BMC Gastroenterology

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Nomogram based on lymphocyte-associated inflammatory indexes predicts portal vein thrombosis after splenectomy with esophagogastric devascularization

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Abstract

Objective The relationship between lymphocyte-associated inflammatory indices and portal vein thrombosis (PVT) following splenectomy combined with esophagogastric devascularization (SED) is currently unclear. This study aims to investigate the association between these inflammatory indices and PVT, and to develop a nomogram based on these indices to predict the risk of PVT after SED, providing an early warning tool for clinical practice.

Methods We conducted a retrospective analysis of clinical data from 131 cirrhotic patients who underwent SED at Lanzhou University's Second Hospital between January 2014 and January 2024. Independent risk factors for PVT were identified through univariate and multivariate logistic regression analyses, and the best variables were selected using the Akaike Information Criterion (AIC) to construct the nomogram. The model's predictive performance was assessed through receiver operating characteristic (ROC), calibration, decision, and clinical impact curves, with bootstrap resampling used for internal validation.

Results The final model incorporated five variables: splenic vein diameter (SVD), D-Dimer, platelet-to-lymphocyte ratio (PLR), monocyte-to-lymphocyte ratio (MLR), and red cell distribution width-to-lymphocyte ratio (RLR), achieving an area under the curve (AUC) of 0.807, demonstrating high predictive accuracy. Calibration and decision curves demonstrated good calibration and significant clinical benefits. The model exhibited good stability through internal validation.

Conclusion The nomogram model based on lymphocyte-associated inflammatory indices effectively predicts the risk of portal vein thrombosis after SED, demonstrating high accuracy and clinical utility. Further validation in larger, multicenter studies is needed.

Keywords Inflammation, Portal vein thrombosis, Splenectomy, Nomogram

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Introduction

Cirrhosis represents the final stage of chronic liver disease, marked by widespread fibrosis and the development of regenerative nodules within the liver tissue. Cirrhosis accounts for 2.4% of global deaths, posing a significant burden on global public health. With the increasing prevalence of obesity and alcohol consumption, the incidence of non-alcoholic fatty liver disease and alcoholic cirrhosis continues to rise [1]. Portal hypertension is a critical stage in the progression of cirrhosis, often accompanied by complications such as hypersplenism, esophagogastric variceal bleeding, and hepatic encephalopathy, which are major causes of mortality in cirrhotic patients [2]. Currently, various therapeutic strategies are available for portal hypertension and its associated complications, including pharmacological treatment, endoscopic treatment, interventional therapy, surgical treatment, liver transplantation, and combinations of these approaches [3]. However, patients treated with drugs and endoscopy have a high recurrence rate, with a one-year recurrence rate of 60% [4]. Transjugular intrahepatic portosystemic shunt (TIPS) is considered an effective intervention for uncontrolled esophagogastric variceal bleeding after medical and endoscopic treatment, but the incidence of post-TIPS hepatic encephalopathy is as high as 20–50%, significantly affecting prognosis and increasing mortality risk [5, 6]. Although liver transplantation is the definitive treatment for patients with cirrhosis, the limited availability of donor livers and financial constraints prevent the majority of patients from receiving this therapy [7]. Splenectomy combined with esophagogastric devascularization (SED) offers advantages over other treatments, including a lower short-term rebleeding rate and the resolution of splenomegaly and hypersplenism. In China, approximately 300 million people suffer from viral hepatitis, with the majority experiencing splenomegaly and hypersplenism, making SED a widely adopted procedure. Recent studies have shown that SED significantly increases hepatic blood flow, improves liver function, and, compared to endoscopic treatment, reduces the incidence of hepatocellular carcinoma, thereby improving patient survival [8, 9]. Therefore, SED can be an effective treatment option for certain patients and warrants further promotion.

However, the incidence of portal vein thrombosis (PVT) after SED ranges from 30.1 to 47.8% [10]. Long-term PVT can exacerbate portal hypertension, increasing the risk of upper gastrointestinal bleeding and gastrointestinal congestion [11]. Early identification of high-risk patients for PVT and timely intervention can effectively improve patient outcomes. Virchow's triad, which includes reduced blood flow, local vascular injury, and a hypercoagulable state, is considered the main mechanism for PVT formation [12]. Current research suggests that an increase in D-Dimer levels, an enlarged spleen, a wider splenic vein diameter (SVD), low platelet count, reduced portal vein flow velocity, and the presence of ascites are significant factors associated with an increased risk of PVT following SED [13]. However, these factors primarily focus on hemodynamics and coagulation mechanisms, and they do not fully capture all the potential contributors to PVT formation after SED. Moreover, there is still a lack of effective predictive tools that can accurately identify high-risk patients either before or shortly after surgery.

