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Original Article

Predictive value of platelets-to-spleen diameter ratio for esophagogastric varices in hepatitis B virus-induced cirrhosis

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Abstract: Objective: To evaluate the predictive efficacy of the platelets-to-spleen diameter ratio (PSDR) for developing esophagogastric varices (EV) in patients with cirrhosis due to hepatitis B virus (HBV). Methods: We conducted a retrospective cohort study using data from patients treated for HBV induced cirrhosis at Xi'an No. 3 Hospital, the Affiliated Hospital of Northwest University, from June 2020 to August 2023. Patients were categorized into two groups based on endoscopic evidence of EV: an EV group and a non-EV group. Clinical, sonographic, and hematological findings were compared within and between these groups. Stratified analyses based on the severity of varices were performed, and multivariate logistic regression was used to identify predictors of EV. Receiver Operating Characteristic (ROC) curve analysis assessed the diagnostic accuracy of PSDR in predicting EV. Results: The study included 139 patients diagnosed with HBV induced cirrhosis, divided into an EV group (86 patients, with 48 low-risk and 38 high-risk) and a non-EV group (53 patients). Significant differences were found between the groups in several parameters: Child-Pugh classification, Child-Pugh score, portal vein diameter, hepatic vein deceleration index, spleen thickness, and PSDR (all P<0.001). These variables also varied significantly across the different risk categories within the EV group (all P<0.001). Multivariate logistic regression indicated PSDR as an independent predictor of EV development (Odds Ratio [OR]=3.569, 95% Confidence Interval [CI]: 0.970-1.001, P<0.001). ROC curve analysis showed that PSDR had an Area Under the Curve (AUC) of 0.865 (95% CI: 0.764-0.965) for predicting EV, with an optimal threshold of 1013.2, achieving 88.46% sensitivity and 69.23% specificity. For high-risk EV, PSDR showed an AUC of 0.763 (95% CI: 0.670-0.856), with a threshold of 883.5, sensitivity of 79.17%, and specificity of 54.17%. Conclusion: The PSDR is a significant risk marker and demonstrates strong predictive utility for both the presence and severity of EV in patients with HBV-induced cirrhosis. PSDR provides a valuable, non-invasive diagnostic tool for anticipating the development of EV in this patient population.

Keywords: Hepatitis B virus-induced cirrhosis, esophagogastric varices, platelet spleen diameter ratio

Introduction

Esophagogastric variceal hemorrhage is the most frequent and severe complication of portal hypertension in patients with hepatitis B virus (HBV)-induced cirrhosis. The cirrhotic process leads to significant structural and fibrotic changes in the liver, impairing its detoxification and metabolic functions [1]. This alteration typically results in portal hypertension and the subsequent formation of esophagogastric varices (EV). Literature indicates that 40-50% of patients with cirrhosis develop EV during the natural course of the disease, and approximately 8% of cases with mild varices progress to moderate or severe stages, which are associated with an increased risk of bleeding [2]. The 6-week mortality rate for these severe cases is alarmingly high, at about 20% [3]. Early prevention and intervention are crucial in improving the prognosis of patients with HBV-induced cirrhosis by preventing the development of EV.

Esophagogastroduodenoscopy (EGD) is the current gold standard for detecting EV. However, EGD is an invasive procedure that requires sophisticated equipment and technical expertise, leading to high cost and potential proce-

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dural complications [4]. Introduced by Giannini et al. in 2003 [5], the platelet-to-spleen diameter ratio (PSDR) has gained traction as a non-invasive predictor of EV. Subsequent studies [6, 7] have confirmed a strong correlation between PSDR and the severity of chronic EV. Despite these insights, research on the predictive value of PSDR in HBV induced cirrhosis remains limited. This study aims to perform a retrospective analysis to assess the prognostic value of PSDR in the development of EV among HBV-induced cirrhosis patients, expanding the range of non-invasive diagnostic tools for clinical screening of these varices in this demographic.

Materials and methods

Study population

This retrospective analysis collected clinical data from patients diagnosed with HBV induced cirrhosis admitted to Xi'an No. 3 Hospital, the Affiliated Hospital of Northwest University, between June 2020 and August 2023. Inclusion criteria included: (1) a documented history of chronic HBV leading to cirrhosis; (2) completion of EGD; (3) availability of comprehensive clinical data.

