

DIAGNOSTIC PERFORMANCE OF PLATELET COUNT, SPLEEN SIZE, PLATELET-TO-SPLEEN RATIO, PLATELET-ALBUMIN-BILIRUBIN SCORE, CHILD-TURCOTTE-PUGH SCORE, AND MODEL FOR END-STAGE LIVER DISEASE WITH SODIUM FOR PREDICTION OF ESOPHAGEAL VARICES IN CHRONIC LIVER DISEASE

Uppati Vamsi Krishna^{1*}, Katuri Saikrishna Bharath², Rohit Uppalapu³, Dr. Ravada V S S K Kinneresh⁴

¹Consultant Medical Gastroenterology, Great Eastern Medical School & Hospital (GEMS), Srikakulam, Andhra Pradesh, India

²Junior Resident, Department of General Medicine, Great Eastern Medical School & Hospital (GEMS), Srikakulam, Andhra Pradesh, India

³Junior Resident, Department of General Medicine, Great Eastern Medical School & Hospital (GEMS), Srikakulam, Andhra Pradesh, India

⁴Senior Resident, Department of Dentistry, Great Eastern Medical School & Hospital (GEMS), Srikakulam, Andhra Pradesh, India

*Corresponding Author: Uppati Vamsi Krishna; Email: vamsikrishnauppati@gmail.com

ABSTRACT

Background: Esophageal varices are a major complication of portal hypertension in patients with chronic liver disease. Upper gastrointestinal endoscopy remains the reference method for detection and grading of esophageal varices. Several non-invasive parameters, including platelet count, spleen size, platelet-to-spleen ratio, platelet-albumin-bilirubin score, Child-Turcotte-Pugh score, and Model for End-Stage Liver Disease with sodium, have been evaluated as potential predictors. **Objective:** To evaluate the diagnostic performance of platelet count, spleen size, platelet-to-spleen ratio, platelet-albumin-bilirubin score, Child-Turcotte-Pugh score, and Model for End-Stage Liver Disease with sodium for predicting esophageal varices and large esophageal varices in patients with chronic liver disease. **Methods:** This retrospective observational study included 104 patients with chronic liver disease who underwent upper gastrointestinal endoscopy and had available data for platelet count, spleen size, bilirubin, albumin, Child-Turcotte-Pugh score, and Model for End-Stage Liver Disease with sodium. Esophageal varices were classified from the documented endoscopic findings as absent, small, or large. Platelet-to-spleen ratio was calculated as platelet count divided by spleen diameter. Platelet-albumin-bilirubin score was calculated using the published formula. Receiver operating characteristic analysis was performed for prediction of any esophageal varices and large esophageal varices. **Results:** Among 104 patients, 83 (79.8%) had esophageal varices and 21 (20.2%) had no documented esophageal varices. Large esophageal varices were documented in 26 (25.0%) patients. For prediction of any esophageal varices, the areas under the receiver operating characteristic curve were 0.509 for platelet count, 0.435 for spleen size, 0.495 for platelet-to-spleen ratio, 0.521 for platelet-albumin-bilirubin score, 0.513 for Child-Turcotte-Pugh score, and 0.558 for Model for End-Stage Liver Disease with sodium. For large esophageal varices, the corresponding areas under the receiver operating characteristic curve were 0.614, 0.522, 0.616, 0.390, 0.458, and 0.354, respectively. None of the individual parameters demonstrated robust discrimination for the presence of esophageal varices. Platelet count and platelet-to-spleen ratio showed modest discrimination for large varices, but their confidence intervals were broad. **Conclusion:** In this retrospective cohort, commonly available laboratory, imaging, and liver disease severity parameters demonstrated limited ability to predict endoscopically detected esophageal varices. Platelet count and platelet-to-spleen ratio showed some discriminatory signal for large varices, but their performance was insufficient to support replacement of endoscopic assessment. Prospective validation of composite non-invasive models incorporating liver and spleen stiffness is warranted.

