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ABOUT COVER

Editorial Board Member of *World Journal of Hepatology*, Nobuyuki Takemura, MD, PhD, FACS, Professor, Department of Hepato-Biliary-Pancreatic Surgery and Pediatric Surgery, Saitama Medical Center, Saitama Medical University, Saitama 350-8550, Japan. takemuranobu-ky@umin.ac.jp

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Observational Study

Predictors of portal vein thrombosis after splenectomy in patients with cirrhosis

Ting Li, Li-Li Wang, Ya-Ping Li, Jian Gan, Xi-Sheng Wei, Xiao-Rong Mao, Jun-Feng Li

Specialty type: Gastroenterology and hepatology**Provenance and peer review:** Unsolicited article; Externally peer reviewed.**Peer-review model:** Single blind**Peer-review report's scientific quality classification**Grade A (Excellent): 0
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Portal vein thrombosis (PVT) is a common complication after splenectomy in patients with cirrhosis. However, the predictors of postoperative PVT are not known.

AIM

To investigate the predictors of PVT after splenectomy in patient with cirrhosis.

METHODS

A total of 45 patients with cirrhosis who underwent splenectomy were consecutively enrolled from January 2017 to December 2018. The incidence of PVT at 1 months, 3 months, and 12 months after splenectomy in patients with cirrhosis was observed. The hematological indicators, biochemical and coagulation parameters, and imaging features were recorded at baseline and at each observation point. The univariable, multivariable, receiver operating characteristic curve and time-dependent curve analyses were performed.

RESULTS

The cumulative incidence of PVT was 40.0%, 46.6%, and 48.9% at 1 months, 3 months, and 12 months after splenectomy. Multivariable analysis showed that portal vein diameter (PVD) ≥ 14.5 mm and months since end-stage liver disease

(MELD) score > 10 were independent predictors of PVT at 1 months, 3 months, and 12 months after splenectomy ($P < 0.05$). Time-dependent curve showed that the cumulative incidence of PVT was significantly different between patients with MELD score ≤ 10 and > 10 ($P < 0.05$). In addition, the cumulative incidence of PVT in the PVD ≥ 14.5 mm group was significantly higher than that in the PVD < 14.5 mm group ($P < 0.05$).

CONCLUSION

Wider PVD and MELD score > 10 were independent predictors of PVT at 1 months, 3 months, and 12 months after splenectomy in patient with cirrhosis.

Key Words: Cirrhosis; Splenectomy; Portal vein thrombosis; Predictors

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Core Tip: Portal vein diameter (PVD) ≥ 14.5 mm was independent predictors of portal vein thrombosis (PVT) at 1-months, 3-months, and 12-months after splenectomy. End-stage liver disease score > 10 was independent predictors of PVT at 1-months, 3-months, and 12-months after splenectomy. The patients with PVD ≥ 14.5 mm and/or end-stage liver disease score > 10 in preoperative, preoperative treatment of reducing portal vein pressure and improving liver function may help to reduce the incidence of PVT after splenectomy.

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INTRODUCTION

Portal vein thrombosis (PVT) involves the portal vein and its main branches, and cirrhosis is one of the most common causes. The natural incidence of PVT in cirrhosis is 3.7%-24.4% [1], and the incidence of advanced cirrhosis is 10%-15% [2]. In cirrhosis, PVT is often latent, and is only discovered accidentally. The treatment of PVT with cirrhosis is controversial.

Currently, splenectomy is one of the main methods of treatment for portal hypertension, hypersplenism and upper gastrointestinal bleeding. Splenectomy can significantly improve the prognosis and survival of patients with cirrhosis [3, 4]. Splenectomy decreases portal hypertension, improves liver function, and reduces fibrosis [5-7]. It also improves liver regeneration. A 10-year retrospective follow-up study based on the inverse probability of treatment weighting method found that splenectomy decreased the risk of hepatocellular carcinoma in cirrhosis patients with portal-hypertension-related bleeding [8]. Splenectomy has been considered an effective option to reverse thrombocytopenia in cirrhosis patients with splenomegaly. Thus, splenectomy may be beneficial for treatment of liver cirrhosis with hypersplenism. Splenectomy have been widely used in Asia for the treatment of esophagogastric variceal hemorrhage and hypersplenism caused by cirrhotic portal hypertension. However, splenectomy can increase the risk of PVT at least 10 times [9]. The incidence of PVT was 18.9%-57.0% after splenectomy, which was significantly higher than the natural incidence in patients with cirrhosis without surgery [2, 10]. PVT can induce or aggravate upper gastrointestinal bleeding, hepatic encephalopathy, and ascites, increase the risk of intestinal ischemia or intestinal necrosis, reduce the survival of patients and grafts after liver transplantation, and result in chronic cavernous transformation of the portal vein system in the long term [11-14].

Alteration in blood flow, hypercoagulability and vascular endothelial injury are the main risk factors for PVT [2]. PVT is associated with preoperative slower portal vein velocity, wider portal vein diameter (PVD) and splenic vein, and lower preoperative and higher postoperative platelet counts [15-18]. A higher end-stage liver disease (MELD) score is associated with hepatic encephalopathy, variceal bleeding, refractory ascites, and spontaneous peritonitis [19-21]. Higher MELD score corresponds with higher mortality in liver transplantation [20]. A higher of MELD score may be associated with postoperative PVT. The mechanisms of PVT after splenectomy are still unclear. Our study aimed to establish the risk factors for PVT after splenectomy and early sensitive indicators, to provide a predictive basis for early PVT.

