Original Article

Predictive Value of Inflammation Biomarkers in Patients with Portal Vein Thrombosis

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Abstract

Background and Aims: To investigate the usefulness of inflammation biomarkers to serve as a predictors of portal vein thrombosis (PVT) postoperatively (post) in patients with portal hypertension after splenectomy and periesophagogastric devascularization. ***Methods:*** A total of 177 liver cirrhosis patients were recruited from January 2013 to December 2017. They were divided into a PVT group (n=71) and a non-PVT group (n=106), according to ultrasound examination findings at 7-day post. Inflammation biomarkers involving platelet-to-lymphocyte ratio (PLR), neutrophil-to-lymphocyte ratio (NLR), monocyte-to-lymphocyte ratio (MLR), red blood cell distribution width-to-platelet ratio(RPR), mean platelet volume-to-platelet ratio (MPR) preoperatively (pre) and at 1, 3, 7-days post were recorded. ***Results:*** The univariate logistic regression analysis indicated that PLR (pre) (odds ratio (OR)=3.963, 95% confidence interval (CI)=2.070–7.587, p<0.000), MLR (pre) (OR=2.760, 95% CI=1.386–5.497, p=0.004), PLR (post-day 7) (OR=3.345, 95% CI=1.767–6.332, p=0.000) were significantly associated with the presence of PVT. The multivariate logistic regression analysis results indicated that PLR (pre) (OR=3.037, 95% CI=1.463–6.305, p=0.003), MLR (pre) (OR=2.188, 95% CI=1.003–4.772, p=0.049), PLR(post-day 7) (OR=2.166, 95% CI=1.053–4.454, p=0.036) were independent factors for predicting PVT. ***Conclusions:*** The PLR (pre), MLR (pre), and PLR (post-day 7) are predictors of portal vein thrombosis post in patients with portal hypertension after splenectomy and periesophagogastric devascularization.

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Introduction

Splenectomy and periesophagogastric devascularization is one of the main procedures for the management of portal hypertension and hypersplenism, especially for patients with variceal bleeding. The incidence of portal vein thrombosis (PVT) after such surgery can be as high as 6.3%~39.0%.[1] PVT is characterized by partial or total occlusion of the portal vein, with the presence of solid intraluminal material. It can elevate the resistance to portal inflow as the portal venous pressure is increased. Consequently, liver function becomes deteriorated due to the decreased blood flow to the liver. It also shows increased rate of re-bleeding and aggravated progression of bleeding.[2,3] The rate of bleeding in liver cirrhosis patients with PVT is higher than in those without PVT.[4] Besides these harmful effects, PVT even influences the eligibility for liver transplantation, since it makes the operative technique more complicated and decreases the 1-year survival rate of the recipient.[5,6]

Vascular endothelial injury, blood flow alteration, and prothrombotic condition are the three major determinants of venous thrombosis, described as Virchow's triad.[7] Previous research has indicated that a higher model for end-stage liver disease (MELD) score, wider splenic vein diameter, increased antithrombin III concentration and prolonged prothrombin time are risk factors of PVT after splenectomy in patients with liver cirrhosis.[8–11]

However, an accumulation of evidence suggests that systemic inflammatory response is associated with the development of venous thrombosis.[12–17] Some inflammation biomarkers involving platelet-to-lymphocyte ratio (PLR), lymphocyte-to-monocyte ratio (LMR), and neutrophil-to-lymphocyte ratio (NLR) have been demonstrated to be useful in predicting venous thromboembolism (deep vein thrombosis of the lower limbs and/or pulmonary embolism or cerebral vein thrombosis).[18–21] Another series of an index involving the monocyte-to-lymphocyte ratio (MLR), NLR, prognostic nutritional index (PNI), red blood cell distribution width-to-platelet ratio (RPR), and mean platelet volume-to-platelet ratio (MPR) consists of inflammation response biomarkers associated with the prognostic of inflammatory disease, viral infection disease, coronary artery disease, and malignant tumor.[18–24] However, whether these inflammation biomarkers are able to detect the probability of PVT formation is still unclear.