Exclusion criteria were: (1) previous management of esophageal varices, including endoscopic band ligation or transjugular intrahepatic portosystemic shunt; (2) other concurrent viral hepatitis (excluding HBV), alcoholic liver disease, non-alcoholic fatty liver disease, or autoimmune liver disorders; (3) co-existing hepatocellular carcinoma or other malignancies; (4) incomplete patient records. The study was approved by the ethics committee of Xi'an No. 3 Hospital, the Affiliated Hospital of Northwest University complied with the Declaration of Helsinki guidelines, and the need for informed consent was waived by the ethics committee.

Methods

Grouping: Patients underwent diagnostic evaluation using electronic gastroscopy based on the latest clinical guidelines for (EV) [8]. Subjects were divided into two groups: those with EV and those without. For patients with varices, the location, morphology, and presence of red signs, indicating a higher risk of bleeding, were meticulously recorded. Varices were classified as mild (G1), moderate (G2), or severe (G3) based on the following criteria: (1) Mild (G1): Varices that were straight or minimally tortuous, without red signs; (2) Moderate (G2): Varices displaying red signs or appearing serpiginous without red signs; (3) Severe (G3): Varices with a serpiginous, nodular, or tumorous appearance, with or without red signs. Varices classified as moderate or severe were considered high-risk for bleeding, while mild varices were deemed low-risk. The patient selection and stratification process is detailed in **Figure 1**.

Data collection: Comprehensive patient data were meticulously collated from electronic health records, including demographic information (gender, age), hepatic functional status as indicated by Child-Pugh classification and score, comorbidities, presence of ascites, and portal vein thrombosis. Ultrasonographic and laboratory parameters recorded at admission were also included. Ultrasonography was performed using a color Doppler system, operating at a frequency range of 4.0-5.5 MHz, after a fasting period of 6-8 hours. Measurements included portal vein diameter (PVD) and hepatic vein deceleration index (HV-DI). Spleen dimensions were assessed as follows: spleen diameter from the apex of the upper margin to the nadir of the lower margin, and spleen thickness measured orthogonally from the splenic hilum to the outer boundary. Key laboratory values included lymphocyte and platelet counts, serum albumin levels, and various indicators of hepatic function at the time of hospital admission.

Calculation of PSDR: The diameter and thickness of the spleen were detected and measured using an ultrasonic diagnostic instrument (GE Logiq S8, USA). The spleen diameter was measured from the highest to the lowest point. Platelet counts were obtained using an automatic blood analyzer at admission. The formula for calculating the PSDR is as follows: PSDR = Platelet Count ($\times 10^9$/L)/Spleen Diameter (mm).

Statistical analysis

Data were statistically analyzed using the Statistical Package for the Social Sciences, version 23.0. Continuous variables following a nor-