KEYWORDS: chronic liver disease; esophageal varices; portal hypertension; platelet count; spleen size; platelet-to-spleen ratio; platelet-albumin-bilirubin; Model for End-Stage Liver Disease

INTRODUCTION

Portal hypertension is a major complication of advanced chronic liver disease and contributes substantially to the development of esophageal varices, ascites, hepatic encephalopathy, and variceal hemorrhage. Esophageal varices develop as a consequence of increased portal venous pressure and portosystemic collateral formation and are clinically important because variceal hemorrhage is associated with substantial morbidity and mortality [1,2].

Upper gastrointestinal endoscopy remains the reference investigation for identifying and grading esophageal varices. However, endoscopy is invasive, resource-dependent, and may be difficult to perform repeatedly in large populations.

Consequently, considerable interest has developed in non-invasive approaches for identifying patients at risk of clinically significant portal hypertension and varices [1,2].

Thrombocytopenia is frequently observed in portal hypertension and may result from splenic sequestration, hypersplenism, reduced thrombopoietin production, or a combination of these mechanisms. Splenomegaly is another readily available surrogate marker of portal hypertension. The platelet-to-spleen ratio was proposed as a simple combination of these two parameters and demonstrated promising performance in early studies [3-5]. However, subsequent studies have demonstrated variable diagnostic performance across different populations.

The severity of underlying liver dysfunction may also be associated with portal hypertensive complications. The Child-Turcotte-Pugh score incorporates bilirubin, albumin, coagulation parameters, ascites, and hepatic encephalopathy, whereas the Model for End-Stage Liver Disease with sodium incorporates laboratory markers of hepatic and renal dysfunction together with serum sodium [6,7]. These scores primarily assess hepatic disease severity and prognosis rather than directly measuring portal pressure.

The platelet-albumin-bilirubin score incorporates platelet count into the albumin-bilirubin score. It was developed as an objective prognostic score and has subsequently been investigated in patients with cirrhosis and variceal bleeding [8,9]. Because platelet count may provide indirect information regarding portal hypertension, PALBI has also been explored as a potential non-invasive marker of esophageal varices [10].

Current consensus recommendations emphasize the use of validated combinations of non-invasive parameters, particularly liver stiffness, spleen stiffness, and platelet count, rather than relying on a single routine laboratory variable for assessment of clinically significant portal hypertension [1,2].

The present study therefore evaluated and compared platelet count, spleen size, platelet-to-spleen ratio, platelet-albumin-bilirubin score, Child-Turcotte-Pugh score, and Model for End-Stage Liver Disease with sodium against endoscopic findings in a real-world cohort of patients with chronic liver disease.

MATERIALS AND METHODS

Study design and population

This was a retrospective observational study based on a de-identified clinical dataset of patients with chronic liver disease who underwent upper gastrointestinal endoscopy.

The final sample size was 104 patients.

Patients were included if they had chronic liver disease, underwent upper gastrointestinal endoscopy, and had available data for the principal study variables.

Endoscopic assessment

Endoscopic findings were reviewed and categorized into three groups: (1) no esophageal varices; (2) small esophageal varices; and (3) large esophageal varices. The primary outcome was the presence of any esophageal varices, defined as either small or large esophageal varices. The secondary outcome was the presence of large esophageal varices.

Platelet count and spleen size

Platelet count closest to the available endoscopic assessment was used. Values recorded in absolute cells/ μL rather than $\times 10^3/\mu\text{L}$ were converted to $\times 10^3/\mu\text{L}$ before analysis. Spleen diameter was recorded in centimeters according to the available imaging documentation.

Platelet-to-spleen ratio

The platelet-to-spleen ratio was calculated as: Platelet-to-spleen ratio = platelet count ($\times 10^3/\mu\text{L}$) / spleen diameter (cm). Higher values represent a combination of relatively preserved platelet count and smaller spleen size.