MATERIALS AND METHODS

Patients

We enrolled 45 consecutive patients with cirrhosis who underwent splenectomy between January 2017 and December 2018 at the First Hospital of Lanzhou University. The flow diagram of the study population is shown in [Figure 1](#). The

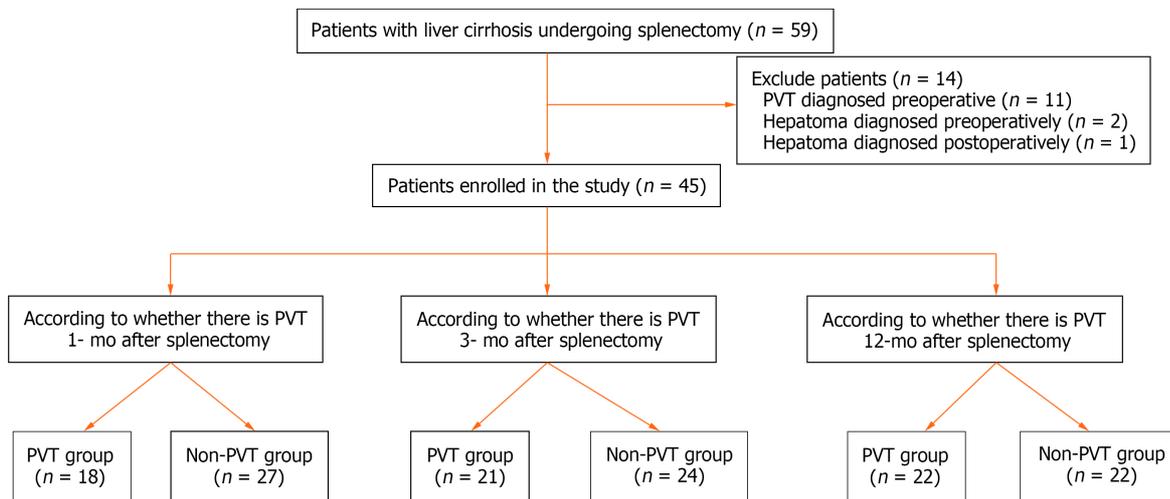


Figure 1 Flowchart of the study population. PVT: Portal vein thrombosis.

study was approved by the ethics committee of the first hospital of Lanzhou University (LDYYLL2019-209) and informed consent was obtained from the patients.

Inclusion criteria were histologically proven cirrhosis or cirrhosis diagnosed by a history of liver disease, clinical manifestations, laboratory tests, and imaging studies, and the patients underwent splenectomy. The indications for splenectomy included: Endoscopic treatment-resistant esophagogastric varices with or without variceal hemonthsrrhage; history of esophageal variceal bleeding or potential bleeding; infection caused by hypersplenism and thrombocytopenia (platelet count $< 50 \times 10^9/L$); and upper abdominal discomfort caused by an enlarged spleen.

The exclusion criteria were as follows: (1) Age > 18 years or < 70 years; (2) patients who developed PVT preoperatively; (3) patients who presented preoperatively with hepatic carcinoma, hepatic encephalopathy, or preoperative Child-Pugh class C, or other tumonthsrs; (4) patients with cirrhosis who underwent liver transplantation; (5) patients who underwent transjugular portal systemic shunt; (6) patients who underwent abdominal surgery; (7) splenectomy for hematological diseases and other reasons (such as trauma); (8) vascular malformation and idiopathic portal hypertension; (9) incomplete clinical data (without hemonthsocytes, imaging and other relevant data); (10) coexistence of other serious diseases (shock, multiple organ failure, uremia, and severe infection); and (11) loss to follow-up.

Diagnosis of PVT

PVT was detected by duplex ultrasonography, computed tomography, or computed tomography angiography. They were performed within 1 wk before the operation to exclude preoperative PVT. Re-examination was performed at 1 months, 3 months, 6 months, and 12 months postoperatively.

Laboratory tests

Routine blood, parameters and coagulation parameters were measured within 3 d before the operation and used as baseline data. Re-examinations were performed at 1 months, 3 months, 6 months, and 12 months postoperatively in the outpatient or inpatient department of the First Hospital of Lanzhou University. BC-5390 CRP automatic blood cell analyzer (Mindray Bio-Medical Electronics Co. Ltd., Shenzhen, Guangdong Province, China) was used for routine blood testing. The AU400 automatic biochemical analyzer (Olympus Optics Co. Ltd., Japan) was used to detect biochemical parameters. Coagulation parameters were detected by PreciC3510 automatic coagulation analyzer (Mindray). PVD was measured using Doppler ultrasound (GE Logic E9).

Statistical analysis

An independent *t* test or single factor analysis was used to analyze the difference in data in accordance with a normal distribution, and the Mann-Whitney test was used to analyze non-normally distributed data. The χ^2 test or Fisher's exact test was used to analyze categorical variables. A logistic regression monthsdel analyzed the multivariable data. A receiver operating characteristic (ROC) curve was used to evaluate the specificity and sensitivity of PVD and MEDL score for predicting PVT. The Kaplan-Meier method was used to calculate the cumulative incidence of PVT, and the log-rank test was used to compare the difference in the cumulative incidence of PVT between the groups.

RESULTS

Basic characteristics

We included 45 consecutive patients with cirrhosis who underwent splenectomy (Table 1). The mean age was 47.62 years ± 11.16 years, and 53.3% were female. In terms of etiology, 84% of patients with cirrhosis had hepatitis B, 4.0% hepatitis C,

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

Variables	Data
Gender	
Men, <i>n</i> (%)	21 (46.7)
Female, <i>n</i> (%)	24 (53.3)
Age (yr)	47.62 ± 11.16
Etiology	
HBV, <i>n</i> (%)	37 (74.0)
HCV, <i>n</i> (%)	2 (4.0)
AIH, <i>n</i> (%)	4 (8.0)
Unexplained cirrhosis, <i>n</i> (%)	2 (4.0)
WBC (10 ⁹ /L)	2.21 ± 1.18
RBC (10 ¹² /L)	3.68 ± 0.78
PLT (10 ⁹ /L)	44.09 ± 28.54
AST (U/L)	35.18 ± 14.82
ALT (U/L)	26.89 ± 14.30
TBIL (μmol/L)	26.77 ± 15.97
ALB (g/L)	38.81 ± 4.16
INR	1.30 ± 0.19
PT (s)	14.23 ± 1.94
PTA (%)	68.71 ± 13.45
D-D2 (mg/L)	0.56 ± 1.03
MELD	10.76 ± 3.30
PVD (mm)	13.66 ± 2.49
PV (cm/s)	19.87 ± 5.88
SVD (mm)	10.35 ± 2.61
Ascites, <i>n</i> (%)	19 (42.2)
Child-Pugh grade	
A, <i>n</i> (%)	32 (71.1)
B, <i>n</i> (%)	13 (28.9)
C, <i>n</i> (%)	0 (0.0)
Operation ways	1.30 ± 0.19
Open splenectomy, <i>n</i> (%)	35 (78.8)
Laparoscopic splenectomy, <i>n</i> (%)	10 (21.2)
Spleen volume (cm ³)	2349.28 (531.12-13080.00)

HBV: Hepatitis B virus; HCV: Hepatitis C virus; AIH: Autoimmune hepatitis; WBC: White blood cell counts; RBC: Red blood cell counts; PLT: Platelet counts; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; TBIL: Total bilirubin; ALB: Albumin; INR: International normalized ratio; PT: Prothrombin time; PTA: Prothrombin activity; MELD: Monthsdel for end-stage liver disease; PVD: Portal vein diameter; PV: Portal vein velocity; SVD: Splenic vein.