In previous studies, inflammation biomarkers were not dynamically observed, and the surgery itself may cause the ongoing inflammatory response resulting in change of these markers. In this study, inflammatory biomarkers were de-
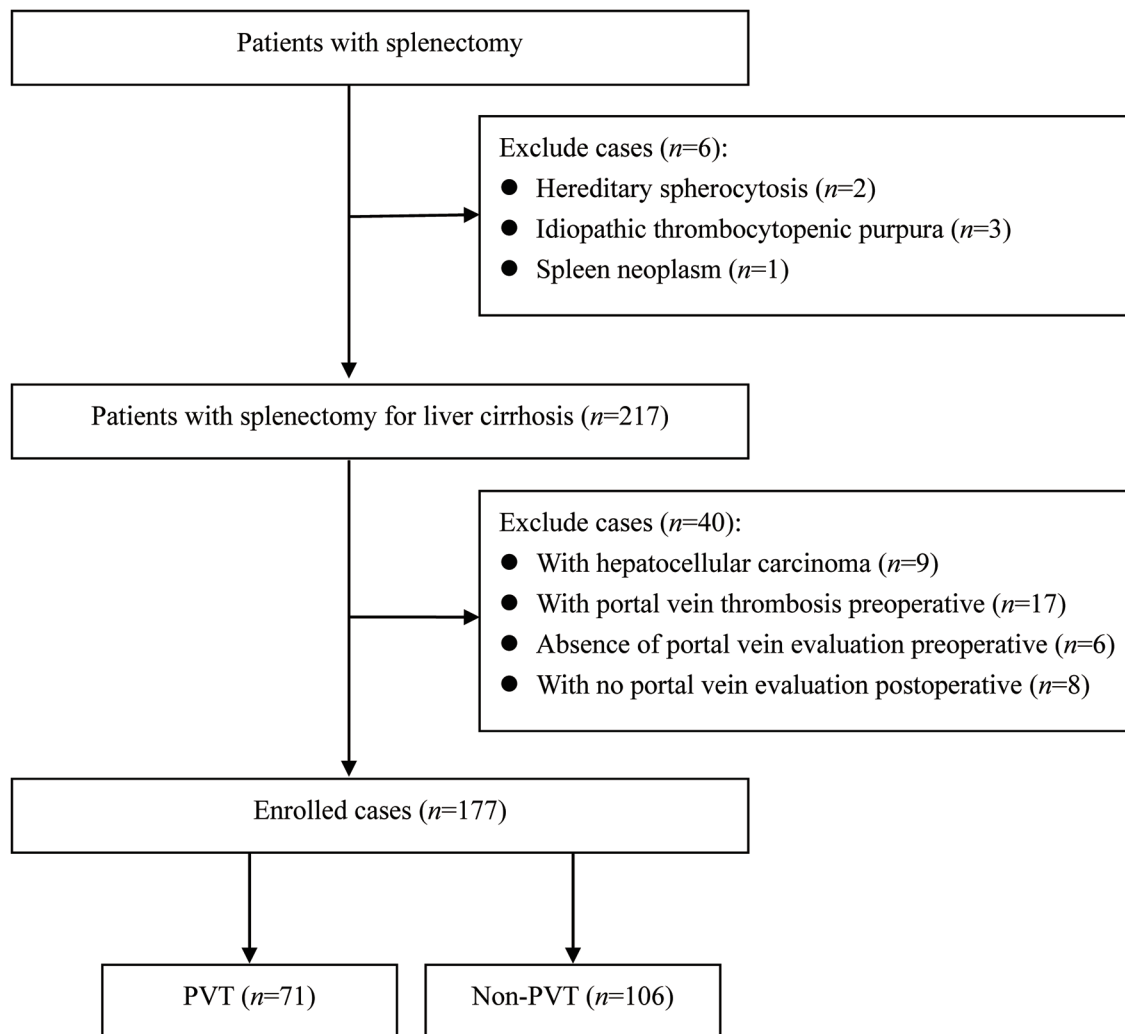
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┌─────────────────────────────────────────┐
│          Patients with splenectomy        │
└─────────────────────────────────────────┘
                    │
                    │      ┌──────────────────────────────────────────────┐
                    ├─────▶│ Exclude cases (n=6):                          │
                    │      │  • Hereditary spherocytosis (n=2)             │
                    │      │  • Idiopathic thrombocytopenic purpura (n=3)  │
                    │      │  • Spleen neoplasm (n=1)                      │
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│ Patients with splenectomy for liver cirrhosis (n=217) │
└─────────────────────────────────────────────────┘
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                    │      ┌──────────────────────────────────────────────────────┐
                    ├─────▶│ Exclude cases (n=40):                                 │
                    │      │  • With hepatocellular carcinoma (n=9)                │
                    │      │  • With portal vein thrombosis preoperative (n=17)    │
                    │      │  • Absence of portal vein evaluation preoperative (n=6)│
                    │      │  • With no portal vein evaluation postoperative (n=8) │
                    │      └──────────────────────────────────────────────────────┘
                    ▼
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│          Enrolled cases (n=177)           │
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              │                │
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     ┌───────────────┐  ┌─────────────────┐
     │  PVT (n=71)   │  │ Non-PVT (n=106) │
     └───────────────┘  └─────────────────┘
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Fig. 1. Flow diagram of the study population. PVT, portal vein thrombosis.