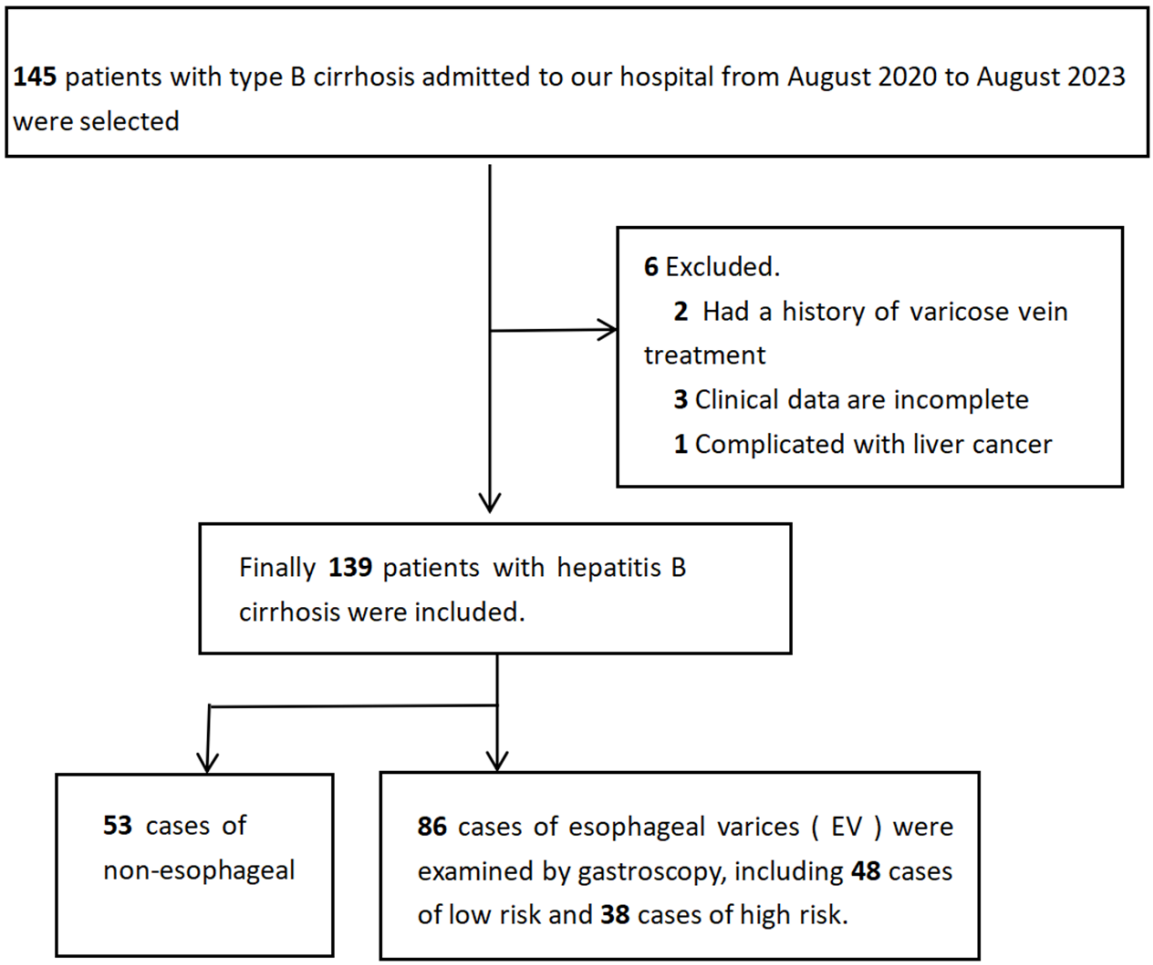

Figure 1. Flow chart.

mal distribution were presented as means ± standard deviations and analyzed between groups using the independent samples t-test. Categorical variables were reported as frequency counts and percentages [n (%)] and compared using the Chi-square test. To identify factors influencing the development of EV in patients with HBV-induced cirrhosis, multivariate logistic regression analysis was employed.

The predictive accuracy of the PSDR for EV was evaluated using Receiver Operating Characteristic (ROC) curve analysis, performed with GraphPad Prism version 8.0. The Area Under the Curve (AUC) was used to assess predictive efficacy, interpreted as follows: ≤0.50 indicated no predictive value; >0.50 to ≤0.70 low predictive value; >0.70 to ≤0.90 good predictive value; and >0.90 high predictive value. A *P*-value of <0.05 was used to determine significance in all analyses.

Results

Comparison of clinical data between EV and non-EV groups

In our cohort of 139 HBV induced cirrhosis patients, 86 were diagnosed with EV, categorized into 48 low-risk and 38 high-risk cases, while 53 were identified as non-EV. Significant differences were observed in Child-Pugh liver function grades and scores between the EV and non-EV groups (both P<0.05). However, other data such as gender, age, and underlying diseases did not show significant differences between the groups (all P>0.05), as detailed in **Table 1**.

Comparison of ultrasonographic and laboratory data between EV and non-EV groups

Statistical analysis revealed significant differences in PVD, HV-DI, spleen thickness, and

Table 1. Comparison of clinical data between the EV group and non-EV group

Indicator	EV groups (n=86)	Non-EV groups (n=53)	x^2/t	*P*
Age (years, $\bar{x} \pm s$)	57.89±9.88	56.64±10.62	0.704	0.483
Gender [n (%)]				
Male	55 (63.95)	34 (64.15)	0.001	0.981
Female	31 (36.05)	19 (35.85)		
Child-Pugh Liver Function Grade [n (%)]				
A	22 (25.58)	35 (66.04)	28.712	<0.001
B	41 (47.67)	18 (33.96)		
C	23 (26.74)	0 (0.00)		
Child-Pugh Score (points, $\bar{x} \pm s$)	8.77±0.81	6.35±1.26	13.791	<0.001
Underlying Disease [n (%)]				
Yes	35 (40.70)	18 (33.96)	0.631	0.427
No	51 (59.30)	35 (66.04)		
Ascites [n (%)]				
Yes	48 (55.81)	22 (45.41)	2.684	0.101
No	38 (44.19)	31 (58.49)		
Portal Vein Thrombosis [n (%)]				
Yes	11 (12.79)	5 (9.43)	0.363	0.547
No	75 (87.21)	48 (90.57)		

EV: esophagogastric varices.

