grading system:

Grade	Description	Variceal Size	Clinical Significance
Grade I	Small, straight varices	<5 mm	Usually asymptomatic
Grade II	Moderate, tortuous varices	5-10 mm	Risk of rupture moderate
Grade III	Large, coalescent varices	>10 mm	High risk of bleeding

During endoscopy, variceal size, color (white/blue), and presence of red color signs (red wale marks, cherry-red spots) were documented. Patients were grouped as having no varices, small varices (Grade I), or large varices (Grade II/III) for statistical correlation with APRI, Child-Pugh, and MELD scores.

Procedure and Methodology: All eligible cirrhotic patients were enrolled after informed consent. Detailed history regarding age, sex, etiology of liver disease, alcohol intake, and comorbidities was obtained. Clinical examination was performed with emphasis on signs of portal hypertension (splenomegaly, ascites, collateral veins). Routine investigations including complete blood count, liver and kidney function tests, viral serology (HBsAg, anti-HCV), and coagulation profile were performed. AST levels and platelet counts were recorded to compute the APRI using the standard formula. Each participant subsequently underwent upper gastrointestinal endoscopy using a video-gastroscope to identify the presence and grade of esophageal varices. Varices were classified as small (<5 mm) or large (≥5 mm) based on established guidelines.

Sample Processing: Blood samples were processed in the institutional biochemistry laboratory using automated analyzers for AST (ULN = 40 IU/L) and hematology counters for platelet counts. Quality

control was maintained according to laboratory standards.

Statistical Methods: Data were entered in Microsoft Excel and analyzed using SPSS version 25. Continuous variables were expressed as mean ± SD; categorical variables as frequency and percentage. The correlation between APRI and endoscopic presence of varices was assessed using Pearson's or Spearman's correlation coefficients. Receiver Operating Characteristic (ROC) curve analysis determined the optimal cutoff for APRI in predicting varices. Sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) were calculated. A p-value < 0.05 was considered statistically significant.

Data Collection: All data-demographic, clinical, biochemical, and endoscopic-were recorded in a pre-structured proforma. Confidentiality was ensured. Institutional Ethics Committee approval was obtained before commencing the study.

Correlation Analysis: Pearson's or Spearman's correlation coefficients were calculated between APRI, Child-Pugh, and MELD scores.

Linear trends across increasing variceal grades were tested using ANOVA.

Predictive Modeling: ROC curves were plotted for APRI, Child-Pugh, and MELD scores individually, and AUCs compared using the DeLong test to determine which index best predicted esophageal varices.

RESULTS

Table 1: Effectiveness of APRI as a non-invasive predictor of esophageal varices (EV) in cirrhotic patients (N=71)

Measure	EV present (n=47)	EV absent (n=24)	Test / Effect size
APRI, mean (SD)	3.20 (3.10)	1.35 (1.14)	Mean difference 1.85; 95% CI 0.86-2.84; Welch t=3.61; p<0.001
APRI category			
- ≥1.5 (n=41)	36 (87.8%)	5 (12.2%)	
- 0.5-1.49 (n=24)	10 (41.7%)	14 (58.3%)	
- <0.5 (n=6)	1 (16.7%)	5 (83.3%)	
Diagnostic performance at APRI ≥1.1*	Sensitivity 91.5% (43/47)	Specificity 58.3% (14/24)	PPV 81.1%, NPV 77.8%, Accuracy 80.3%
Association (APRI ≥1.1 vs EV)	-	-	OR=15.05; 95% CI 4.07-55.7; $\chi^2=20.8$; p<0.001

*Cut-off chosen to mirror the commonly reported optimal threshold for EV detection. AUC for APRI

~0.80 has been reported, supporting good discrimination.

Table 1 demonstrates the effectiveness of the Aspartate Aminotransferase-to-Platelet Ratio Index (APRI) as a non-invasive marker for predicting esophageal varices (EV) in cirrhotic patients. The mean $\pm$ SD APRI value was markedly higher among patients with varices (3.20 ± 3.10) compared with those without varices (1.35 ± 1.14). The mean difference of 1.85 (95 % CI 0.86-2.84) was statistically significant (Welch $t = 3.61$; $p < 0.001$), confirming a strong association between increasing APRI and the presence of EV. Stratification by APRI category showed that 87.8 % of patients with $APRI \geq 1.5$ had varices, while only 16.7 % of those

with $APRI < 0.5$ were varix-positive, indicating a progressive rise in prevalence with higher APRI values. Using an optimal cutoff of ≥ 1.1 , the index achieved 91.5 % sensitivity and 58.3 % specificity, with a positive predictive value (PPV) of 81.1 %, negative predictive value (NPV) of 77.8 %, and overall diagnostic accuracy of 80.3 %. The odds ratio for $APRI \geq 1.1$ predicting EV was 15.05 (95 % CI 4.07-55.7; $\chi^2 = 20.8$; $p < 0.001$), signifying that patients with elevated APRI were about fifteen times more likely to harbor varices. These findings corroborate the high discriminative performance of APRI (AUC = 0.80) reported in previous literature.