8.0% autoimmune hepatitis, and 4.0% unexplained cirrhosis. Thirty-five (78.8%) liver cirrhosis patients underwent open splenectomy, and 10 (21.2%) underwent laparoscopic splenectomy.

There were 18 (40.0%) patients with PVT and 27 (60.0%) without PVT at 1 months after splenectomy; 21 (46.6%) with PVT and 27 (53.4%) without PVT at 3 months after splenectomy; and 22 (48.9%) with PVT and 27 (61.1%) without PVT at 12 months after the operation.

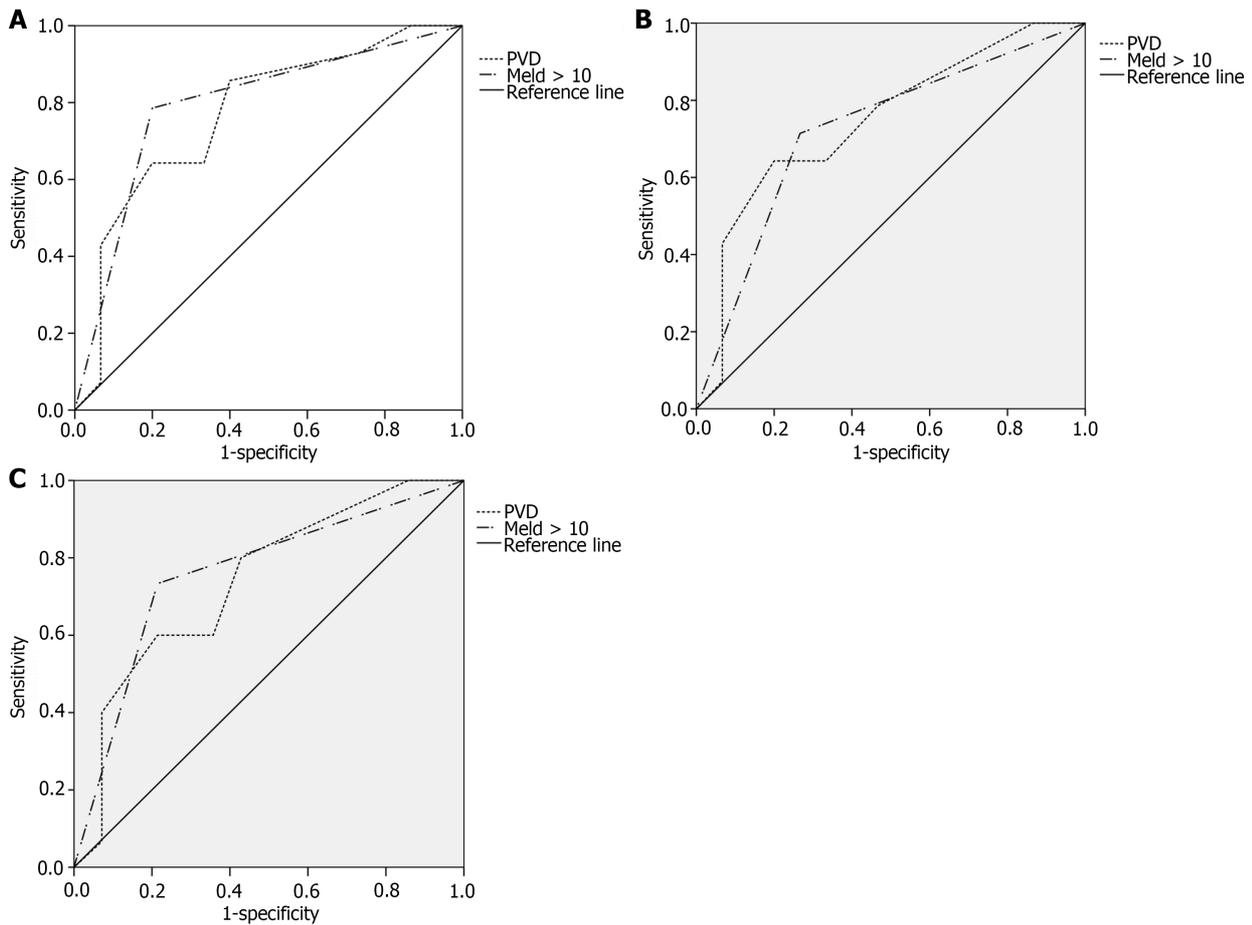


Figure 2 Receiver operating characteristic curve. A: Receiver operating characteristic (ROC) curve of portal vein thrombosis (PVT) in patients with cirrhosis after splenectomy predicted by independent predictors (postoperative 1 months); B: ROC curve of PVT in patients with cirrhosis after splenectomy predicted by independent predictors (postoperative 3 months); C: ROC curve of PVT in patients with cirrhosis after splenectomy predicted by independent predictors (postoperative 12 months). PVT: Portal vein thrombosis; MELD: Monthsdel end-stage liver disease.

There were 18 (40.0%) patients with ascites at 1 wk after splenectomy; six (13.3%) patients with ascites at 1 months after splenectomy; one (2.2%) patient with hepatic encephalopathy, four (8.9%) with ascites, and one (2.2%) with upper gastrointestinal hemonthsrrhage at 3 months after splenectomy; one (2.2%) patient with upper gastrointestinal hemonthsrrhage at 6 months after splenectomy; and no decompensation occurred 1 year after the operation. During 1-year follow-up, there was no postoperative monthsrtality.