Table 2. Comparison of ultrasonographic and laboratory data between EV and non-EV groups ($\bar{x} \pm s$)

Indicator	EV groups (n=86)	Non-EV groups (n=53)	*t*	*P*
PVD (cm)	1.62±0.41	1.39±0.32	3.481	0.001
HV-DI (cm/s)	0.45±0.18	0.27±0.09	6.771	<0.001
Splenic thickness (mm)	38.61±2.47	35.33±2.83	7.190	<0.001
Albumin (g/L)	34.11±1.87	34.39±1.95	0.844	0.400
Glutamate aminotransferase (U/L)	21.63±4.38	23.27±6.57	1.766	0.080
Aspartate aminotransferase (U/L)	29.59±5.26	30.62±5.91	1.069	0.287
Platelet-spleen diameter ratio	841.55±89.74	1203.65±113.27	20.875	<0.001

EV: esophagogastric varices; PVD: portal vein diameter; HV-DI: hepatic vein deceleration index.

PSDR between the EV and non-EV groups (all P<0.05). In contrast, albumin levels and liver enzymes (alanine aminotransferase and aspartate aminotransferase) did not differ significantly between these groups (all P>0.05), as detailed in **Table 2**.

Comparison of clinical data between low-risk and high-risk EV groups

Significant variations were noted in Child-Pugh liver function grades and scores between low-risk and high-risk EV groups (both P<0.05). However, no significant differences were observed in gender, age, underlying diseases, or other clinical data between these groups (all P>0.05), as depicted in **Table 3**.

Comparison of ultrasonographic and laboratory data between low-risk and high-risk EV groups

There were significant differences in PVD, HV-DI, spleen thickness, and PSDR between the low-risk and high-risk EV groups (all P< 0.05). Albumin levels and alanine aminotransferase and aspartate aminotransferase levels did not show significant differences between the two groups (all P>0.05), as illustrated in **Table 4**.

Table 3. Comparison of clinical data between low-risk and high-risk EV groups

Indicator	Low-Risk groups (n=48)	High-Risk groups (n=38)	x^2/t	*P*
Age (years, $\bar{x} \pm s$)	56.84±8.12	58.01±9.56	0.613	0.541
Gender [n (%)]				
Male	28 (58.33)	25 (65.79)	0.499	0.480
Female	20 (41.67)	13 (34.21)		
Child-Pugh Liver Function Grade [n (%)]				
A	17 (35.42)	5 (13.16)	7.873	0.020
B	27 (56.25)	14 (36.84)		
C	4 (8.33)	19 (50.00)		
Child-Pugh Score (points, $\bar{x} \pm s$)	8.03±0.79	8.95±1.36	3.927	<0.001
Comorbid underlying disease [n (%)]				
Yes	19 (39.58)	16 (42.11)	0.056	0.813
No	29 (60.42)	22 (57.89)		
Ascites [n (%)]				
Yes	26 (54.17)	22 (57.89)	0.120	0.730
No	22 (45.83)	16 (42.11)		
Portal vein thrombosis [n (%)]				
Yes	6 (12.50)	5 (13.16)	0.008	0.928
No	42 (87.50)	33 (86.84)		

EV: esophagogastric varices.

Table 4. Comparison of ultrasonographic and laboratory data between low-risk and high-risk EV groups

Indicator	Low-Risk groups (n=48)	High-Risk groups (n=38)	*t*	*P*
PVD (cm)	1.12±0.35	1.72±0.29	8.504	<0.001
HV-DI (cm/s)	0.37±0.12	0.53±0.10	6.601	<0.001
Splenic thickness (mm)	34.49±2.33	38.27±2.97	6.616	<0.001
Albumin (g/L)	34.45±1.92	34.28±2.28	0.375	0.708
Glutamate aminotransferase (U/L)	21.29±3.56	22.85±4.89	1.711	0.091
Aspartate aminotransferase (U/L)	29.41±5.03	30.15±5.68	0.640	0.524
Platelet-spleen diameter ratio	1015.84±90.15	782.14±102.37	11.243	<0.001

PVD: portal vein diameter; HV-DI: hepatic vein.

Multifactorial logistic regression analysis for the development of EV in HBV-induced cirrhosis patients

Using variables with significant differences from the univariate analysis (as shown in **Table 5**) as independent variables, and considering the presence of EV as the dependent variable (non-EV=0, EV=1), a multifactorial logistic regression analysis was conducted. The results identified PSDR as an independent risk factor for the development of EV in patients with HBV-induced cirrhosis (P<0.001), as presented in **Table 6**.