Platelet-albumin-bilirubin score

The platelet-albumin-bilirubin score was calculated according to the published formula [8]:

$$\text{PALBI} = 2.02 \times \log_{10}(\text{TBIL}) - 0.37 \times [\log_{10}(\text{TBIL})]^2 - 0.04 \times \text{ALB} - 3.48 \times \log_{10}(\text{PLT}) + 1.01 \times [\log_{10}(\text{PLT})]^2$$

Total bilirubin (TBIL) was expressed in $\mu\text{mol/L}$, albumin (ALB) in g/L , and platelet count (PLT) in $10^9/\text{L}$.

Child-Turcotte-Pugh score

Child-Turcotte-Pugh scores were obtained from the analysis-ready dataset and incorporated bilirubin, albumin, coagulation parameters, ascites, and hepatic encephalopathy according to the established scoring system [6].

Model for End-Stage Liver Disease with sodium

Model for End-Stage Liver Disease with sodium values were obtained from the analysis-ready dataset. This score was developed for prognostic risk stratification in advanced liver disease and incorporates renal and hepatic laboratory parameters together with serum sodium [7].

Statistical analysis

Continuous variables were summarized as median and interquartile range because several variables demonstrated non-normal distributions. Categorical variables were summarized as frequencies and percentages.

The Mann-Whitney U test was used for comparison of continuous variables between groups.

Receiver operating characteristic curves were generated to evaluate the diagnostic performance of platelet count, spleen size, platelet-to-spleen ratio, platelet-albumin-bilirubin score, Child-Turcotte-Pugh score, and Model for End-Stage Liver Disease with sodium.

Two outcomes were analyzed: (1) any esophageal varices versus no esophageal varices; and (2) large esophageal varices versus all other patients.

For platelet count and platelet-to-spleen ratio, the direction of the predictor was reversed for ROC analysis because lower values are biologically associated with greater portal hypertension.

The area under the receiver operating characteristic curve was used as the principal measure of discrimination. Exploratory optimal thresholds were identified using the Youden index. Because of the modest sample size and relatively small number of patients with large varices, threshold estimates were considered exploratory rather than clinically validated.

A two-sided P value of <0.05 was considered statistically significant.

RESULTS

After exclusion of three linked duplicate or superseded records, 104 patients were included in the final analysis.

Esophageal varices were documented in 83 patients (79.8%), while 21 patients (20.2%) had no documented esophageal varices. Small esophageal varices were documented in 57 patients (54.8%) and large esophageal varices in 26 patients (25.0%). The high prevalence of varices reflects the fact that this was a clinically selected cohort of patients with chronic liver disease undergoing upper gastrointestinal endoscopy rather than a general screening population.

The cohort predominantly consisted of patients with advanced liver disease. Two patients were classified as Child-Turcotte-Pugh class A, 37 as class B, and 65 as class C. Among patients without esophageal varices, nine were Child-Turcotte-Pugh class B and 12 were class C. Among patients with esophageal varices, two were class A, 28 were class B, and 53 were class C.

Comparison of non-invasive parameters

The median platelet count was $107 \times 10^3/\mu\text{L}$ (interquartile range [IQR], 70-187) in patients without esophageal varices and $119 \times 10^3/\mu\text{L}$ (IQR, 68-183) in patients with any esophageal varices ($P=0.897$).

Median spleen size was 14 cm in both groups (IQR, 13-14 cm in both groups; $P=0.308$).

The median platelet-to-spleen ratio was 8.23 (IQR, 5.23-13.36) in patients without varices and 8.57 (IQR, 5.00-13.58) in patients with any varices ($P=0.942$).

Median PALBI was -1.53 (IQR, -1.88 to -1.08) in patients without varices and -1.46 (IQR, -1.82 to -1.24) in patients with any varices ($P=0.771$).