tected at certain times before and after surgery. We aimed to determine whether these inflammation biomarkers could serve as predictors of PVT in liver cirrhosis patients with portal hypertension after splenectomy and periesophagogastric devascularization.

Methods

Patients

We conducted a retrospective analysis of the patients consecutively admitted to the Hospital for portal hypertension, who had been diagnosed according to the following criteria: hypersplenism (platelet count <100×10^9/L) or gastroesophageal varices between the dates of January 2013 to December 2017 (Fig. 1). All selected patients had undergone splenectomy combined with periesophagogastric devascularization. Exclusion criteria were as follows: 1) liver tumor; 2) liver cirrhosis patients associated with preoperative PVT; 3) liver cirrhosis patients associated with congenital thrombotic disease or hematopoietic disease; 4) use of anticoagulation or anti-inflammation drugs; 5) severe organ

dysfunction; or 6) incomplete clinical information. Before surgical procedures, all patients or their relatives provided informed consent and the investigation was carried out in line with the principles of the Declaration of Helsinki (as revised in Fortaleza, Brazil, October 2013). The Ethics Committee of the Second Hospital of Nanjing approved the study protocol.

Laboratory tests

Blood specimens were collected from the peripheral vein. Data collected from blood tests included assessment of liver function, renal function, coagulation parameters, etiology of liver disease, and blood morphology. Electrocardiography, chest radiography, ultrasound examination, endoscopy of the upper gastrointestinal tract, and contrast-enhanced spiral computed tomography were performed on each patient before the operation. Cirrhosis was confirmed by pathological investigation postoperatively (post). The severity of cirrhosis was evaluated by the MELD and Child-Turcotte-Pugh (CTP) scores. Blood tests, including assessment of liver function, renal function, blood coagulation, and blood morphology, were conducted again on days 1, 3, and 7 after

the operation. Two independent radiologists evaluated the presence and extent of PVT, velocity of portal blood flow of PVT preoperatively (pre) and at day 7 post by color Doppler ultrasound examination. Besides, the basic demographic and clinical characteristics (age, gender, body mass index), etiology of liver cirrhosis, and emergency surgery were also recorded.

Definitions

PLR was defined as the absolute platelet count divided by lymphocyte count (10^9/L). NLR was the ratio of absolute neutrophils count to lymphocyte count (10^9/L). MLR was calculated as absolute monocyte count divided by lymphocyte ratio count (10^9/L), and the MPR as mean platelet volume divided by platelet count (10^9/L). While RPR referred to the ratio of red cell distribution width to platelet count (10^9/L). PNI was calculated as albumin (g/L) + 5× total lymphocyte count (10^9/L).[20]

Operation

Although the standard surgical procedure of splenectomy with periesophagogastric devascularization has been commonly described, we still need to make a brief statement. The open operation was performed by using an extended left subcostal incision. The routine splenectomy was firstly performed, and then periesophagogastric devascularization was performed. Firstly, the gastric branch of the right gastric vein near the gastric angular incisura and small branches of the gastric coronary veins were disconnected. Secondly, the esophageal branch (i.e. esophageal branch of the gastric coronary vein; high esophageal branch of the gastric coronary vein; aberrant high esophageal branch of the gastric coronary vein) was disconnected and suture-ligated, involving up to 10 cm of the esophageal inferior segment. The gastric posterior veins and short gastric veins were disconnected, and then the left subphrenic vein was also disconnected. In addition, the corresponding arteries, including the left gastric artery, left gastroepiploic artery, gastric posterior artery, and left subphrenic artery, were also ligated.