ROC curve analysis for PSDR in predicting the development of EV in HBV induced cirrhosis patients

ROC curve analysis demonstrated that the PSDR predicts the development of EV in patients with HBV induced cirrhosis, achieving an area under the curve (AUC) of 0.865 (95% CI: 0.764-0.965). The optimal cutoff value was

Table 5. Assignment of independent variables

Variable	Assignment
Child-Pugh Liver Function Grade	A grade =1, B grade =2, C grade =3
Child-Pugh Score	Original values used
PVD	Original values used
HV-DI	Original values used
Spleen Thickness	Original values used
Platelet to Spleen Diameter Ratio	Original values used

PVD: portal vein diameter; HV-DI: hepatic vein.

Table 6. Multifactorial logistic regression analysis for the development of esophagogastric varices in hepatitis B cirrhosis patients

Factor	*β*	*SE*	Ward χ^2	*P*	*OR*	*95% CI*
Child-Pugh Liver Function Grade	0.283	0.204	3.432	0.061	1.923	0.890-1.979
Child-Pugh Score	0.133	0.119	3.068	0.652	1.245	0.904-1.442
PVD	1.207	0.873	1.297	0.077	1.913	0.604-18.514
HV-DI	0.066	0.049	1.297	0.198	1.803	0.970-1.176
Spleen Thickness	0.112	0.083	1.297	0.182	1.806	0.950-1.316
Platelet to Spleen Diameter Ratio	-0.015	0.008	1.297	<0.001	3.569	0.970-1.001

PVD: portal vein diameter; HV-DI: hepatic vein.

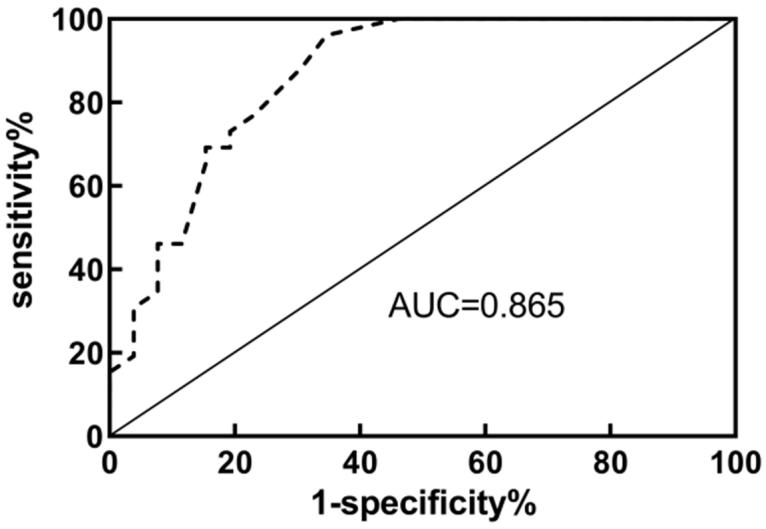

Figure 2. ROC curve for platelet-spleen diameter ratio in predicting the development of esophagogastric varices in hepatitis B cirrhosis patients. AUC: area under curve.

1013.2, yielding a sensitivity of 88.46% and a specificity of 69.23%, as illustrated in **Figure 2**.

ROC curve analysis for PSDR in predicting the development of high-risk EV in HBV-induced cirrhosis patients

Further ROC analysis indicated that PSDR also predicts the development of high-risk EV in these patients, with an AUC of 0.763 (95% CI: 0.670-0.856). The optimal cutoff value was 883.5, achieving a sensitivity of 79.17% and a specificity of 54.17%, as shown in **Figure 3**.

Discussion

EVs are a frequent and clinically significant complication in advanced stages of cirrhosis [9], posing a substantial risk to patient survival. This complication necessitates prompt and focused management in the routine care of patients with cirrhosis. EGD is the recognized gold standard for diagnosing EV [10], enabling detailed assessment of their extent, severity, and the presence of signs indicative of an increased bleeding risk [11]. However, EGD has limitations due to its invasive nature, which can cause discomfort and, by its procedural nature, may induce hemorrhage in patients predisposed to variceal bleeding, exacerbating their condition. Given these challenges, there is an increasing drive within the medical community to develop and utilize non-invasive diagnos-