Median Child-Turcotte-Pugh score was 10 in both groups (IQR, 9-11; $P=0.856$).

Median Model for End-Stage Liver Disease with sodium was 18 (IQR, 15-22) in patients without varices and 19 (IQR, 15-23) in patients with any varices ($P=0.417$).

Diagnostic performance for any esophageal varices

Platelet count demonstrated an area under the receiver operating characteristic curve of 0.509 (95% CI, 0.364-0.647). Spleen size demonstrated an area under the receiver operating characteristic curve of 0.435 (95% CI, 0.302-0.571). The platelet-to-spleen ratio demonstrated an area under the receiver operating characteristic curve of 0.495 (95% CI, 0.350-0.637).

PALBI demonstrated an area under the receiver operating characteristic curve of 0.521 (95% CI, 0.364-0.676). Child-Turcotte-Pugh score demonstrated an area under the receiver operating characteristic curve of 0.513 (95% CI, 0.379-0.656). Model for End-Stage Liver Disease with sodium demonstrated the highest numerical area under the receiver operating characteristic curve at 0.558 (95% CI, 0.423-0.688).

Thus, none of the evaluated parameters demonstrated clinically useful discrimination for the presence of any esophageal varices.

Diagnostic performance for large esophageal varices

Patients with large esophageal varices had a lower median platelet count than those without large varices: $86.5 \times 10^3/\mu\text{L}$ (IQR, 54-140.75) versus $130.5 \times 10^3/\mu\text{L}$ (IQR, 72-189.75), respectively ($P=0.083$).

The median platelet-to-spleen ratio was also lower in patients with large varices: 6.41 (IQR, 3.75-10.83) versus 9.21 (IQR, 5.38-14.40), respectively ($P=0.077$).

Median MELD-Na was lower among patients with large varices than among those without large varices: 16.5 (IQR, 13.25-19.75) versus 20 (IQR, 15.25-23.75), respectively ($P=0.026$).

For large esophageal varices, platelet count demonstrated an area under the receiver operating characteristic curve of 0.614 (95% CI, 0.482-0.736). The platelet-to-spleen ratio demonstrated an area under the receiver operating characteristic curve of 0.616 (95% CI, 0.486-0.739). Spleen size demonstrated an area under the receiver operating characteristic curve of 0.522 (95% CI, 0.410-0.634).

PALBI demonstrated an area under the receiver operating characteristic curve of 0.390 (95% CI, 0.271-0.516). Child-Turcotte-Pugh score demonstrated an area under the receiver operating characteristic curve of 0.458 (95% CI, 0.334-0.581). Model for End-Stage Liver Disease with sodium demonstrated an area under the receiver operating characteristic curve of 0.354 (95% CI, 0.238-0.478).

Although platelet count and platelet-to-spleen ratio showed the highest numerical AUCs for large varices, the confidence intervals were broad and included values compatible with poor discrimination.

Table 1. Clinical characteristics and non-invasive parameters according to the presence of esophageal varices

Variable	No esophageal varices (n=21)	Any esophageal varices (n=83)	P value
Platelet count, $\times 10^3/\mu\text{L}$	107 (70-187)	119 (68-183)	0.897
Spleen size, cm	14 (13-14)	14 (13-14)	0.308
Platelet-to-spleen ratio	8.23 (5.23-13.36)	8.57 (5.00-13.58)	0.942
PALBI	-1.53 (-1.88 to -1.08)	-1.46 (-1.82 to -1.24)	0.771
Child-Turcotte-Pugh score	10 (9-11)	10 (9-11)	0.856
MELD-Na	18 (15-22)	19 (15-23)	0.417

Values are median (interquartile range). PALBI: platelet-albumin-bilirubin; MELD-Na: Model for End-Stage Liver Disease with sodium.