Risk factors of development of PVT after splenectomy in patients with cirrhosis

The demonthsgraphic and laboratory data were compared in patients who developed PVT at 1 months, 3 months, and 12 months postoperatively. Univariable analysis revealed that the incidence of PVT at 1 months, 3 months, and 12 months postoperatively in the MELD score > 10 group was significantly higher than in the MELD score ≤ 10 group ($P < 0.05$). Patients with PVT had a wider PVD than those without PVT ($P < 0.05$). At 3 months postoperatively, the PVT group had a longer prothrombin time (PT) ($P < 0.05$).

Independent predictors of PVT after splenectomy in cirrhotic patients

Multivariable analysis identified the following as independent predictors of PVT at 1 months, 3 months, and 12 months postoperatively: Wider preoperative PVD [odds ratio (OR): 2.194, 95% confidence interval (CI): 1.090-4.415, $P = 0.028$; OR: 1.70, 95% CI: 1.052-2.746, $P = 0.030$; OR: 1.776, 95% CI: 1.036-3.046, $P = 0.037$]; and MELD score > 10 (OR: 76.215, 95% CI: 2.534-2287.318, $P = 0.013$; OR: 12.392, 95% CI: 1.318-116.548, $P = 0.028$; OR: 23.925, 95% CI: 1.875-305.323, $P = 0.015$) (Table 2).

To evaluate the ability of independent predictors to predict PVT after splenectomy, ROC curve analysis was performed (Figure 2). The AUCs of PVD were 0.769, 0.745, and 0.738, respectively ($P < 0.05$). The AUCs of MELD score > 10 was 0.793, 0.724, and 0.760, respectively ($P < 0.05$).

Cumulative incidence of PVT

The mean time to occurrence of PVT after splenectomy was 27 d. The optimal cut-off value of PVD was 14.5 mm. The time-dependent curve analysis is shown (Figure 3).

Table 2 Independent risk factors of portal vein thrombosis formation in patients with liver cirrhosis after splenectomy

Variable	PVT	Non-PVT	P value	OR	95%CI
Postoperative 1-month					
Age	45.72 ± 7.84	48.89 ± 12.91			
PVD	14.79 ± 2.08	12.60 ± 2.38	0.028	2.194	1.090-4.415
MELD > 10	12 (70.6)	8 (32.0)	0.013	76.215	2.534-2287.318
PLT	47.00 ± 41.96	42.15 ± 14.78			
Postoperative 3-months					
Age	44.71 ± 7.96	44.71 ± 7.96			
PT	14.69 ± 1.94	14.69 ± 1.94			
PVD	14.71 ± 2.16	14.71 ± 2.16	0.03	1.7	1.052-2.746
Meld > 10	12 (70.6)	8 (32.0)	0.028	12.392	1.318-116.548
PLT	47.57 ± 38.80	41.04 ± 15.11			
Postoperative 12-months					
Age	44.64 ± 7.77	50.4 ± 13.19			
PVD	14.60 ± 2.13	12.64 ± 2.47	0.037	1.776	1.036-3.046
MELD > 10	14 (63.6)	6 (26.1)	0.015	23.925	1.875-305.323
PLT	46.55 ± 38.17	41.74 ± 15.05			

PVT: Portal vein thrombosis; OR: Odd ratio; CI: Confidence interval; PVD: Portal vein diameter; MELD: Monthsdel for end-stage liver disease; PLT: Platelet counts.

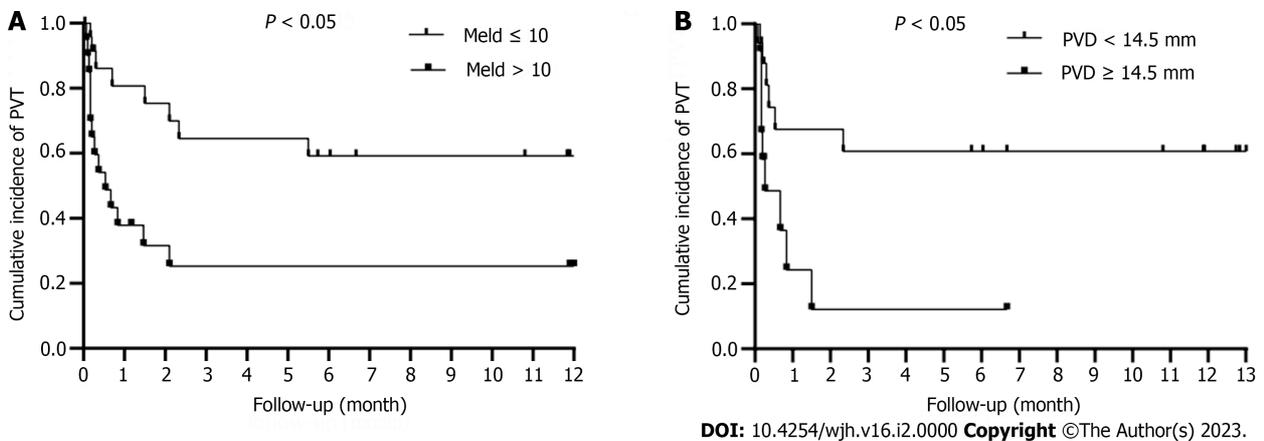


Figure 3 Time-dependent curve analysis. A: Cumulative incidence of portal vein thrombosis in the groups with monthsdel end-stage liver disease score ≤ 10 and > 10 was significantly different ($P < 0.05$); B: Cumulative incidence of PVT in patients with portal vein diameter (PVD) < 14.5 mm and PVD ≥ 14.5 mm group was significantly different ($P < 0.05$). PVT: Portal vein thrombosis; MELD: Monthsdel end-stage liver disease.

DISCUSSION

In our observational study, the cumulative incidence of PVT after splenectomy in patients with cirrhosis was 40.0%, 46.6%, 46.6%, and 48.9% at 1 months, 3 months, 6 months, and 12 months, respectively. Wider preoperative PVD and MELD score > 10 may predict the development of PVT after splenectomy. The time-dependent curve analyzed that the development of PVT in patients with MELD score ≤ 10 was lower than in those with MELD score > 10 ($P < 0.05$). And in the PVD ≥ 14.5 mm group was significantly higher than that in the PVD < 14.5 mm group ($P < 0.05$).