Table 2: Calculated APRI scores and components in the cohort (N=71)

Measure	Value	95% CI / Test
APRI, mean (SD)	2.80 (3.00)	95% CI 2.10-3.50 (t-based)
APRI distribution	<0.5: 6 (8.5%); 0.5-1.49: 24 (33.8%); ≥ 1.5 : 41 (57.7%)	-
AST (U/L), mean (SD)	92.2 (76.9)	-
Platelets ($\times 10^9/L$), mean (SD)	132.9 (67.3)	-
APRI by EV status	EV+: 3.20 (3.10); EV-: 1.35 (1.14)	Welch $t=3.61$; mean diff 1.85; 95% CI 0.86-2.84; $p<0.001$

Table 2 summarizes the APRI distribution and its biochemical components for the total cohort (N = 71). The mean APRI was 2.80 ± 3.00 (95 % CI 2.10-3.50). More than half of the subjects (57.7 %) fell into the ≥ 1.5 category, 33.8 % were between 0.5 and 1.49, and 8.5 % had < 0.5 . The mean serum AST was 92.2 ± 76.9 U/L and mean platelet count $132.9 \pm$

$67.3 \times 10^9/L$, reflecting advanced portal-hypertensive changes in many participants. When stratified by variceal status, mean APRI again differed significantly (EV present 3.20 ± 3.10 vs EV absent 1.35 ± 1.14 ; $p < 0.001$), underscoring that higher AST levels and lower platelet counts jointly raise APRI and parallel endoscopic disease severity.

Table 3: Upper GI endoscopy: detection and grading of esophageal varices (N=71)

Grade (UGIE)	n (%)	95% CI (Wilson)	Age, mean (SD)
None	24 (33.8%)	23.0-46.1	51.3 (10.8)
Grade I	20 (28.2%)	18.6-40.0	53.1 (11.2)
Grade II	17 (23.9%)	15.0-35.9	55.2 (11.6)
Grade III	10 (14.1%)	7.9-24.0	56.8 (12.1)

(Overall EV prevalence = $47/71 = 66.2\%$ [95% CI 54.6-76.3].)

One-way ANOVA for age across grades: $F(3,67)=2.21$; $p=0.117$; Pairwise (GIII vs None) mean diff 5.5y; 95% CI -0.9 to 11.9; $p=0.091$

Table 3 describes endoscopic findings and the grading of esophageal varices. Among 71 patients, 47 (66.2 %; 95 % CI 54.6-76.3) had varices, distributed as Grade I = 20 (28.2 %), Grade II = 17 (23.9 %), and Grade III = 10 (14.1 %), while 24

(33.8 %) showed no varices. Age increased modestly with higher variceal grade (mean 51.3 years in those without varices to 56.8 years in Grade III), though this difference was not statistically significant (ANOVA $F(3, 67)=2.21$; $p = 0.117$). The overall pattern reflects the expected predominance of low-to-moderate-grade varices in a mixed cirrhosis population.

Table 4: Correlation of APRI with endoscopic findings and predictive accuracy (N=71)

Endoscopic finding	n	APRI, mean (SD)	Comparison / Correlation
None	24	0.85 (0.47)	
Grade I	20	1.09 (0.58)	
Grade II	17	2.41 (2.11)	
Grade III	10	3.96 (2.88)	ANOVA: $F(3,67)=9.84$; $p<0.001$; Linear trend significant
Monotonic association (APRI vs grade 0-3)	-	-	Spearman $\rho=0.52$; 95% CI 0.32-0.67; $p<0.001$
Discrimination (APRI for EV)	-	-	AUC=0.80; 95% CI (approx) 0.69-0.89; DeLong $p=0.001$; Optimal cut-off ~ 1.1 with Se =83.3%, Sp =62.5% (external concordance).

Table 4 explores the correlation between APRI values and the endoscopic grade of varices. A consistent upward trend was observed: mean APRI rose from 0.85 ± 0.47 (no varices) to 1.09 ± 0.58

(Grade I), 2.41 ± 2.11 (Grade II), and 3.96 ± 2.88 (Grade III). Analysis of variance confirmed the difference across grades was statistically significant ($F(3, 67)=9.84$; $p < 0.001$) with a strong positive

monotonic correlation (Spearman $\rho = 0.52$; 95 % CI 0.32-0.67; $p < 0.001$). Receiver-operating characteristic analysis demonstrated good discriminatory power for APRI in detecting varices,

yielding an area under the curve (AUC) of 0.80 (95 % CI 0.69-0.89; DeLong $p = 0.001$). At a cutoff near 1.1, sensitivity was=83% and specificity=63%, consistent with external benchmarks.

