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Association Between Changes in Splanchnic Hemodynamics and Risk Factors of Portal Venous System Thrombosis After Splenectomy with Periesophagogastric Devascularization

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Background: The purpose of this study was to investigate splanchnic hemodynamic changes and determine an optimal cutoff value for risk factors of portal venous system thrombosis (PVST) after splenectomy with periesophagogastric devascularization (SPD) in cirrhotic patients with esophageal and gastric variceal bleeding (EGVB) and portal hypertension (PH).

Material/Methods: Data on patients who underwent SPD were collected retrospectively from January 2013 to December 2017. Color Doppler ultrasound was performed to detect hemodynamic changes of the hepatic artery, splenic artery, splenic vein, and portal vein in included patients (n=60) and healthy volunteers (n=30). Outcomes were compared between preoperative and postoperative biochemical indicators. The cutoff values for hemodynamics were identified using receiver operating characteristic (ROC) curve analysis, and univariate and multivariate analyses of risk factors of PVST were performed.

Results: In our series, hemodynamic indexes of splenic artery, spleen vein, and portal vein in the study group were significantly higher than that of the control group ($P<0.05$). Multivariate analysis revealed that the portal vein flow and the internal diameter of the portal vein were significantly correlated with PVST. The ROC analysis revealed that the cutoff points for portal vein flow and internal diameter of the splenic vein and portal vein were ≥1822.32 ml/min, ≥1.37 cm, and ≥1.56 cm, respectively.

Conclusions: SPD is an effective treatment in cirrhotic patients with concomitant EGVB and PH by increasing hepatic artery flow and decreasing portal vein flow. High portal vein flow and wider diameters of the portal vein and splenic vein are important markers of PVST.

MeSH Keywords: Hemodynamics • Hypertension, Portal • Liver Cirrhosis • Splenectomy • Venous Thrombosis

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Background

The spleen, as the largest lymphoid organ in the body, is anatomically linked to the liver via the portal vein system. In the course of liver cirrhosis, splenomegaly and hypersplenism are relatively specific complications which may contribute to leukopenia, erythropenia, and thrombocytopenia in cirrhotic patients [1,2]. The exact causes of liver cirrhosis associated with splenomegaly and hypersplenism remain complex, but the altered hemodynamics of the portal vein system are obvious [3–5]. Increased portal pressure contributes to formation of portosystemic venous collaterals in order to decompress the portal vein system, which results in esophagogastric varices [6,7]. Additionally, the splanchnic and systemic hemodynamics, including portal vein, splenic vessel, and hepatic artery system, varies in cirrhotic patients with concomitant portal hypertension (PH) [8–11]. Although the data is not comprehensive, the splanchnic hemodynamic disorder of cirrhotic patients with concomitant PH in a few studies reveals the significance of increased splenic artery flow and decreased hepatic artery flow [8–14].

Advanced liver disease, such as cirrhosis, is often accompanied by hypersplenism and esophagogastric varices, which may contribute to esophageal and gastric variceal bleeding (EGVB) [15,16]. Furthermore, recent studies reported that splenectomy with periesophagogastric devascularization (SPD) is the optimal choice to manage hypersplenism and EGVB, with low incidence of complications and better liver function [17–24]. Additionally, SPD can contribute to decreased portal vein flow and increased hepatic artery flow via splenic arteriovenous disconnection [25–27]. Nevertheless, the splanchnic and systemic hemodynamics in cirrhotic patients with EGVB and PH before and after SPD still remains unclear in some aspects due to limited sample numbers and data deficiency [25–27].