Statistical analysis

The measurement data were presented as mean±standard deviation (normal distribution). Statistically significant differences were evaluated with Student's *t*-test (normal distribution). Qualitative data were summarized as *n* (%), and statistically significant differences were evaluated using the chi-square test. Receiver operating characteristic curves were constructed to assess the indicative values of the inflammation biomarkers. The areas under the curves (AUCs) were calculated with 95% confidence intervals (CIs). The Youden index was applied to determine the optimal cutoff value for every indicator. Significant variables of PVT from univariate analysis were included in multivariate analysis when performing forward stepwise logistic regression modeling. Data were analyzed by SPSS version 16 (SPSS Inc., Chicago, IL, USA). A *p*-value of less than 0.05 was considered statistically significant.

Results

We collected data for 223 patients with splenectomy. In total, 46 patients were excluded for the following reasons:

hematopoietic disease (*n*=6); liver tumor (*n*=9); PVT pre (*n*=17); no portal vein evaluation pre and post (*n*=14). Finally, 177 patients who meet the criteria were enrolled in this study. The patients were divided into the PVT group (*n*=71) and the non-PVT group (*n*=106) according to the finding of PVT post. Baseline characteristics and clinical and laboratory parameters of the two groups are presented in Table 1. There were no significant differences between the two groups, with respect to gender, body mass index, etiology of liver cirrhosis, emergency surgery, portal blood flow velocity, CTP score, and MELD score.

Significant differences in basic characteristics of the patients, including age (*p*=0.027), PLR (*p*=0.007), NLR (*p*=0.035), MLR (*p*=0.037), and lymphocyte count (*p*=0.002), were observed between the two groups. On day 3 post, the PLR of the PVT group was higher than that of the non-PVT group (*p*=0.027). In day 7 post, PLR (*p*=0.001), MLR (*p*=0.023), PLT (*p*=0.030), and lymphocyte count (*p*=0.009) were significantly different among the groups. The data are illustrated in Table 2.

Youden index analysis showed the optimal cutoff points for NLR (pre), PLR (post-day 3), MLR (post-day 7), and PLT (post-day 7) were 3.7, 139, 1.055, and 263.5, respectively. The cutoff value for PLR (pre) was 70.5, with a sensitivity of 0.714 and a specificity of 0.614. The cutoff value for MLR (pre) was 0.295, with a sensitivity of 0.783 and a specificity of 0.434. The cutoff value for PLR (post-day 7) was 230.5, with a sensitivity of 0.657 and a specificity of 0.647 (Table 3). Receiver operating characteristic curve analysis identified the AUC for PLR (pre), MLR (pre), PLR (post-day 7), and PLR (pre) combined with MLR (pre) as 0.665, 0.618, 0.655, and 0.697, respectively (Fig. 2). Obviously, they were all better than the AUC values for NLR (pre) (0.600), PLR (post-day 3) (0.595), MLR (post-day 7) (0.607), and PLT (post-day 7) (0.604).

The univariate logistic regression analysis indicated that age [odds ratio (OR)=1.958, 95% CI=1.051–3.647, *p*=0.034], NLR (pre) (OR=2.969, 95% CI=1.417–6.220, *p*=0.004), PLR (pre) (OR=3.963, 95% CI=2.070–7.587, *p*<0.000), MLR (pre) (OR=2.760, 95% CI=1.386–5.497, *p*=0.004), PLR (post-day 3) (OR=2.615, 95% CI=1.342–5.098, *p*=0.005), PLR (post-day 7) (OR=3.345, 95% CI=1.767–6.332, *p*=0.000), MLR (post-day 7) (OR=2.567, 95% CI=1.312–5.022, *p*=0.006), and PLT (post-day 7) (OR=2.437, 95% CI=1.313–4.527, *p*=0.005) were significantly associated with the presence of PVT.

Multivariate logistic regression analysis was conducted to verify the predictive value of the factors including age, NLR (pre), PLR (pre), MLR (pre), PLR (post-day 3), PLR (post-day 7), MLR (post-day 7), and PLT (post-day 7). The results indicated that PLR (pre) (OR=3.037, 95% CI=1.463–6.305, *p*=0.003), MLR (pre) (OR=2.188, 95% CI=1.003–4.772, *p*=0.049), and PLR (post-day 7) (OR=2.166, 95% CI=1.053–4.454, *p*=0.036) were independent factors for predicting PVT (Table 4).