Table 2. Diagnostic performance of non-invasive parameters for esophageal varices

Parameter	Any esophageal varices, AUC (95% CI)	Large esophageal varices, AUC (95% CI)
Platelet count	0.509 (0.364-0.647)	0.614 (0.482-0.736)
Spleen size	0.435 (0.302-0.571)	0.522 (0.410-0.634)
Platelet-to-spleen ratio	0.495 (0.350-0.637)	0.616 (0.486-0.739)
PALBI	0.521 (0.364-0.676)	0.390 (0.271-0.516)
Child-Turcotte-Pugh score	0.513 (0.379-0.656)	0.458 (0.334-0.581)
MELD-Na	0.558 (0.423-0.688)	0.354 (0.238-0.478)

AUC: area under the receiver operating characteristic curve; CI: confidence interval; PALBI: platelet-albumin-bilirubin; MELD-Na: Model for End-Stage Liver Disease with sodium.

Table 3. Exploratory thresholds for prediction of large esophageal varices

Parameter	Exploratory threshold	Sensitivity	Specificity
Platelet count	$\leq 57 \times 10^3/\mu\text{L}$	34.6%	87.2%
Platelet-to-spleen ratio	≤ 4.07	34.6%	88.5%
Spleen size	≥ 14 cm	61.5%	44.9%
PALBI	≥ -2.10	96.2%	6.4%
Child-Turcotte-Pugh score	≥ 10	69.2%	39.7%
MELD-Na	≥ 11	100.0%	1.3%

Thresholds were derived using the Youden index and are exploratory only. They should not be interpreted as validated clinical cutoffs.

DISCUSSION

The present study evaluated six readily available clinical and laboratory parameters for prediction of endoscopically detected esophageal varices in patients with chronic liver disease. The principal finding was that none of the individual parameters demonstrated robust discrimination for the presence of any esophageal varices.

For any esophageal varices, all evaluated AUCs were close to 0.50. MELD-Na demonstrated the highest numerical AUC at 0.558, but this level of discrimination is insufficient for clinical decision-making. Similarly, PALBI and Child-Turcotte-Pugh score showed AUCs of 0.521 and 0.513, respectively.

The findings are relevant because platelet count and spleen size are frequently used as practical surrogates for portal hypertension. Thrombocytopenia may reflect hypersplenism and altered thrombopoietin physiology, whereas splenic enlargement reflects increased portal pressure and congestion. However, neither variable is specific for clinically significant portal hypertension, and both may be influenced by multiple other factors.

The platelet-to-spleen ratio was initially proposed as a simple non-invasive method for predicting esophageal varices. Giannini et al. reported promising results in the original study and subsequent prospective validation, including a commonly cited cutoff of 909 [3,4]. Follow-up validation also suggested that the ratio could help identify patients at low risk of varices [5]. However, subsequent studies have demonstrated heterogeneous diagnostic performance, particularly when applied to populations with different etiologies and stages of chronic liver disease.

In the present cohort, the platelet-to-spleen ratio did not discriminate patients with any esophageal varices, with an AUC of 0.495. For large varices, the AUC increased to 0.616, suggesting a modest signal. Nevertheless, the 95% confidence interval extended from 0.486 to 0.739, indicating substantial uncertainty around the estimate.

The apparent discrepancy between the present findings and earlier studies may reflect several factors. The current cohort was enriched for patients with established portal hypertension and had a high prevalence of esophageal varices. The restricted distribution of spleen measurements, with a median of approximately 14 cm in both groups, may also have reduced the discriminatory contribution of spleen size. In addition, the dataset represents routine clinical documentation rather than a prospectively standardized research cohort.

PALBI was included because it incorporates platelet count into the albumin-bilirubin framework. PALBI was originally developed as an objective score for liver disease prognostication and subsequently evaluated in patients with cirrhosis and variceal bleeding [8,9]. Studies have shown that PALBI may provide prognostic information in patients with acute variceal bleeding [9]. However, prognostic performance does not necessarily translate into diagnostic performance for the presence of esophageal varices.