In recent years, numerous studies have shown a close relationship between inflammation and both deep vein thrombosis and cerebral venous thrombosis. Certain inflammatory markers have been identified as useful predictors for the formation of cerebral venous thrombosis, with lymphocyte counts, in particular, being significantly lower in patients with cerebral venous thrombosis compared to control groups [14, 15]. Therefore, investigating the role of lymphocyte-related inflammatory indices in the formation of PVT is of significant research importance. Indices such as the neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), monocyte-to-lymphocyte ratio (MLR), and red cell distribution width-to-lymphocyte ratio (RLR) have recently been found to not only reflect the body's inflammatory status but also be closely associated with the formation of thrombosis. A study found that the NLR and PLR are independent risk factors for the formation of PVT in cirrhotic patients who have not undergone surgery. The predictive model based on these indices demonstrated high accuracy and significant clinical value [16]. Additionally, monocytes are considered key players in the interaction between inflammation and thrombosis, with elevated levels closely associated with an increased risk of thrombosis formation [17]. Red cell distribution width (RDW), which reflects the variability in red blood cell size, is widely used to assess systemic inflammation. Numerous studies have shown that RDW is not only a strong marker for cardiovascular diseases but is also closely linked to the occurrence of venous thromboembolism [18, 19]. Despite significant progress in understanding these inflammatory markers in other types of thrombosis, their role in the formation of PVT following SED has yet to be studied. Our research aims to explore the relationship between lymphocyte-related inflammatory indices, such as NLR, PLR, MLR, and RLR, and the development of PVT after SED. Additionally, we seek to construct a predictive model for PVT formation post-SED by incorporating these inflammatory indices.

Materials and methods

Study patients

This study retrospectively analyzed patients with cirrhotic portal hypertension who underwent splenectomy combined with esophagogastric devascularization (SED) at the Second Hospital of Lanzhou University between January 2014 and January 2024. Cirrhosis was diagnosed through a combination of clinical signs, laboratory results, imaging findings, or liver biopsy. PVT was diagnosed using Doppler ultrasound of the portal vein or abdominal CT, with postoperative PVT defined as PVT detected within 30 days after surgery. All patients were required to first meet the inclusion criteria, followed by screening against the exclusion criteria. Ultimately, only those who met the inclusion criteria and did not meet any of the exclusion criteria were included in the study. The inclusion criteria were as follows: (1) age between 18 and 75 years; (2) confirmed diagnosis of cirrhotic portal hypertension based on clinical, laboratory, radiological, or histological criteria; (3) significant splenomegaly and hypersplenism that negatively affected the patient's quality of life; (4) upper gastrointestinal bleeding caused by portal hypertension, especially in cases where medical treatment was ineffective or recurrent bleeding occurred; and (5) preoperative liver function classified as Child-Pugh A or B, or Child-Pugh C with liver function improved to A or B after active liver-protective treatment. The exclusion criteria were as follows: (1) patients with abnormal coagulation function prior to surgery; (2) patients with PVT indicated by preoperative portal vein Doppler ultrasound or abdominal CT; (3) patients with malignant hepatic tumors or other malignancies; and (4) patients who had undergone preoperative splenic artery embolization. (5) The patient has a history of hematologic disorders, autoimmune diseases, or chronic inflammatory conditions.

This study adhered to the Declaration of Helsinki and was approved by the Medical Ethics Committee of the Second Hospital of Lanzhou University (Approval Number: 2024 A-961). Given the retrospective design of the study and the safeguards in place to protect patient privacy and personal information, the committee waived the need for informed consent.

Anticoagulation protocol

Prophylactic anticoagulation was administered to all patients in the study. Patients without signs of bleeding within 3 days postoperatively received subcutaneous injections of enoxaparin sodium at a dose of 0.4 mL once daily for 7–10 days. If thrombosis was detected, the treatment duration was appropriately extended. After discharge, patients were switched to oral aspirin enteric-coated tablets for 3–6 months of anticoagulation therapy. During treatment, drainage, complete blood count, liver and kidney function, and coagulation parameters were closely monitored to assess the risk of bleeding.

Data collection

All data were obtained from the hospital information system. The collected data included demographic information, medical record text, laboratory test results, and imaging data. Demographic information included age and gender. Medical record data encompassed surgical method, BMI, etiology, history of hypertension, diabetes, smoking, alcohol consumption, history of upper gastrointestinal bleeding, previous surgeries, and other past medical conditions. Laboratory test results included preoperative total bilirubin, serum creatinine, international normalized ratio (INR), and albumin levels; as well as postoperative day 7 counts of platelets, neutrophils, lymphocytes, monocytes, red cell distribution width (RDW), white blood cell count, prothrombin time, and D-Dimer levels. Imaging data included preoperative portal vein diameter (PVD), SVD, and the depth of ascites. The grading of ascites was based on the depth of abdominal fluid as measured by ultrasound: <3 cm was defined as mild ascites, 3–10 cm as moderate ascites, and >10 cm as severe ascites [20]. The Child-Pugh score was calculated based on total bilirubin, albumin, international normalized ratio (INR), ascites, and hepatic encephalopathy. The ALBI score was calculated based on albumin and total bilirubin levels. The lymphocyte-associated inflammatory indices were calculated as follows: NLR = neutrophil count / lymphocyte count; PLR = platelet count / lymphocyte count; MLR = monocyte count / lymphocyte count; RLR = red cell distribution width (RDW) / lymphocyte count [21, 22].