According to the cutoff values for PLR (pre) and MLR (pre), the patients were divided into the following three groups: PLR (pre) ≤70.5 with MLR (pre) ≤0.295; PLR (pre) ≤70.5 with MLR (pre) > 0.295 or PLR (pre) >70.5 with MLR (pre) ≤0.295; and, PLR (pre) >70.5 with MLR (pre) > 0.295. PLR (pre) ≤70.5 with MLR (pre) ≤0.295 was selected as reference. Multivariate logistic regression analysis was performed and showed that PLR (pre) >70.5 and MLR (pre) >0.295 were associated with the greatest predictive value between the three groups (Table 5).

Discussion

PVT after splenectomy and periesophagogastric devascu-

Table 1. Demographic and clinical characteristics of the study population at baseline

Variable	PVT, *n*=71	Non-PVT, *n*=106	*p*-value
Age in years	45.90±9.50	49.36±10.48	0.027
Gender, male/female	51/20	68/38	0.286
BMI	23.29±2.88	22.94±3.22	0.478
Etiology, hepatitis B/others	62/9	87/19	0.348
Emergency surgery, yes/no	23/48	26/80	0.252
Velocity of portal blood flow in cm/s	18.33±4.87	17.85±5.34	0.598
Thickness of spleen in mm	61.56±12.59	58.31±15.28	0.184
Longitudinal diameter of spleen in mm	184.84±31.55	169.81±22.87	0.053
CTP score, A/B/C	53/18/0	75/31/0	0.570
MELD score	10.94±2.37	10.77±2.36	0.639
PT in s	15.83±2.04	15.56±2.25	0.423
TBIL in µmol/L	22.88±13.20	23.52±11.64	0.732
DBIL in µmol/L	9.24±5.49	10.95±6.76	0.079
ALB in g/L	37.21±5.07	36.43±5.51	0.345
GLB in g/L	25.77±4.79	27.13±6.92	0.152
AKP in U/L	145.61±53.14	85.42±40.41	0.348
GGT in U/L	143.38±47.89	134.29±73.39	0.299
ACE	3,876.00±1,320.28	3,866.90±1,398.56	0.966
BUN in mmol/L	5.65±2.36	6.73±7.48	0.244
Cr in µmol/L	65.54±15.42	62.97±18.62	0.341
INR	1.37±0.17	1.34±0.20	0.278

ACE, acetylcholinestrase; AKP, alkaline phosphatase; ALB, albumin; BMI, body mass index; BUN, blood urea nitrogen; Cr, creatine; DBIL, direct bilirubin; GGT, glutamyl transpeptidase; GLB, globulin; INR, international normalized ratio; PT, prothrombin time; TBIL, total bilirubin.

larization is a life-threatening complication for its serious consequences involving the increased rate of re-bleeding, complicated liver transplantation technique, and deterioration of liver function.[25] The relationship between venous thromboembolism and inflammation response has been controversial, with it being unknown whether inflammation is casual in the development of venous thrombosis or instead a consequence of venous thrombosis. A growing body of data suggests that inflammation plays a vital role in the pathogenesis of venous thromboembolism.[13–17] The inflammatory process can influence coagulation from the following three aspects: down-regulation of physiological anticoagulant pathways, inhibition of fibrin removal, and initiation of coagulation activation.[26] This would result in the shift of hemostatic balance toward a prothrombotic state. Besides, inflammation may increase the damage of endothelial cells. Some inflammatory biomarkers, such as PLR, NLR, and LMR, have been confirmed as useful predictive measures of deep vein thrombus.[27–30]

There is no significant difference with respect to pre platelet count and monocyte count between the PVT group and the non-PVT group. Elevated levels of pre PLR and MLR in the PVT group primarily result from a decreased number of lymphocytes compared with that in the control group. The lymphocyte is the major cell component of the immune system that represents the immunomodulatory pathway and plays a crucial role in regulating systemic inflammation.[31] As systemic inflammation worsens, peripheral lymphocyte count becomes reduced as a result of cell apoptosis, necrosis, and redistribution. In hepatitis B virus-related acute-on-chronic liver failure, systemic inflammation is the result of depletion in circulating lymphocytes.[32] The lymphocyte count in the PVT group of our study was lower than that of the control group, indicating suppressed immunity. We hypothesized that the intestinal lumen bacterial products penetrated into the circulation in patients with advanced cirrhosis and portal hypertension due to depressed immunity. The combination of bacterial distribution and suppressed immunity ultimately may result in more critical portal vein and systemic inflammation in the PVT group. The inflammation of the vessel wall will initiate thrombus formation.