The cumulative postoperative incidence of PVT was 40.0% at 1 months, 46.6% at 3 months, 46.6% at 6 months, and 48.9% at 12 months. This was similar to the previous study. There are several potential causes of postoperative PVT. Firstly, the occlusion of splenic portal vessels resulted in a reduction in blood flow around the ligation area and enhanced the venous stasis at the splenic vein stump. Secondly, patients with liver cirrhosis are often complicated with changes in blood coagulation proteins, including factor VIII, von Willebrand factor fibrinogen, and tissue factor, putting the blood in

a hypercoagulable state[22], which is involved in venous thrombosis. Thirdly, splenectomy can reduce the portal vein flow velocity[23]. The lack of the portal vein flow velocity in our study, we did not obtain a similar conclusion. Previous reports found that wider preoperative splenic vein diameter was an independent predictor of the development of PVT[15, 24]. In our study, we found that diameter of the splenic vein in the PVT group was wider than that in the non-PVT group, but there was no significant difference.

Zhang *et al*[25] considered that the main cause of PVT was the change in portal vein blood flow and not the change in PT or platelet count. We found that a lower preoperative platelet count was not associated with the postoperative development of PVT. In our study, wider PVD was an independent predictor of PVT at 1 months, 3 months, and 12 months after splenectomy. The optimal cut-off value was 14.5 mm. Previous studies reported that PVD > 13.0 mm and > 15.6 mm were independent predictors of PVT after splenectomy[10,26]. Wider PVD means portal hypertension and slower blood flow velocity toward the liver. In addition, a wider PVD can cause a vortex, increase portal vein endothelial cell space, and result in intimal injury and sclerotic changes. The detachment of endothelial cells and the exposure of subintimal collagen fibers activate the endogenous coagulation pathway, increasing the incidence of thrombosis[16,18, 27]. Our study provided a favorable indicator for the prediction of PVT after splenectomy.

MELD score is an indicator of the severity of chronic liver disease and the monthsrality risk of patients with end-stage liver disease. Patients with liver cirrhosis, liver cancer and liver transplantation have different MELD scores, and their prognosis is different[14,28-30]. Previous studies found that a higher MELD score was closely associated with the development of PVT after splenectomy[31]. Our study found that the cumulative incidence of PVT in the MELD score > 10 group was significantly higher than in the MELD score ≤ 10 group. The liver can synthesize coagulation factors and fibrinolytic and antifibrinolytic substances, and inactivate fibrinolysis and antifibrinolytic substances, which play an important role in maintaining the balance of procoagulation and anticoagulation systems. However, the synthesis and inactivation of patients with liver cirrhosis are weakened. Zocco *et al*[23] found that the reduction in antithrombotic proteins and activation of the hemonthsstatic system were associated with the severity of cirrhosis. Abdel-Razik *et al*[31] drew the same conclusion. The development of coagulation is associated with the severity of cirrhosis. The balance of the coagulation system in patients with cirrhosis is weak, and this balance is monthsre easily broken after splenectomy. PVT is a marker of portal hypertension and advanced liver cirrhosis, and not a cause. In addition, the MELD score can independently predict PVT recanalization in patients with cirrhosis[32]. The preoperative MELD score can be used as a predictor of postoperative PVT. Preoperative liver function improvement may reduce the incidence of postoperative PVT. Therefore, we should implement splenectomy in patients with good liver function as much as possible.

There were some limitations to our study. Firstly, some patients did not undergo scheduled examinations, and there may have been errors in judgment of the formation time of PVT. Secondly, our study population was small. Thirdly, our study lacked anticoagulation therapy data. However, monthsre prospective, large, randomized studies are needed to assess the risk of development of PVT after splenectomy and provide evidence for anticoagulation therapy.

CONCLUSION

In conclusion, wider PVD and MELD score > 10 were independent predictors of the development of PVT at 1 months, 3 months, and 12 months after splenectomy in patients with cirrhosis.

ARTICLE HIGHLIGHTS

Research background

Splenectomy has been considered an effective option to reverse thrombocytopenia in cirrhosis patients with splenomegaly. Thus, splenectomy have been widely used in Asia for the treatment of esophagogastric variceal hemonthsrrhage and hypersplenism caused by cirrhotic portal hypertension. However, splenectomy can increase the risk of portal vein thrombosis (PVT) at least 10 times. The incidence of PVT was 18.9%-57.0% after splenectomy, which was significantly higher than the natural incidence in patients with cirrhosis without surgery. PVT can induce or aggravate upper gastrointestinal bleeding, hepatic encephalopathy, and ascites, increase the risk of intestinal ischemia or intestinal necrosis, reduce the survival of patients and grafts after liver transplantation, and result in chronic cavernous transformation of the portal vein system in the long term.

Research monthstivation

Splenectomy plays an important role in the treatment of cirrhosis. Splenectomy is widely used for the treatment of esophagogastric variceal haemonthsrrhage and hypersplenism owing to cirrhotic portal hypertension. However, splenectomy can increase the risk of PVT at least 10 times. Our study aims to seek the risk factors of PVT after splenectomy and early sensitive indicators, to provide a predictive basis for early PVT and reduce the incidence of PVT.

Research objectives

To establish the risk factors for PVT after splenectomy and early sensitive indicators, to provide a predictive basis for early PVT.

Research methods

A total of 45 patients with cirrhosis who underwent splenectomy were consecutively enrolled from January 2017 to December 2018. The incidence of PVT at 1 months, 3 months, and 12 months after splenectomy in patients with cirrhosis was observed. The hematological indicators, biochemical and coagulation parameters, and imaging features were recorded at baseline and at each observation point. The univariable, multivariable, receiver operating characteristic curve and time-dependent curve analyses were performed.

Research results

PVD ≥ 14.5 mm and monthsdel end-stage liver disease (MELD) > 10 were independent predictors of PVT at 1-months, 3-months, and 12-months after splenectomy. The patients with PVD ≥ 14.5 mm and/or MELD > 10 in preoperative, preoperative treatment of reducing portal vein pressure and improving liver function may help to reduce the incidence of PVT after splenectomy. However, monthsre large-scale studies will be needed to provide reliable and effective evidence for the specific time, drug selection and dosage of anticoagulants.