Statistical analysis

Data analysis was carried out using SPSS (Version 27) and R software (Version 4.3.3). Continuous data were summarized as mean ± standard deviation or interquartile range, while categorical data were reported as frequencies and percentages. To compare continuous variables, we employed the t-test or Mann-Whitney U test, and for categorical variables, the $\chi 2$ test or Fisher's exact test was used. Missing data under 10% were addressed through multiple imputation. ROC curves and the Youden index were utilized to establish optimal cutoff points for certain continuous variables. Both univariate and multivariate logistic regression analyses were performed to identify independent predictors of PVT. When constructing the nomogram, we used the Akaike Information Criterion (AIC) for variable selection. Specifically, variables that showed statistical significance in univariate analysis were included in the initial model. We then applied a stepwise backward regression method, guided by the AIC, to identify the optimal model, selecting the combination of

variables with the lowest AIC value. The model's accuracy was measured by the area under the ROC curve (AUROC), and calibration was assessed via Hosmer-Lemeshow curves. Decision curves and clinical impact curves were used to assess the model's practical value. Internal validation was conducted using 1,000 bootstrap resamples. Statistical significance was set at $P<0.05$.

Results

Patient characteristics

This study retrospectively collected clinical data from 146 patients with cirrhosis. A total of 131 cirrhotic patients met the inclusion and exclusion criteria and were included in the final analysis (Fig. 1). The clinical characteristics of these patients are shown in Table 1. Among the 131 cirrhotic patients, 55 had PVT. The mean age of all patients was 47 years, with 59% being male and 41% female. The majority of patients had a normal BMI, and the predominant etiology was hepatitis B (71%). The optimal thresholds for continuous variables were identified through ROC analysis, were as follows: PVD of 1.535 cm, SVD of 1.017 cm, prothrombin time of 16.05 s, D-Dimer of 13.255 mg/L, platelet count of 311.5, NLR of 8.7, PLR of 336.07, monocyte-to-lymphocyte ratio (MLR) of 1.18, and RLR of 11.46. There were no statistically significant differences between cirrhotic patients with and without PVT in terms of age, gender, smoking history, alcohol consumption, BMI, etiology, Child-Pugh classification, ALBI grade, diabetes, hypertension, history of upper gastrointestinal bleeding, ascites, PVD, prothrombin time, and platelet count. However, patients with PVT had a significantly larger SVD (P = 0.043) and higher D-Dimer levels (P = 0.014) compared to those without PVT.

Univariate and multivariate analysis

As shown in Table 2, Univariate and multivariate logistic regression analyses were employed to identify potential predictive factors. The univariate analysis showed significant associations between D-Dimer, MLR, NLR, PLR, RLR, PVD, SVD, and the occurrence of PVT following SED ($P<0.05$). Further multivariate analysis confirmed that D-Dimer (Odds Ratio [OR]=2.98, 95% Confidence Interval [CI]=1.29–6.87, $P=0.011$) and MLR (OR=3.74, 95% CI=1.52–9.19, $P=0.004$) were independent risk factors for PVT post-SED.

Development and validation of the nomogram model

The optimal nomogram model for predicting PVT after SED in cirrhotic patients was selected by comparing AIC values, as shown in Fig. 2. The total score is calculated by summing the values from multiple variables (SVD, PLR, MLR, RLR, and D-Dimer), and then mapping the "Total Points" to the "Diagnostic Possibility" axis below to predict the probability of PVT occurrence. A higher total score corresponds to an increased probability of PVT. The nomogram's predictive ability was assessed using the ROC curve, resulting in an AUC of 0.807 (95% CI: 0.733–0.881), indicating high predictive value and superior predictive efficiency compared to each independent factor (Table 3; Fig. 3). The calibration curve