The elevated level of PLR (post-day 7) may primarily result from an increased platelet count and a decreased lymphocyte count compared to counts in the control group. We found the platelet count to be increasing gradually after splenectomy, and the platelet count of the PVT group in the 7-day post group to be significantly higher than that of the non-PVT group; there were no significant differences for that between the groups at pre and post-day 1 or post-day 3. Evidence suggest that platelets play a less important role in venous thrombosis than in arterial thrombosis. This was supported by the findings from pathological analysis, which showed the arterial thrombi to mainly consist of platelets and the venous thrombi to mainly consist of red blood cells and fibrin, at least initially. The involvement of platelets in the formation of venous thrombosis is slight at an early stage; at a later stage, platelets appear to play a slightly more major role because the subsequent layers of venous thrombi contain some platelets.[14] To concretely determine the roles of platelets in the formation of venous thrombosis more research is needed. This may be helpful for clini-

Table 2. Characteristics of inflammation biomarkers at pre and post-days 1, 3, and 7

Variables	PVT (pre)	Non-PVT (pre)	P-value	PVT (post-D1)	Non-PVT (post-D1)	*p*-value
WBC as 10^9/L	2.60±2.40	2.66±1.75	0.867	17.61±6.92	16.96±6.80	0.537
Neutrophils as 10^9/L	1.82±2.14	1.69±1.41	0.609	15.55±6.53	15.06±6.47	0.623
Lymphocytes as 10^9/L	0.51±0.25	0.67±0.37	0.002	0.69±0.45	0.65±0.37	0.526
Monocytes as 10^9/L	0.22±0.14	0.23±0.14	0.720	1.29±0.76	1.20±0.63	0.403
PLT as 10^9/L	44.39±25.68	43.74±24.90	0.869	83.01±29.72	82.69±30.02	0.944
PNI	39.86±5.06	39.62±6.02	0.780	39.48±4.50	38.59±4.55	0.205
PLR	92.61±43.68	73.72±46.13	0.007	175.92±176.30	168.51±112.74	0.735
NLR	3.63±3.21	2.70±2.18	0.035	37.78±59.96	32.26±29.37	0.421
MLR	0.46±0.25	0.38±0.24	0.037	2.69±2.62	2.36±1.87	0.322
RPR	0.49±0.23	0.47±0.21	0.411	0.25±0.14	0.23±0.12	0.307
MPR	0.30±0.14	0.32±0.17	0.334	0.180±0.181	0.16±0.08	0.376

| Variables	PVT (post-D3)	Non-PVT (post-D3)	*p*-value	PVT (post-D7)	Non-PVT (post-D7)	*p*-value
WBC as 10^9/L	13.64±4.75	14.03±5.04	0.608	11.30±4.20	11.65±4.96	0.628
Neutrophils as 10^9/L	11.21±4.40	11.49±4.70	0.688	8.45±3.67	8.64±4.45	0.770
Lymphocytes as 10^9/L	0.84±0.43	0.93±0.51	0.266	1.00±0.41	1.19±0.52	0.009
Monocytes as 10^9/L	1.37±0.65	1.36±0.63	0.927	1.45±0.69	1.39±0.67	0.552
PLT as 10^9/L	143.35±54.45	130.39±64.60	0.162	275.17±122.08	233.98±123.30	0.030
PNI	39.59±3.69	40.09±4.55	0.458	40.56±4.19	40.75±5.50	0.811
PLR	230.60±160.50	182.03±121.49	0.027	319.23±192.50	223.99±137.22	0.001
NLR	17.19±11.53	15.44±10.42	0.311	9.16±4.74	8.34±5.38	0.301
MLR	2.04±1.23	1.77±1.25	0.166	1.60±0.87	1.31±0.77	0.023
RPR	0.147±0.083	0.153±0.089	0.651	0.08±0.05	0.10±0.08	0.155
MPR	0.096±0.062	0.108±0.057	0.214	0.05±0.04	0.07±0.05	0.071

WBC, white blood cell count.

cians to determine when and which anticoagulation therapy should be recommended. The result indicates that taking an anti-platelet drug from the post-day 7 time point should be an alternative.