Research conclusions

Portal vein diameter (PVD) ≥ 14.5 mm was independent predictors of PVT at 1-months, 3-months, and 12-months after splenectomy. End-stage liver disease score > 10 was independent predictors of PVT at 1-months, 3-months, and 12-months after splenectomy. The patients with PVD ≥ 14.5 mm and/or end-stage liver disease score > 10 in preoperative, preoperative treatment of reducing portal vein pressure and improving liver function may help to reduce the incidence of PVT after splenectomy.

Research perspectives

How to prophylactic anticoagulation therapy after splenectomy? Anticoagulant therapy of PVT should be explored.

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FOOTNOTES

Co-first authors: Ting Li and Li-Li Wang.

Co-corresponding authors: Jun-Feng Li and Xiao-Rong Mao.

Author contributions: Li T and Wang LL contributed equally to this work; Li JF and Mao XR designed the research study; Li T and Wang LL performed the research; Li YP, Gan J and Wei XS contributed new reagents and analytic tools; Li T and Wang LL analyzed the data and wrote the manuscript; all authors have read and approve the final manuscript. Li T and Wang LL contributed equally to this work as co-first authors; Li JF and Mao XR contributed equally to this work as co-corresponding authors. The reasons for designating Li JF and Mao XR as co-corresponding authors are threefold. First, the research was performed as a collaborative effort, and the designation of co-corresponding authorship accurately reflects the distribution of responsibilities and burdens associated with the time and effort required to complete the study and the resultant paper. This also ensures effective communication and management of post-submission matters, ultimately enhancing the paper's quality and reliability. Second, the overall research team encompassed authors with a variety of expertise and skills from different fields, and the designation of co-corresponding authors best reflects this diversity. This also promonthsthes the monthsst comprehensive and in-depth examination of the research topic, ultimately enriching readers' understanding by offering various expert perspectives. Third, Li JF and Mao XR contributed efforts of equal substance throughout the research process. The choice of these researchers as co-corresponding authors acknowledges and respects this equal contribution, while recognizing the spirit of teamwork and collaboration of this study. In summary, we believe that designating Li JF and Mao XR as co-corresponding authors of is fitting for our manuscript as it accurately reflects our team's collaborative spirit, equal contributions, and diversity.

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Country/Territory of origin: China

ORCID number: Ting Li [0000-0002-9716-0515](https://orcid.org/0000-0002-9716-0515); Li-Li Wang [0000-0002-5348-565X](https://orcid.org/0000-0002-5348-565X); Ya-Ping Li [0000-0002-0900-5559](https://orcid.org/0000-0002-0900-5559); Jian Gan [0000-0003-2645-6076](https://orcid.org/0000-0003-2645-6076); Xiao-Rong Mao [0000-0003-1952-1554](https://orcid.org/0000-0003-1952-1554); Jun-Feng Li [0000-0002-5638-706X](https://orcid.org/0000-0002-5638-706X).