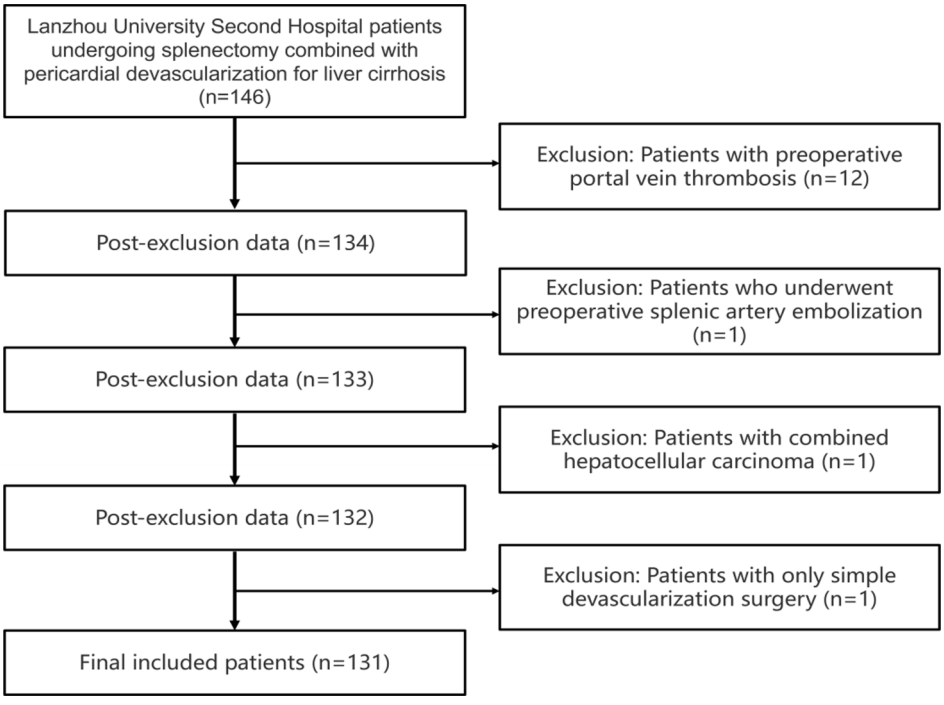

Fig. 1 Flow diagram of study cohort selection

Table 1 Characteristics of the study cohort

Variable	Overall (*n* = 131)	no PVT (*n* = 76)	PVT (*n* = 55)	*P*-value
Age (years)	46.50 ± 10.17	47.50 ± 10.36	45.11 ± 9.86	0.186
Gender				0.786
Female	53 (41%)	32 (42%)	21 (38%)	
Male	78 (59%)	44 (58%)	34 (62%)	
Smoked				0.176
No	120 (92%)	67 (88%)	53 (96%)	
Yes	11 (8%)	9 (12%)	2 (4%)	
Alcohol				0.371
No	122 (93%)	69 (91%)	53 (96%)	
Yes	9 (7%)	7 (9%)	2 (4%)	
BMI				0.695
Normal	108 (82%)	64 (84%)	44 (80%)	
Overweight or Obesity	23 (18%)	12 (16%)	11 (20%)	
Etiology				0.571
Hepatitis B	93 (71%)	52 (68%)	41 (75%)	
Others	38 (29%)	24 (32%)	14 (25%)	
Child-Pugh grade				0.307
A	84 (64%)	52 (68%)	32 (58%)	
B	47 (36%)	24 (32%)	23 (42%)	
ALBI grade				0.795
1	26 (20%)	14 (18%)	12 (22%)	
2	105 (80%)	62 (82%)	43 (78%)	
Diabetes				1
No	122 (93%)	71 (93%)	51 (93%)	
Yes	9 (7%)	5 (7%)	4 (7%)	
Hypertension				1
No	126 (96%)	73 (96%)	53 (96%)	
Yes	5 (4%)	3 (4%)	2 (4%)	
UGIB				0.297
No	46 (35%)	30 (39%)	16 (29%)	
Yes	85 (65%)	46 (61%)	39 (71%)	
Ascites				0.169
None	60 (46%)	40 (53%)	20 (36%)	
Mild	43 (33%)	21 (27%)	22 (40%)	
Moderate to Severe	28 (21%)	15 (20%)	13 (24%)	
PVD (cm)				0.071
< 1.535	82 (63%)	53 (70%)	29 (53%)	
≥ 1.535	49 (37%)	23 (30%)	26 (47%)	
SVD (cm)				**0.043**
< 1.017	60 (46%)	41 (54%)	19 (35%)	
≥ 1.017	71 (54%)	35 (46%)	36 (65%)	
PT (s)				0.659
< 16.050	124 (95%)	73 (96%)	51 (93%)	
≥ 16.050	7 (5%)	3 (4%)	4 (7%)	
D-Dimer (mg/L)				**0.014**
< 13.255	77 (59%)	52 (68%)	25 (45%)	
≥ 13.255	54 (41%)	24 (32%)	30 (55%)	
PLT				0.078
< 311.5	84 (64%)	54 (71%)	30 (55%)	
≥ 311.5	47 (36%)	22 (29%)	25 (45%)	

PVT: portal vein thrombosis; BMI: Body Mass Index; ALBI grade: Albumin-Bilirubin Grade; UGIB: History of Upper Gastrointestinal Bleeding; PVD: Portal Vein Diameter; SVD: Splenic Vein Diameter; PLT: Platelet

demonstrated a strong correlation between the predicted probabilities and the observed outcomes (Fig. 4A). The DCA curve demonstrated significant clinical benefit of the nomogram in predicting PVT after SED (Fig. 4B). Further analysis using the clinical impact curve, assuming 1,000 cirrhotic patients are assessed with the model for predicting PVT after SED, showed that the number of predicted cases closely matched the actual number of cases at a threshold of approximately > 0.6, with the number of high-risk patients identified by the model being close to the number of true positives (Fig. 4C). Additionally, internal validation was carried out using the bootstrap method, with 1,000 iterations, which yielded a C-index value of 0.770, further confirming the accuracy of the predictions. The ROC curves generated from different bootstrap samples clustered around and close to the central red curve, indicating stable performance of the nomogram model (Fig. 4D).