The use of anticoagulation for PVT is strongly recommended because of the fact that spontaneous recanalization of PVT rarely happens; however, this therapy is associated with anticoagulation-related bleeding. Which subgroup of patients who undergo surgery and will benefit most from anticoagulant therapy remains an unresolved issue. Early selection of appropriate patients is critical. The two inflammation biomarkers, pre PLR and MLR, are significantly asso-

ciated with a diagnosis of PVT. Further analysis demonstrated that with a combination index of pre PLR >70.5 and MLR >0.295, the risk of PVT increased 8.148-fold compared with that of PLR ≤70.5 and MLR≤ 0.295. Patients with PLR >70.5 and MLR >0.295 are at high risk for development of post PVT. The increased PLR and MLR preoperatively may reflect a serious thrombus burden. As the thrombus burden becomes aggravated, the risk of PVT becomes increased. This contributes to enhancing our ability to identify the high-risk population and provide a bias for clinic intervention at an early stage. Resveratrol has been demonstrated to reduce the incidence of PVT after splenectomy in an animal model,

Table 3. Receiver operating characteristics curve of predictive variables for patients with PVT

Variable	Cutoff value	AUC (95%CI)	Sensitivity, %	Specificity, %	*p*-value
NLR (pre)	3.7	0.600 (0.515–0.685)	32.9	85.8	0.025
PLR (pre)	70.5	0.665 (0.585–0.746)	71.4	61.4	0.000
MLR (pre)	0.295	0.618 (0.534–0.703)	78.3	43.4	0.008
PLR (post-day 3)	139	0.595 (0.508–0.682)	73.1	48	0.037
PLR (post-day 7)	230.5	0.655 (0.571–0.739)	65.7	64.7	0.001
MLR (post-day 7)	1.055	0.607 (0.523–0.692)	75.4	45.6	0.017
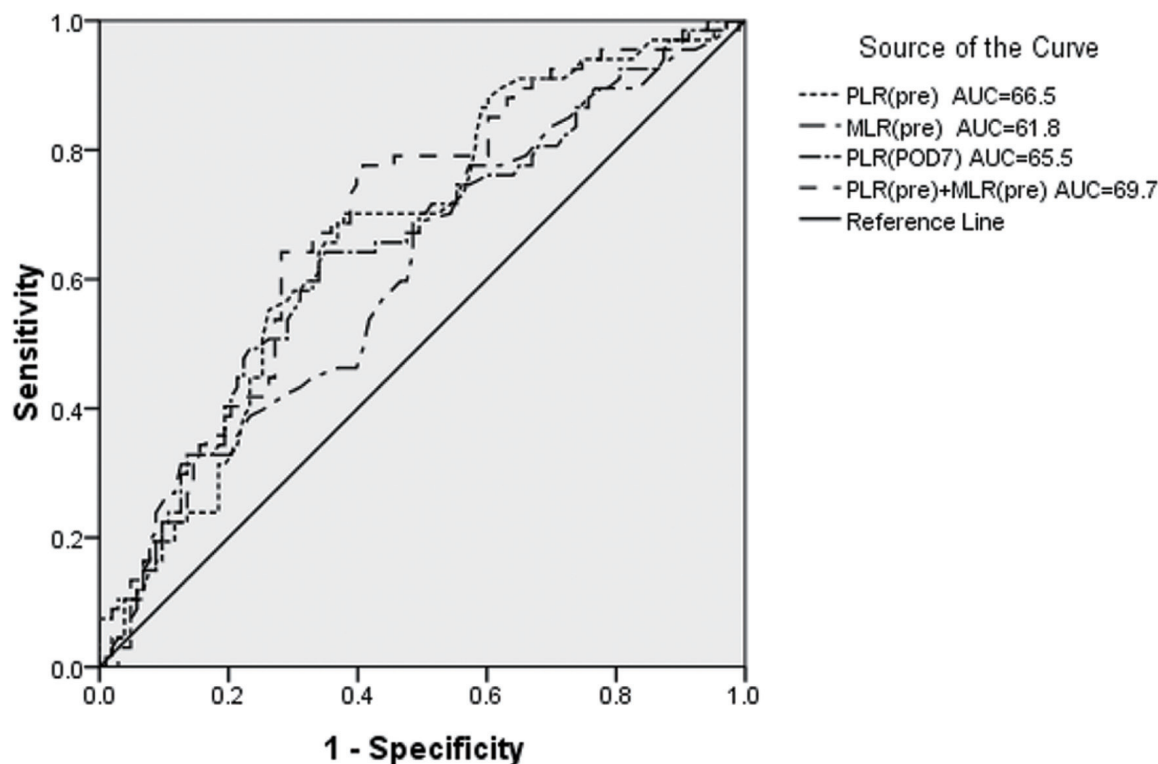
Fig. 2. ROC curve analysis for predicting PVT by PLR (pre), MLR (pre), PLR (post-D7) and combined markers in the estimation cohort. ROC, receiver operating characteristic.