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REFERENCES

- 1 **Senzolo M**, Garcia-Tsao G, García-Pagán JC. Current knowledge and management of portal vein thrombosis in cirrhosis. *J Hepatol* 2021; **75**: 442-453 [PMID: [33930474](https://pubmed.ncbi.nlm.nih.gov/33930474/) DOI: [10.1016/j.jhep.2021.04.029](https://doi.org/10.1016/j.jhep.2021.04.029)]
- 2 **Intagliata NM**, Caldwell SH, Tripodi A. Diagnosis, Development, and Treatment of Portal Vein Thrombosis in Patients With and Without Cirrhosis. *Gastroenterology* 2019; **156**: 1582-1599.e1 [PMID: [30771355](https://pubmed.ncbi.nlm.nih.gov/30771355/) DOI: [10.1053/j.gastro.2019.01.265](https://doi.org/10.1053/j.gastro.2019.01.265)]
- 3 **Ushitora Y**, Tashiro H, Takahashi S, Amano H, Oshita A, Kobayashi T, Chayama K, Ohdan H. Splenectomy in chronic hepatic disorders: portal vein thrombosis and improvement of liver function. *Dig Surg* 2011; **28**: 9-14 [PMID: [21293126](https://pubmed.ncbi.nlm.nih.gov/21293126/) DOI: [10.1159/000321886](https://doi.org/10.1159/000321886)]
- 4 **Yang L**, Zhang Z, Zheng J, Kong J, Yang X, Wang W. Long-term outcomes of oesophagogastric devascularization and splenectomy in patients with portal hypertension and liver cirrhosis. *ANZ J Surg* 2020; **90**: 2269-2273 [PMID: [32407001](https://pubmed.ncbi.nlm.nih.gov/32407001/) DOI: [10.1111/ans.15994](https://doi.org/10.1111/ans.15994)]
- 5 **Zhao Y**, Zhou R, Guo Y, Chen X, Zhang A, Wang J, Ji F, Qin B, Geng J, Kong G, Li Z. Improvement of gut microbiome and intestinal permeability following splenectomy plus pericardial devascularization in hepatitis B virus-related cirrhotic portal hypertension. *Front Immunol* 2022; **13**: 941830 [PMID: [36159870](https://pubmed.ncbi.nlm.nih.gov/36159870/) DOI: [10.3389/fimmu.2022.941830](https://doi.org/10.3389/fimmu.2022.941830)]
- 6 **Liang QS**, Xie JG, Yu C, Feng Z, Ma J, Zhang Y, Wang D, Lu J, Zhuang R, Yin J. Splenectomy improves liver fibrosis via tumonthr necrosis factor superfamily 14 (LIGHT) through the JNK/TGF- β 1 signaling pathway. *Exp Monthl Med* 2021; **53**: 393-406 [PMID: [33654222](https://pubmed.ncbi.nlm.nih.gov/33654222/) DOI: [10.1038/s12276-021-00574-2](https://doi.org/10.1038/s12276-021-00574-2)]
- 7 **Bai DS**, Zhou BH, Qian JJ, Zhang C, Jin SJ, Jiang GQ. Effects of laparoscopic splenectomy and azygoportal disconnection on liver synthesis function and cirrhosis: a 2-year prospective study. *Surg Endosc* 2020; **34**: 5074-5082 [PMID: [31820157](https://pubmed.ncbi.nlm.nih.gov/31820157/) DOI: [10.1007/s00464-019-07307-7](https://doi.org/10.1007/s00464-019-07307-7)]
- 8 **Gao TM**, Zhou J, Xiang XX, Jin SJ, Qian JJ, Zhang C, Zhou BH, Tang H, Bai DS, Jiang GQ. Splenectomy and azygoportal disconnection decreases the risk of hepatocellular carcinoma for cirrhosis patients with portal hypertension bleeding: a 10-year retrospective follow-up study based on the inverse probability of treatment weighting method. *J Gastroenterol* 2023; **58**: 503-512 [PMID: [36943530](https://pubmed.ncbi.nlm.nih.gov/36943530/) DOI: [10.1007/s00535-023-01982-z](https://doi.org/10.1007/s00535-023-01982-z)]
- 9 **Qi X**, Han G, Ye C, Zhang Y, Dai J, Peng Y, Deng H, Li J, Hou F, Ning Z, Zhao J, Zhang X, Wang R, Guo X. Splenectomy Causes 10-Fold Increased Risk of Portal Venous System Thrombosis in Liver Cirrhosis Patients. *Med Sci Monthsnit* 2016; **22**: 2528-2550 [PMID: [27432511](https://pubmed.ncbi.nlm.nih.gov/27432511/) DOI: [10.12659/MSM.898866](https://doi.org/10.12659/MSM.898866)]
- 10 **Noronha Ferreira C**, Marinho RT, Cortez-Pinto H, Ferreira P, Dias MS, Vasconcelos M, Alexandrino P, Serejo F, Pedro AJ, Gonçalves A, Palma S, Leite I, Reis D, Damião F, Valente A, Xavier Brito L, Baldaia C, Fatela N, Ramalho F, Velosa J. Incidence, predictive factors and clinical significance of development of portal vein thrombosis in cirrhosis: A prospective study. *Liver Int* 2019; **39**: 1459-1467 [PMID: [31021512](https://pubmed.ncbi.nlm.nih.gov/31021512/) DOI: [10.1111/liv.14121](https://doi.org/10.1111/liv.14121)]
- 11 **Hirashita T**, Ohta M, Kai S, Masuda T, Eguchi H, Iwashita Y, Ogawa T, Kitano S. Implications of portal vein thrombosis after splenectomy for patients with idiopathic portal hypertension. *Surg Today* 2011; **41**: 1475-1480 [PMID: [21969148](https://pubmed.ncbi.nlm.nih.gov/21969148/) DOI: [10.1007/s00595-010-4523-6](https://doi.org/10.1007/s00595-010-4523-6)]
- 12 **Agbim U**, Jiang Y, Kedia SK, Singal AK, Ahmed A, Bhamidimarri KR, Bernstein DE, Harrison SA, Younossi ZM, Satapathy SK. Impact of Nonmalignant Portal Vein Thrombosis in Transplant Recipients With Nonalcoholic Steatohepatitis. *Liver Transpl* 2019; **25**: 68-78 [PMID: [30091296](https://pubmed.ncbi.nlm.nih.gov/30091296/) DOI: [10.1002/lt.25322](https://doi.org/10.1002/lt.25322)]
- 13 **Intagliata NM**, Saad WE, Caldwell SH. Effects of restoring portal flow with anticoagulation and partial splenorenal shunt embolization. *Hepatology* 2015; **61**: 1088-1090 [PMID: [24867875](https://pubmed.ncbi.nlm.nih.gov/24867875/) DOI: [10.1002/hep.27241](https://doi.org/10.1002/hep.27241)]
- 14 **Berry K**, Taylor J, Liou IW, Ioannou GN. Portal vein thrombosis is not associated with increased monthsrtality amonthsng patients with cirrhosis. *Clin Gastroenterol Hepatol* 2015; **13**: 585-593 [PMID: [25459555](https://pubmed.ncbi.nlm.nih.gov/25459555/) DOI: [10.1016/j.cgh.2014.10.010](https://doi.org/10.1016/j.cgh.2014.10.010)]
- 15 **Kuroki T**, Kitasato A, Tokunaga T, Takeshita H, Taniguchi K, Maeda S, Fujioka H. Predictors of portal and splenic vein thrombosis after laparoscopic splenectomy: a retrospective analysis of a single-center experience. *Surg Today* 2018; **48**: 804-809 [PMID: [29569060](https://pubmed.ncbi.nlm.nih.gov/29569060/) DOI: [10.1007/s00595-018-1655-6](https://doi.org/10.1007/s00595-018-1655-6)]
- 16 **Jiang GQ**, Bai DS, Chen P, Xia BL, Qian JJ, Jin SJ. Predictors of portal vein system thrombosis after laparoscopic splenectomy and azygoportal disconnection: A Retrospective Cohort Study of 75 Consecutive Patients with 3-monthsnths follow-up. *Int J Surg* 2016; **30**: 143-149 [PMID: [27154616](https://pubmed.ncbi.nlm.nih.gov/27154616/) DOI: [10.1016/j.ijssu.2016.04.047](https://doi.org/10.1016/j.ijssu.2016.04.047)]
- 17 **Li MX**, Zhang XF, Liu ZW, Lv Y. Risk factors and clinical characteristics of portal vein thrombosis after splenectomy in patients with liver cirrhosis. *Hepatobiliary Pancreat Dis Int* 2013; **12**: 512-519 [PMID: [24103282](https://pubmed.ncbi.nlm.nih.gov/24103282/) DOI: [10.1016/S1499-3872\(13\)60081-8](https://doi.org/10.1016/S1499-3872(13)60081-8)]
- 18 **Huang L**, Yu Q, Wang J. Association Between Changes in Splanchnic Hemonthsdynamics and Risk Factors of Portal Venous System Thrombosis After Splenectomy with Periesophagogastric Devascularization. *Med Sci Monthsnit* 2018; **24**: 4355-4362 [PMID: [29937539](https://pubmed.ncbi.nlm.nih.gov/29937539/) DOI: [10.12659/MSM.909403](https://doi.org/10.12659/MSM.909403)]
- 19 **Obstein KL**, Campbell MS, Reddy KR, Yang YX. Association between monthsdel for end-stage liver disease and spontaneous bacterial peritonitis. *Am J Gastroenterol* 2007; **102**: 2732-2736 [PMID: [17714556](https://pubmed.ncbi.nlm.nih.gov/17714556/) DOI: [10.1111/j.1572-0241.2007.01485.x](https://doi.org/10.1111/j.1572-0241.2007.01485.x)]
- 20 **Bertot LC**, Gomez EV, Almeida LA, Soler EA, Perez LB. Monthsdel for End-Stage Liver Disease and liver cirrhosis-related complications.