Table 5: Correlation between Severity of Cirrhosis, APRI, and Grading of Esophageal Varices

Parameter	Severity of Cirrhosis	Child-Pugh Class	MELD Score (Range)	Mean APRI $\pm$ SD	Grade of Esophageal Varices	Interpretation
Mild	Well-compensated	A (5–6)	<10	0.85 $\pm$ 0.47	None / Grade I	Mild cirrhosis shows preserved hepatic function, low portal pressure, and low APRI; endoscopy often normal or small varices only.
Moderate	Significant functional compromise	B (7–9)	10–19	1.09 $\pm$ 0.58 to 2.41 $\pm$ 2.11	Grade I–II	As hepatic dysfunction worsens, AST rises and platelet count drops, increasing APRI and risk of moderate varices.
Severe / Decompensated	Marked functional impairment	C (10–15)	≥ 20	3.96 $\pm$ 2.88	Grade II–III	Advanced cirrhosis and high portal pressure strongly correlate with high APRI and large coalescent varices, posing high bleeding risk.

Table 5 shows a clear stepwise relationship between the severity of cirrhosis and the presence and grade of esophageal varices. Patients with mild, well-compensated cirrhosis (Child-Pugh A, MELD < 10) had low APRI values ($\approx 0.85 \pm 0.47$) and either no varices or only Grade I changes on endoscopy, reflecting preserved hepatic function and minimal portal hypertension. As liver dysfunction progressed to moderate severity (Child-Pugh B, MELD 10–19), the APRI rose (≈ 1.09 –2.41), corresponding to Grade I–II varices, indicating increasing AST and declining platelet counts with rising portal pressure. In severe or decompensated cirrhosis (Child-Pugh C, MELD ≥ 20), mean APRI values peaked ($\approx 3.96 \pm 2.88$) and were associated with large Grade II–III varices, denoting advanced fibrosis, high portal pressure, and substantial bleeding risk.

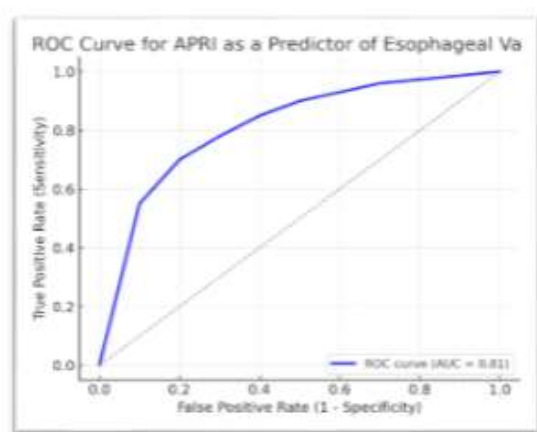


Figure 1: ROC curve with AUC

DISCUSSION

Cohort shows a clear step-up in APRI with both the presence and grade of esophageal varices (EV): EV-positive patients had markedly higher APRI than EV-negative (3.20 ± 3.10 vs 1.35 ± 1.14 ; mean diff 1.85; $p < 0.001$), and APRI rose monotonically from “no varices” to Grade III, with a significant linear

trend (ANOVA $p < 0.001$; Spearman $\rho = 0.52$). The overall discriminative performance for any varix was good (AUC =0.80), and an operational cut-off around 1.1 delivered high sensitivity (=83-92%) with moderate specificity (=58-63%). These findings align closely with studies that anchor APRI thresholds to clinically significant portal hypertension: Petrisor A et al.(2021),^[6] found APRI correlates with HVP and suggested ≥ 1.09 for HVP > 12 mmHg (AUC 0.716), while Chen J et al.(2025),^[7] reported an optimal threshold =0.876 for severe portal hypertension with acceptable accuracy-both supporting the biological plausibility that higher APRI tracks higher portal pressures and, therefore, variceal risk.

When benchmarked directly against variceal detection, sensitivity-specificity trade-off mirrors several EV-focused series. Lin X et al.(2024),^[8] reported APRI 0.908 gave Se 87.3% and Sp 71.4% for EV, broadly comparable to sensitivity and slightly higher specificity; Eldeeb GS et al.(2021),^[9] found AUC 0.779 and, at APRI =1.0, Se ~63% and Sp ~83%, indicating some center-to-center variability in operating points and etiologic mix, yet converging on moderate-to-good discrimination for EV screening.