Discussion

In this study, we identified D-Dimer and MLR as independent risk factors for PVT following SED. To develop a model that is clinically practical and capable of effectively identifying high-risk patients, we utilized the AIC. By balancing model fit and complexity, AIC allowed us to select the most predictive combination of variables. Although some variables did not show statistical significance in the multivariate analysis, their inclusion based on the AIC improved the overall predictive accuracy and stability of the model. Ultimately, we included PLR, MLR, RLR, SVD, and D-Dimer levels to successfully construct a nomogram for predicting PVT formation in cirrhotic patients after SED. The model demonstrated strong predictive performance in distinguishing high-risk from low-risk patients, with an AUC of 0.807, indicating high predictive accuracy. Furthermore, calibration curves, decision curves, and clinical impact curves all indicated good calibration and significant clinical benefit. The model also showed robust stability through internal validation.

Based on these results, the nomogram model not only deepens our understanding of the mechanisms underlying PVT formation but also provides a reliable tool for clinical practice. In practical use, clinicians first need to collect the patient's hematological and imaging data, calculating the PLR, MLR, RLR, SVD, and D-Dimer values.

Table 2 Univariate and multivariate analysis

Variable	Univariate analysis			Multivariate analysis		
	OR	95%CI	*P* value	OR	95%CI	*P* value
BMI	1.33	0.54–3.29	0.533			
UGIB	1.59	0.76–3.33	0.221			
CTP	1.56	0.76–3.2	0.229			
Ascites	1.39	0.89–2.17	0.147			
D-dimer	2.6	1.27–5.33	**0.009**	2.98	1.29–6.87	**0.011**
PT	1.91	0.41–8.89	0.410			
PLT	2.05	0.99–4.23	0.053			
MLR	4.67	2.13–10.23	**0.000**	3.74	1.52–9.19	**0.004**
NLR	2.7	1.29–5.63	**0.008**			
PLR	3.6	1.71–7.57	**0.001**	1.86	0.79–4.39	0.157
RLR	18	2.33-139.02	**0.006**	8.37	1-69.92	0.05
PVD	2.07	1-4.25	**0.049**			
SVD	2.22	1.09–4.54	**0.029**	2.2	0.96–5.04	0.063

BMI: Body Mass Index; UGIB: History of Upper Gastrointestinal Bleeding; CTP: Child–Pugh Grade; PT: Prothrombin Time; PLT: Platelet; MLR: Monocyte-Lymphocyte Ratio; NLR: Neutrophil-to-Lymphocyte Ratio; PLR: Platelet to Lymphocyte Ratio; RLR: red blood cell distribution width-to-lymphocyte ratio; PVD: Portal Vein Diameter; SVD: Splenic Vein Diameter

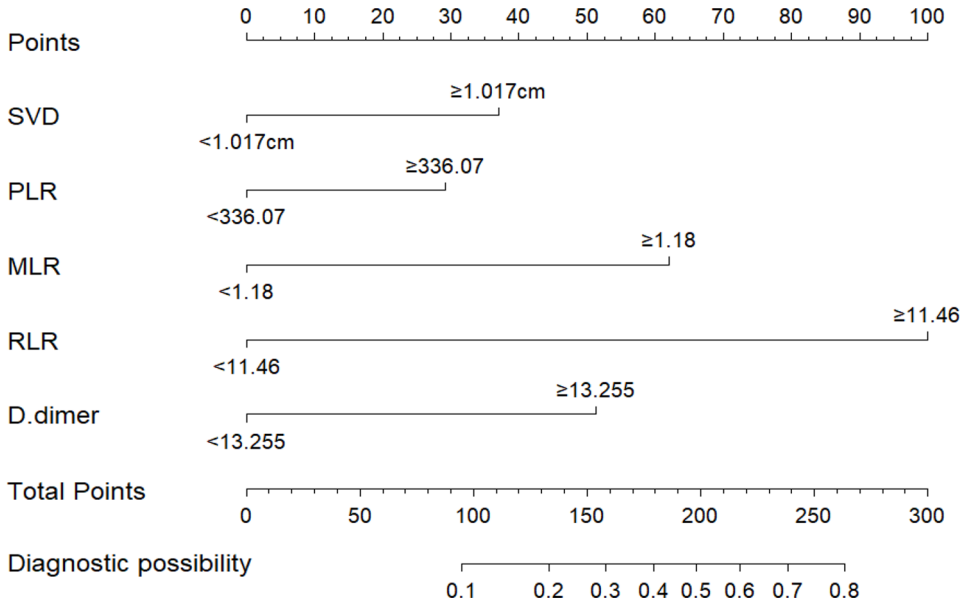

Fig. 2 Nomogram model predicting the probability of portal vein thrombosis (PVT)