- Hepatol Int* 2013; **7**: 347-355 [PMID: 26201769 DOI: 10.1007/s12072-012-9403-2]
- 21 **Kamath PS**, Kim WR; Advanced Liver Disease Study Group. The monthsdel for end-stage liver disease (MELD). *Hepatology* 2007; **45**: 797-805 [PMID: 17326206 DOI: 10.1002/hep.21563]
 - 22 **Carnevale R**, Raparelli V, Nocella C, Bartimonthscia S, Novo M, Severino A, De Falco E, Cammisotto V, Pasquale C, Crescioli C, Scavalli AS, Riggio O, Basili S, Violi F. Gut-derived endotoxin stimulates factor VIII secretion from endothelial cells. Implications for hypercoagulability in cirrhosis. *J Hepatol* 2017; **67**: 950-956 [PMID: 28716745 DOI: 10.1016/j.jhep.2017.07.002]
 - 23 **Zocco MA**, Di Stasio E, De Cristofaro R, Novi M, Ainora ME, Ponziani F, Riccardi L, Lancellotti S, Santoliquido A, Flore R, Pompili M, Rapaccini GL, Tondi P, Gasbarrini GB, Landolfi R, Gasbarrini A. Thrombotic risk factors in patients with liver cirrhosis: correlation with MELD scoring system and portal vein thrombosis development. *J Hepatol* 2009; **51**: 682-689 [PMID: 19464747 DOI: 10.1016/j.jhep.2009.03.013]
 - 24 **Kinjo N**, Kawanaka H, Akahoshi T, Tomikawa M, Yamashita N, Konishi K, Tanoue K, Shirabe K, Hashizume M, Maehara Y. Risk factors for portal venous thrombosis after splenectomy in patients with cirrhosis and portal hypertension. *Br J Surg* 2010; **97**: 910-916 [PMID: 20474001 DOI: 10.1002/bjs.7002]
 - 25 **Zhang Y**, Wen TF, Yan LN, Yang HJ, Deng XF, Li C, Wang C, Liang GL. Preoperative predictors of portal vein thrombosis after splenectomy with periesophagogastric devascularization. *World J Gastroenterol* 2012; **18**: 1834-1839 [PMID: 22553410 DOI: 10.3748/wjg.v18.i15.1834]
 - 26 **Stamonthsu KM**, Toutouzas KG, Kekis PB, Nakos S, Gafou A, Manouras A, Krespis E, Katsaragakis S, Bramis J. Prospective study of the incidence and risk factors of postsplenectomy thrombosis of the portal, mesenteric, and splenic veins. *Arch Surg* 2006; **141**: 663-669 [PMID: 16847237 DOI: 10.1001/archsurg.141.7.663]
 - 27 **Huang D**, Tao M, Cao L, Wang X, Zheng S, Cao Y. Risk Factors and Anticoagulation Effects of Portal Vein System Thrombosis After Laparoscopic Splenectomy in Patients With or Without Cirrhosis. *Surg Laparosc Endosc Percutan Tech* 2019; **29**: 498-502 [PMID: 31436648 DOI: 10.1097/SLE.0000000000000710]
 - 28 **Kamath PS**, Wiesner RH, Malinchoc M, Kremers W, Therneau TM, Kosberg CL, D'Amico G, Dickson ER, Kim WR. A monthsdel to predict survival in patients with end-stage liver disease. *Hepatology* 2001; **33**: 464-470 [PMID: 11172350 DOI: 10.1053/jhep.2001.22172]
 - 29 **Ferlitsch M**, Reiberger T, Hoke M, Salzl P, Schwengerer B, Ulbrich G, Payer BA, Trauner M, Peck-Radosavljevic M, Ferlitsch A. von Willebrand factor as new noninvasive predictor of portal hypertension, decompensation and monthsrtality in patients with liver cirrhosis. *Hepatology* 2012; **56**: 1439-1447 [PMID: 22532296 DOI: 10.1002/hep.25806]
 - 30 **Limquiacco JL**, Wong GL, Wong VW, Lai PB, Chan HL. Evaluation of monthsdel for end stage liver disease (MELD)-based systems as prognostic index for hepatocellular carcinoma. *J Gastroenterol Hepatol* 2009; **24**: 63-69 [PMID: 19054256 DOI: 10.1111/j.1440-1746.2008.05701.x]
 - 31 **Abdel-Razik A**, Monthsusa N, Elhelaly R, Tawfik A. De-novo portal vein thrombosis in liver cirrhosis: risk factors and correlation with the Monthsdel for End-stage Liver Disease scoring system. *Eur J Gastroenterol Hepatol* 2015; **27**: 585-592 [PMID: 25769098 DOI: 10.1097/MEG.0000000000000325]
 - 32 **Noronha Ferreira C**, Reis D, Cortez-Pinto H, Tato Marinho R, Gonçalves A, Palma S, Leite I, Rodrigues T, Pedro AJ, Alexandrino P, Serejo F, Sobral Dias M, Ferreira P, Vasconcelos M, Damião F, Xavier Brito L, Baldaia C, Fatela N, Ramalho F, Velosa J. Anticoagulation in Cirrhosis and Portal Vein Thrombosis Is Safe and Improves Prognosis in Advanced Cirrhosis. *Dig Dis Sci* 2019; **64**: 2671-2683 [PMID: 30852769 DOI: 10.1007/s10620-019-05572-z]



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