In our cohort, PALBI demonstrated an AUC of 0.521 for any esophageal varices and 0.390 for large esophageal varices. These findings suggest that PALBI should not be interpreted as a standalone diagnostic test for esophageal varices. The poor performance for large varices also illustrates the difference between a score designed for overall liver disease assessment and a parameter specifically measuring portal hypertension.

The Child-Turcotte-Pugh score demonstrated similarly limited discrimination. Although CTP class is associated with increasing severity of chronic liver disease, it incorporates subjective variables such as ascites and hepatic encephalopathy and was not specifically developed as a portal hypertension measurement [6].

MELD-Na demonstrated the highest numerical AUC for any varices, but the AUC of 0.558 remained inadequate for clinically meaningful discrimination. Interestingly, MELD-Na showed an AUC below 0.50 for large varices in this cohort. This inverse association should not be interpreted as evidence that lower MELD-Na protects against large varices. Rather, it likely reflects the characteristics and small sample size of this selected cohort. The lower median MELD-Na among patients with large varices emphasizes that liver disease mortality scores and variceal burden are related but distinct clinical constructs.

An important observation was that the platelet count and platelet-to-spleen ratio showed modest discrimination for large varices, with AUCs of 0.614 and 0.616, respectively. The median platelet count was also lower among patients with large varices. These findings are biologically plausible and support the continued use of thrombocytopenia as one component of portal hypertension assessment. However, the sensitivity of the exploratory platelet threshold was only 34.6%, making it unsuitable as a standalone screening threshold.

Contemporary portal hypertension guidance increasingly favors validated non-invasive combinations incorporating liver stiffness, spleen stiffness, and platelet count rather than individual routine laboratory markers [1,2]. Spleen stiffness in particular can provide information more directly related to portal hypertension than spleen diameter alone. Studies combining Baveno criteria with spleen stiffness have demonstrated improved identification of clinically significant portal hypertension [11].

The current study therefore supports an important practical distinction: readily available parameters may contribute to clinical risk assessment but should not automatically be interpreted as substitutes for endoscopy or validated non-invasive portal hypertension algorithms.

Strengths

The study has several strengths. First, multiple commonly used non-invasive parameters were evaluated within the same patient cohort. Second, all patients had documented upper gastrointestinal endoscopy, providing a clinical reference outcome. Third, spleen measurements were available, allowing direct assessment of the platelet-to-spleen ratio. Fourth, PALBI was evaluated alongside CTP and MELD-Na, allowing comparison between a score incorporating platelet count and conventional liver disease severity scores. Finally, both any esophageal varices and large esophageal varices were examined as separate outcomes.

LIMITATIONS

The sample size was relatively small, particularly for the large-varix subgroup. Consequently, confidence intervals around the AUC estimates were wide.

This was a retrospective study based on routine clinical documentation from a single clinical setting. The findings may therefore not be generalizable to other populations.

The cohort was clinically selected for endoscopic assessment and had a high prevalence of esophageal varices. This spectrum of disease may differ substantially from an unselected population of patients with compensated chronic liver disease.

The relatively narrow range of spleen measurements may have limited the discriminatory value of spleen size and PSR. Liver stiffness and spleen stiffness measurements were unavailable. These are important contemporary non-invasive markers of clinically significant portal hypertension and would have provided a more meaningful comparison with platelet-based measures.

The dataset included heterogeneous chronic liver disease etiologies and stages. The small sample size prevented reliable subgroup analyses according to etiology.

The exploratory thresholds derived from this dataset were not externally validated and should not be used as clinical decision thresholds.

CONCLUSIONS

In this retrospective cohort of 104 patients with chronic liver disease, platelet count, spleen size, platelet-to-spleen ratio, platelet-albumin-bilirubin score, Child-Turcotte-Pugh score, and Model for End-Stage Liver Disease with sodium demonstrated limited discrimination for the presence of esophageal varices.