Table 3 Predictive performance of the model and independent factors

Variable	AUROC	95% CI	Sensitivity	Specificity
Nomogram	0.807	0.733–0.881	0.836	0.684
SVD	0.597	0.499–0.695	0.655	0.539
PLR	0.648	0.551–0.745	0.545	0.750
MLR	0.674	0.581–0.767	0.782	0.566
RLR	0.616	0.521–0.711	0.982	0.250
D-dimer	0.615	0.516–0.713	0.545	0.684

SVD: Splenic Vein Diameter; PLR: Platelet to Lymphocyte Ratio; MLR: Monocyte-Lymphocyte Ratio; RLR: red blood cell distribution width-to-lymphocyte ratio

Next, they refer to the nomogram's variable ranges to assign corresponding "points" to each variable. These scores are then summed to obtain the "total points." The total score is matched against the PVT risk curve provided in the nomogram, allowing clinicians to estimate the patient's risk of developing PVT. Based on this risk assessment, more aggressive interventions—such as adjusting anticoagulant therapy or increasing follow-up frequency—can be taken for high-risk patients, while low-risk patients can continue with standard care protocols. Moreover, the model's simplicity and ease of use make it highly adaptable across various healthcare settings. In resource-rich environments, the model can be integrated into electronic health record (EHR) systems,

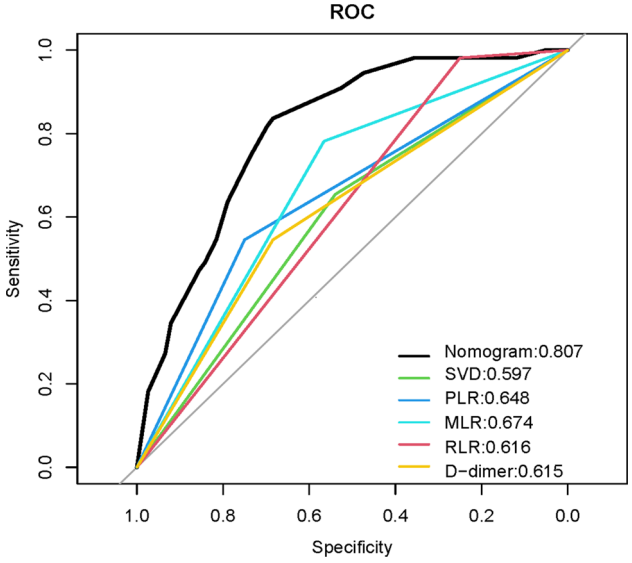

Fig. 3 Receiver operating characteristic curve of the nomogram and independent factors

allowing for automated data input and real-time PVT risk calculation to support clinician decision-making. In resource-limited environments, clinicians can manually input basic blood test and imaging data to assess risk, ensuring that even in remote or underserved areas, high-risk patients can be accurately identified and timely interventions provided. This adaptability enhances the model's practical value across diverse healthcare settings.

Cirrhotic patients are prone to PVT due to coagulopathy and portal hypertension. Following SED, the increase in platelet counts and alterations in portal vein anatomy further elevate the risk of PVT formation [11]. At our center, the incidence of PVT after SED was found to be 42%. Therefore, identifying the risk factors for PVT formation, enabling early prediction and timely intervention, can benefit a greater number of cirrhotic patients. The formation of PVT is a complex process that involves the interplay of multiple factors. Previous studies have highlighted hemodynamic factors such as PVD, SVD, and