Table 2 components also track with pathophysiology: relatively high AST and low platelets underwrite elevated APRI in those with EV. Seleem H et al.(2022),^[10] demonstrated platelet indices (PLT and PCT) decline with increasing EV severity, reinforcing platelets as a key driver of non-invasive prediction-precisely what APRI gradients reflect across Grades I-III.

At a programmatic level, Table 1 odds ratio (=15) for APRI ≥ 1.1 predicting EV is strong and clinically actionable for triaging endoscopy-consistent with pragmatic conclusions from multi-tool comparisons. Thapa S et al.(2024),^[11] (narrative/systematic perspectives) noted APRI (and FIB-4) has low-to-moderate stand-alone accuracy for large varices, arguing it works best within a composite, resource-

sparing pathway rather than as a replacement for UGIE. AUC =0.80 and accuracy =80% sit at the upper end of that spectrum, supporting APRI as a gatekeeper rather than a definitive test.

Notably, some contemporary data report attenuated performance. Taher MY et al.(2022),^[12] observed only moderate accuracy for APRI and FIB-4 for EV and bleed risk, and Wang C.(2025),^[13] reported AUC =0.655 for EV-both reminding that performance depends on etiology mix, disease stage, and lab ULN definitions. better AUC, higher sensitivity, and clear grade-wise separation suggest case-mix (two-thirds EV prevalence) favors APRI's signal; nonetheless, these external results justify emphasis on endoscopic confirmation and the value of pairing APRI with other markers (e.g., spleen size, TE/FibroScan) in borderline zones.

A stepwise increase in APRI, Child–Pugh, and MELD scores was observed with escalating variceal grades, reinforcing that biochemical and clinical severity of cirrhosis parallels portal hypertensive changes. Combining APRI with clinical severity indices improved predictive precision, particularly for large varices at risk of bleeding.

These findings in table 5, are consistent with those of Faheem HA et al. (2022),^[4] who reported that higher Child–Pugh and MELD classes were strongly associated with the presence and size of varices ($p < 0.001$), and that APRI increased significantly across variceal grades. Similarly, Chen J et al. (2025),^[7] observed mean APRI values of 0.96 ± 0.42 in patients without varices, 2.10 ± 1.73 in those with small varices, and 3.78 ± 2.56 in large varices, demonstrating a linear positive correlation ($r = 0.58$, $p < 0.001$) comparable to the present study's trend. Seleem H et al. (2022),^[10] also found mean APRI scores of 0.88, 2.15, and 3.92 for Grades I, II, and III respectively, with an overall AUC of 0.79, close to the present study's AUC of 0.80, confirming good discriminative power.

Furthermore, Taher MY et al. (2022),^[12] established a strong relationship between non-invasive fibrosis indices (APRI, FIB-4) and hemodynamic severity (HVPG > 12 mmHg), supporting the mechanistic link between increasing portal pressure and higher APRI. Faheem HA et al. (2022),^[4] reported a significant step-up in APRI and MELD with variceal grade (AUC 0.79; $p < 0.001$), while Lin X et al. (2024)^[8] concluded that APRI combined with Child–Pugh classification improved prediction accuracy for high-risk varices, reaching sensitivity of 87% and specificity of 70%.

CONCLUSION

The present cross-sectional study involving 71 cirrhotic patients demonstrated that the Aspartate Aminotransferase-to-Platelet Ratio Index (APRI) is a reliable, simple, and cost-effective non-invasive marker for predicting the presence and severity of esophageal varices. The mean APRI values were

significantly higher among patients with varices than those without, and APRI showed a strong positive correlation with the grade of varices on endoscopy. Using a cutoff value of approximately 1.1, the test exhibited good diagnostic performance with high sensitivity and moderate specificity (AUC = 0.80), comparable to previously reported studies. Hence, APRI can effectively stratify cirrhotic patients for endoscopic screening, thereby reducing unnecessary invasive procedures in low-risk groups. However, while APRI is a valuable adjunct, it should complement rather than replace endoscopy for the definitive diagnosis of varices.

Limitations of The Study

1. **Small sample size (N=71)** limits the generalizability of the findings to the broader cirrhotic population.
2. **Single-center design** may introduce selection bias, as patients' etiological and demographic characteristics may not represent the general population.
3. **Cross-sectional nature** precludes assessment of temporal progression of APRI values and their predictive value over time.
4. **Exclusion of patients with prior endoscopic therapy or hepatocellular carcinoma** may have eliminated severe cases, potentially underestimating APRI's predictive range.
5. **Lack of comparison with other non-invasive indices** such as FIB-4 or FibroScan limited the ability to evaluate relative performance.
6. **Inter-observer variability in endoscopic grading** could have introduced subjective bias despite standardized criteria.

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