via a regulation of platelet function and induction of platelet apoptosis.[33] Besides that, statins have been used as anti-thrombotic therapy for their anti-inflammatory effect.[34] This type of therapy could decrease the rate of venous throm-bosis via the reduction of proinflammatory cytokines and chemokines. However, the current guideline of anticoagu-lation management did not recommend the treatment of inhibiting inflammation. This study may help to suggest the

Table 4. Predictive variables of portal vein thrombosis by univariate and multivariate analyses

Variable	Univariate analysis		Multivariate analysis	
	OR (95% CI)	*p*-value	OR (95% CI)	*p*-value
Age (≤50 vs. >50 years)	1.958 (1.051–3.647)	0.034		
NLR (pre) (>3.7 vs. ≤3.7)	2.969 (1.417–6.220)	0.004		
PLR (pre) (>70.5 vs. ≤70.5)	3.963 (2.070–7.587)	0.000	3.037 (1.463–6.305)	0.003
MLR (pre) (>0.295 vs. ≤0.295)	2.760 (1.386–5.497)	0.004	2.188 (1.003–4.772)	0.049
PLR (post-day 3) (>139 vs. ≤139)	2.615 (1.342–5.098)	0.005		
PLR (post-day 7) (>230.5 vs. ≤230.5)	3.345 (1.767–6.332)	0.000	2.166 (1.053–4.454)	0.036
MLR (post-day 7) (>1.055 vs. ≤1.055)	2.567 (1.312–5.022)	0.006		

Table 5. Multivariate logistic regression analysis of predictive variables

Variable	OR	95%CI	P-value
PLR (pre) ≤70.5 and MLR (pre) ≤0.295			
PLR (pre) >70.5 and MLR (pre) ≤0.295	2.750	1.008–7.502	0.048
PLR (pre) ≤70.5 and MLR (pre) >0.295			
PLR (pre) >70.5 and MLR (pre) >0.295	8.148	3.005–22.093	<0.000

role of anti-inflammation therapy as an optimal prophylactic strategy. The identification of inflammation markers relevant to the formation of PVT could provide definite targets for future therapy.

In this study, we found that PLR (pre), MLR (pre), and PLR (post-day7) are predictors of PVT post in patients undergoing splenectomy and periesophagogastric devascularization. Some potential limitations of this study should be noted. Firstly, this is a retrospective study performed in a single-center, and additional prospective and multicenter studies are needed. Secondly, the portal vein was assessed only on the seventh day after surgery. Dynamic observation of portal vein is proposed, as changes of inflammation biomarkers may represent an after-effect. Thirdly, former research confirmed that the anatomic extent of deep vein thrombosis was associated with changes of inflammation marker levels;[12] the PVT group was not divided into any subgroups according to the extent of PVT.

Acknowledgments

Conflict of interest

The authors have no conflict of interests related to this publication.

Author contributions

Study concept and design (YXY, YFZ), acquisition of data (JBH, QHS), analysis and interpretation of data (JBH, QHS, YFZ, YXY), drafting of the manuscript (JBH, QHS), critical revision of the manuscript for important intellectual content (JBH, QHS), administrative, technical, or material support, study supervision (YXY, YFZ).

Data sharing statement

All data are available upon request.

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