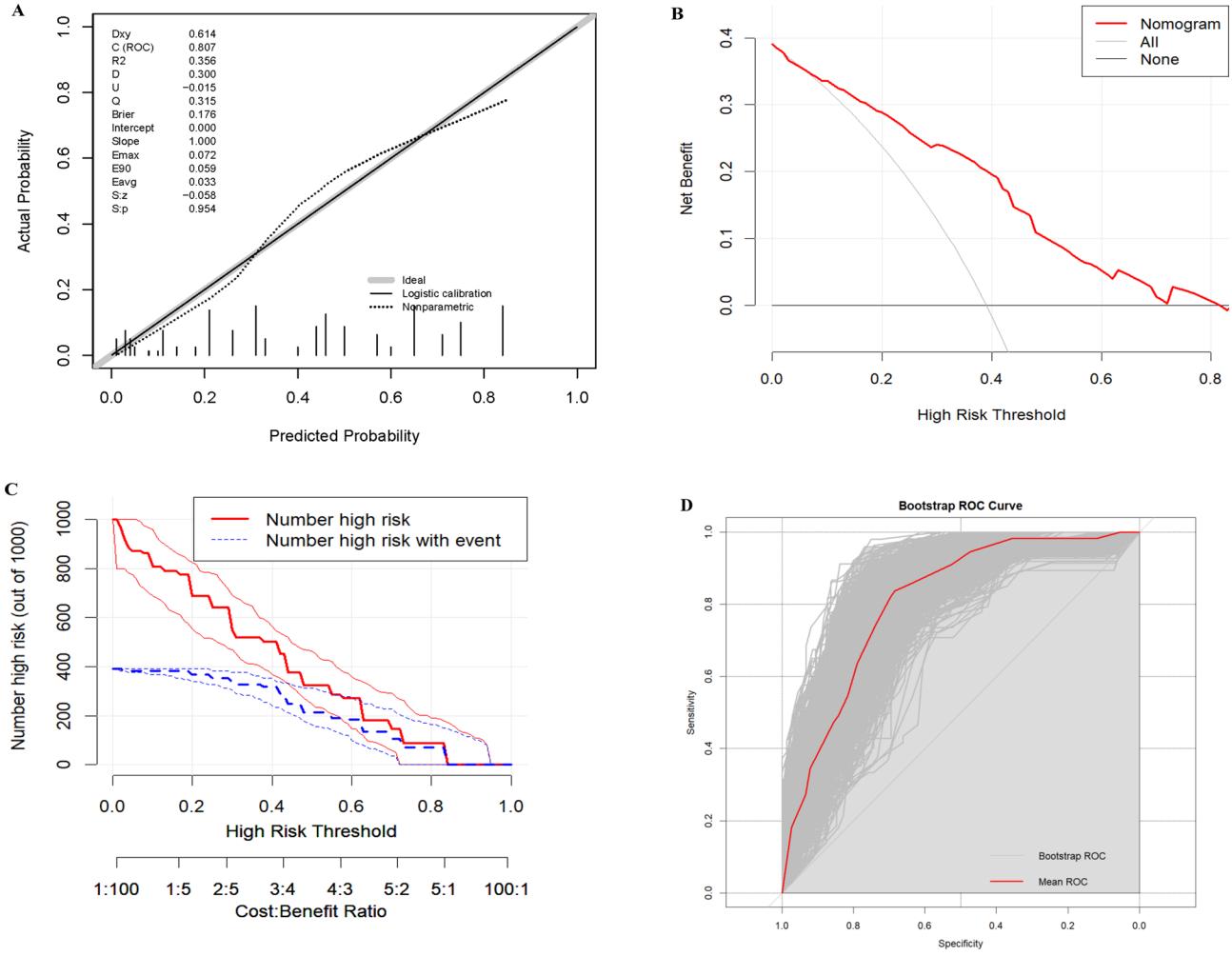

Fig. 4 Internal validation of nomogram. **A**: Calibration curve of the nomogram. **B**: Decision curve of the nomogram. **C**: Clinical impact curve of the nomogram. **D**: Receiver operating characteristic curve of bootstrap

portal vein flow velocity as important predictors of PVT. Larger PVD, larger SVD, and lower portal vein flow velocity increase the likelihood of PVT, possibly due to excessive portal vein pressure leading to reduced flow velocity, which in turn widens PVD and SVD, increasing the risk of blood aggregation and thrombosis [23–25]. D-Dimer, as a byproduct of fibrin degradation, is typically elevated when there is increased activity in the coagulation system and ongoing fibrin formation [26]. Studies have shown that D-Dimer can be used as a predictor for the occurrence of PVT [27]. In addition to the factors mentioned above, a meta-analysis has shown that platelet count and ascites can also be used to predict the occurrence of PVT [28]. In our study, we similarly identified SVD and D-Dimer as effective predictors, which were included in the construction of our predictive model. However, PVD, PLT, and ascites were not included in the predictive model. Although PVD showed significance in univariate analysis, this may have been influenced by confounding factors or the sample size, as it did not remain significant in multivariate analysis. Further research is needed to explore the relationship between these variables, and larger sample studies are required to validate their predictive value.