Platelet count and platelet-to-spleen ratio showed modest discriminatory signal for large esophageal varices, but the uncertainty surrounding these estimates and their limited sensitivity do not support their use as standalone replacements for endoscopic assessment. Larger prospective studies incorporating liver stiffness, spleen stiffness, platelet count, and validated composite algorithms are needed to identify patients who can safely avoid screening endoscopy.

DECLARATIONS

Human subjects: The study involved human clinical data but no intervention or prospective experimentation.

Animal subjects: No animal subjects were involved.

Conflict of interest: The authors declare that they have no competing interests.

Funding: No specific funding was received for this study.

Acknowledgements

The authors acknowledge the medical gastroenterology and endoscopy teams involved in the evaluation and management of the patients included in this retrospective analysis.

REFERENCES

1. de Franchis R: Baveno VII - renewing consensus in portal hypertension. *J Hepatol.* 2022, 76:959-974. 10.1016/j.jhep.2021.12.022
2. Garcia-Tsao G, Abraldes JG, Berzigotti A, Bosch J: Portal hypertensive bleeding in cirrhosis: risk stratification, diagnosis, and management: 2016 practice guidance by the American Association for the Study of Liver Diseases. *Hepatology.* 2017, 65:310-335. 10.1002/hep.28906
3. Giannini E, Botta F, Borro P, et al.: Platelet count/spleen diameter ratio: proposal and validation of a non-invasive parameter to predict the presence of oesophageal varices in patients with liver cirrhosis. *Gut.* 2003, 52:1200-1205. 10.1136/gut.52.8.1200
4. Giannini EG, Zaman A, Kreil A, et al.: Platelet count/spleen diameter ratio for the noninvasive diagnosis of esophageal varices: results of a multicenter, prospective, validation study. *Am J Gastroenterol.* 2006, 101:2511-2519. 10.1111/j.1572-0241.2006.00874.x
5. Giannini EG, Botta F, Borro P, et al.: Application of the platelet count/spleen diameter ratio to rule out the presence of oesophageal varices in patients with cirrhosis: a validation study based on follow-up. *Dig Liver Dis.* 2005, 37:779-785. 10.1016/j.dld.2005.05.007
6. Pugh RN, Murray-Lyon IM, Dawson JL, Pietroni MC, Williams R: Transection of the oesophagus for bleeding oesophageal varices. *Br J Surg.* 1973, 60:646-649. 10.1002/bjs.1800600817
7. Biggins SW, Kim WR, Terrault NA, et al.: Evidence-based incorporation of serum sodium concentration into MELD. *Gastroenterology.* 2006, 130:1652-1660. 10.1053/j.gastro.2006.02.010
8. Roayaie S, Jibara G, Berhane S, et al.: PALBI-an objective score based on platelets, albumin & bilirubin stratifies HCC patients undergoing resection & ablation better than Child's classification. *Hepatology.* 2015, 62:631A-632A.
9. Elshaarawy O, Allam N, Abdelsameea E, Gomaa A, Waked I: Platelet-albumin-bilirubin score - a predictor of outcome of acute variceal bleeding in patients with cirrhosis. *World J Hepatol.* 2020, 12:99-107. 10.4254/wjgh.v12.i3.99

10. Malik A, et al.: The utility of the platelet-albumin-bilirubin score as a non-invasive predictor of esophageal varices and variceal hemorrhage in patients with liver cirrhosis compared to Child-Turcotte-Pugh and Model of End-Stage Liver Disease-sodium scores. *Cureus*. 2024, 16:e62577. 10.7759/cureus.62577
11. Dajti E, Ravaioli F, Marasco G, et al.: A combined Baveno VII and spleen stiffness algorithm to improve the noninvasive diagnosis of clinically significant portal hypertension in patients with compensated advanced chronic liver disease. *Am J Gastroenterol*. 2022, 117:1825-1833. 10.14309/ajg.0000000000001887