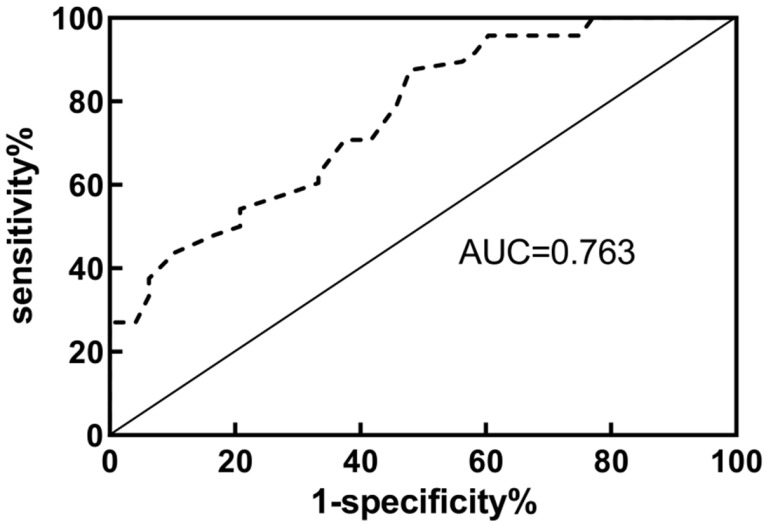

Figure 3. ROC curve for platelet-spleen diameter ratio in predicting the development of high-risk esophagogastric varices in hepatitis B cirrhosis patients. AUC: area under curve.

tic methods for assessing the risk of EV in individuals with HBV-induced cirrhosis. Biochemical blood assays provide multiple advantages for clinical diagnosis across various pathologies, including minimal invasiveness, high reproducibility, rapid turnaround times, and simple sample collection [12, 13]. Previous research [14] has consistently shown reduced platelet counts in patients with HBV-induced cirrhosis, which is closely associated with the development of EV. In cirrhosis, hepatic impairment leads to increased portal pressure, resulting in portal hypertension. This condition impedes venous return from the portal venous system, clinically manifesting as EVs and collateral vessel proliferation in the abdominal wall. Platelets play a crucial role in hemostatic processes, and their depletion increases bleeding risks, further complicating variceal conditions [15].

Studies by Jamil [16] and others have confirmed a correlation between reduced platelet counts and portal hypertension, a precursor to esophageal varices. Additionally, Park [17] and colleagues identified a relationship between platelet levels and the development of EVs through univariate analysis, though this association was not supported by multivariate analysis. HBV-induced cirrhosis often leads to splenomegaly, which is directly relevant to EV. Splenomegaly increases splenic blood flow, contributing to vascular dilatation and further portal hypertension. It also shortens the lifespan of platelets, causing thrombocytopenia. The PSDR has been recognized as a non-invasive, efficient metric for assessing the risk of EV in patients with HBV induced cirrhosis. Previous studies [18] have highlighted a significant correlation between this ratio and the severity of cirrhotic varices. A comprehensive meta-analysis [19] involving 49 studies reported the sensitivity of the PSDR for diagnosing EV and high-risk varices at 78% and 67%, respectively, highlighting its substantial prognostic value. In this study, both univariate and multivariate logistic regression analyses consistently identified the PSDR as an independent predictor of the development of EV in patients with HBV-induced cirrhosis. This finding supports existing literature [20], emphasizing the strong correlation between this ratio and the incidence of EV. Furthermore, ROC curve analysis demonstrated that the PSDR achieved an AUC of 0.865, indicating its efficacy in predicting the development of EV in this patient group. A PSDR below 1013.2 was associated with an increased likelihood of developing EV.

Additionally, the study explored the utility of this ratio in predicting the occurrence of high-risk EV in HBV-induced cirrhosis patients, resulting in an AUC of 0.763 with an optimal cutoff value of 883.5. These findings suggest that the PSDR holds significant predictive value for high-risk variceal development, possibly reducing the need for invasive endoscopic procedures in certain cases.

In conclusion, the PSDR is a key risk factor for the development of EV in patients with HBV induced cirrhosis, exhibiting substantial predictive accuracy for both standard and high-risk varices. This ratio serves as a valuable, non-invasive biomarker for anticipating the onset of EV. It is important to note that this study is retrospective in nature with a relatively limited sample size, which may have introduced bias. Future research should aim to expand the sam-

ple population and further assess the predictive capability of the PSDR across a wider range of cirrhotic patients with EV.

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Disclosure of conflict of interest

None.

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