Although previous studies have identified various predictors of PVT and have constructed predictive models combining these factors with certain predictive value, the relationship between lymphocyte-associated inflammatory indices and PVT after SED remains unclear. Recent evidence suggests a close relationship between inflammation and thrombosis. The study by Manz et al. [29] demonstrated that inflammation promotes thrombosis through mechanisms mediated by von Willebrand factor (VWF). Moreover, Wu et al. [30] highlighted the complex interplay between inflammation and the coagulation system, where inflammation activates the coagulation system, leading to platelet aggregation and fibrin formation, thereby promoting thrombosis. The formed thrombi, in turn, exacerbate the inflammatory response, creating a vicious cycle. Lymphocytes are a key component of the immune system, and studies have found that patients with thrombosis often experience a decrease in lymphocyte count. This may be related to lymphocyte depletion or redistribution caused by the activation of the inflammatory response [31]. Another study [32] showed that regulatory T cells modulate immune responses, suppress inflammation, and limit thrombosis. However, when the inflammatory response becomes excessive, the regulatory capacity of T cells may be insufficient, leading to overactivation of the coagulation system and an increased risk of thrombosis. Neutrophils can release DNA and antimicrobial proteins to form neutrophil extracellular traps (NETs). These NETs not only capture pathogens but also form web-like structures within blood vessels, promoting the accumulation of platelets, red blood cells, and coagulation factors, thereby enhancing thrombosis [33]. Platelets play a crucial role in thrombosis by adhering to damaged blood vessel walls, activating and releasing procoagulant substances, and aggregating to form clots [34]. Monocytes are a major source of tissue factor (TF), which, upon vascular injury, binds to coagulation factor VII to initiate the extrinsic coagulation cascade, promoting thrombus formation. Activated monocytes directly contribute to the coagulation process by releasing TF, thereby increasing the risk of thrombosis [35]. Elevated RDW is closely associated with inflammation and oxidative stress. Oxidative stress not only damages red blood cells but also harms vascular endothelial cells, which in turn activates platelets and promotes thrombosis [36]. Moreover, the greater the variation in red blood cell size, the poorer the blood flow, further contributing to thrombosis. NLR, PLR, MLR, and RLR are calculated by dividing neutrophils, platelets, monocytes, and RDW by lymphocyte count, respectively. These ratios are considered surrogate markers of inflammation and have been widely used in prognostic studies across various systemic diseases [37–39]. In studies related to thrombotic diseases, NLR and PLR have demonstrated significant clinical relevance. Kuplay et al. [40] found that NLR and PLR were closely associated with the location and burden of deep vein thrombosis (DVT) in the lower extremities, with higher NLR levels correlating with more extensive DVT involvement. Similarly, Selvaggio et al. [41] reported that PLR was significantly associated with lower extremity DVT (OR: 3.379, $P=0.007$), suggesting its utility as a biomarker for DVT in hospitalized patients. Other studies have also noted a significant association between NLR, PLR, and retinal vein thrombosis [42]. However, the value of MLR and RLR in thrombotic diseases remains to be further explored. In our study, PLR was also identified as an effective predictor of thrombosis. However, due to potential multicollinearity with other factors, NLR was not included in the final predictive model. Furthermore, this study is the first to systematically evaluate the role of NLR, PLR, MLR, and RLR in predicting PVT after SED. The results showed that PLR, MLR, and RLR are effective predictors for PVT in cirrhotic patients following SED, with higher values being closely associated with an increased risk of PVT. When combined with factors such as SVD and D-Dimer, the nomogram constructed from these predictors proved to be an effective tool for forecasting PVT.

As far as we know, this research is the first to assess the impact of lymphocyte-associated inflammatory indices (NLR, PLR, MLR, RLR) in PVT formation after SED and to successfully develop a nomogram model for predicting PVT occurrence. This model not only enhances our understanding of the factors contributing to PVT

formation after SED but also provides clinicians with an easy-to-use tool for promptly identifying of high-risk patients, enabling timely interventions to enhance patient outcomes. The nomogram model in our study demonstrated an AUC of 0.807, indicating high accuracy and predictive value for PVT. Additionally, the model's curves for calibration, decision-making, and clinical impact all demonstrate reliable alignment with predictions and offer substantial clinical value. Internal validation showed the model to be stable. The use of PLR, MLR, and RLR, which are derived from routine blood tests, not only reduces costs but also makes the predictive model highly practical and applicable, especially in resource-limited settings.

However, there are several limitations to this study. Firstly, as this study is a single-center retrospective analysis with a relatively small sample size, the external validity of the results may be limited. The stability and predictive accuracy of the model might be influenced by the sample size. Therefore, although our model demonstrated good stability in internal validation, it is necessary to further validate these findings in multi-center, large-sample prospective studies to ensure the model's broad applicability and reliability. Second, although we included various inflammatory indices, other potential influencing factors may not have been accounted for in the model, warranting further exploration in future research. Additionally, due to the uniform anticoagulation protocol used in our study, the impact of different anticoagulation strategies on PVT occurrence was not fully evaluated, providing a new direction for future research.

Conclusion

In conclusion, this study successfully developed a nomogram model based on lymphocyte-associated inflammatory indices (PLR, MLR, RLR) to predict the risk of PVT after SED. The model demonstrated high accuracy and stability, providing an important tool for clinical practice. Future studies should aim to further validate this model in larger samples, with the goal of promoting its application in a wider range of clinical settings, thereby offering greater benefits in the management of patients with cirrhosis.

Acknowledgements
We sincerely acknowledge the financial contributions from the Natural Science Foundation of Gansu Province and the Youth Science and Technology Foundation of Gansu Province.

Author contributions
CF G was involved in the design, data collection, data analysis, manuscript drafting, and revision. MY L contributed to data analysis and manuscript revision. FX W and XD Xu were responsible for funding acquisition and also participated in manuscript revision.

Funding
The authors declare that financial support was received for the research, authorship, and/or publication of this article. Specifically, this work was supported by the Natural Science Foundation of Gansu Province (21JR11RA103) and the Gansu Provincial Youth Science and Technology Fund (21JR1RA161).

Data availability
The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
This study adhered to the Declaration of Helsinki and was approved by the Medical Ethics Committee of the Second Hospital of Lanzhou University (Approval Number: 2024 A-961). Given the retrospective design of the study and the safeguards in place to protect patient privacy and personal information, the committee waived the need for informed consent.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

Received: 14 August 2024 / Accepted: 11 September 2024
Published online: 19 September 2024

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