Portal venous system thrombosis (PVST) is a common and potentially life-threatening complication after surgical intervention for PH due to cirrhosis; however, research data on risk factors of PVST after SPD are few in number and limited in scope [28–31]. Furthermore, periesophagogastric devascularization without splenectomy was reported to have a lower incidence of PVST compared with SPD, suggesting that hemodynamic changes in the splenic vein and portal vein may result in PVST [32,33]. Furthermore, the relationship between the internal diameter of splenic artery and proper hepatic artery was reported to be a predictor of morbidity after splenectomy [34]. Therefore, there is a need to determine the optimal cutoff values of hemodynamic changes, which could be an important marker of PVST.

Therefore, the present retrospective study was performed to more comprehensively define the best hemodynamic indicators, including blood flow, blood flow velocity, internal diameter of blood vessel, and RI to use in investigating the hemodynamic changes before and after SPD in cirrhotic patients with EGVB and PH. We also assessed risk factors of PVST and relationships between PVST and hemodynamic indicators.

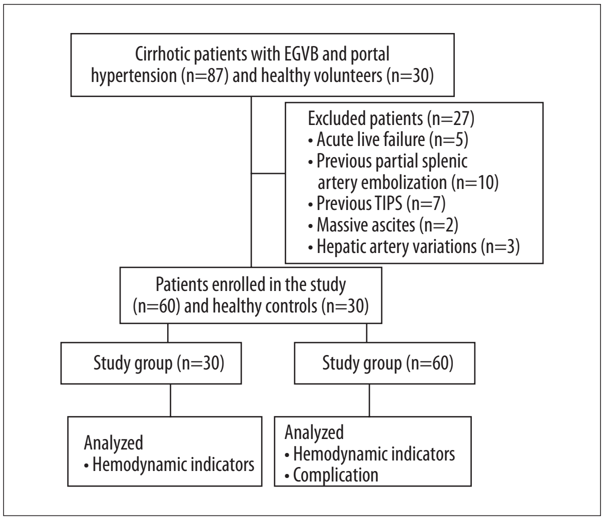

Figure 1. Flow diagram of the study population.
EGVB – esophageal and gastric variceal bleeding;
TIPS – transjugular Intrahepatic portosystemic shunt.

Material and Methods

At Anhui Chinese Medical Research Institute of Surgery, the records of 87 consecutive cirrhotic patients with EGVB and PH who met the inclusion criteria from January 2013 to December 2017 were reviewed retrospectively (Figure 1).

The inclusion criteria included clinically diagnosed PH related to hepatitis B cirrhosis in patients with a history of EGVB and hypersplenism. Hypersplenism was defined as a leukocyte count <3500/μl and a platelet count $<7.5\times10^4$/μl [35]. All the patients had endoscopically confirmed esophageal and gastric varices and underwent SPD. All surgical procedures were non-emergent.

Of the 87 patients who meet the inclusion criteria, 27 were excluded: 5 had acute live failure, 10 had previous partial splenic artery embolization, 7 had previous transjugular intrahepatic portosystemic stent shunting, 2 had massive ascites, and 3 had hepatic artery variations. Thus, 27 patients were excluded and 60 were finally included in the study. Records of all included patients with PH and EGVB were analyzed carefully. Data included age, gender, details of the initial operation, perioperative diagnostic strategy, Child-Pugh grade of liver function, and surgical outcomes. Patients ranked as Child C should receive liver protection treatment and regulation of

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blood coagulation and nutrition until they are ranked above Child B. Human albumin and Vit-K1 are commonly used to improve patient condition.

Thirty healthy volunteers who participated in a routine physical examination were also enrolled. All included patients were divided into 2 groups: those who underwent SPD for PH and EGVB (Study group, n=60), and those who were normal healthy people attending a routine physical examination during the same period (Control group, n=30). Before surgical procedures, all included subjects or their relatives provided informed consent and the investigation was carried out in accordance with the principles of the Helsinki Declaration (as revised in Fortaleza, Brazil, October 2013). The Ethics Committee of Anhui Provincial Traditional Chinese Hospital approved the study protocol.

Operation

The details of our standard surgical procedure of SPD have been commonly described. Open operation was performed by placing the patient in a supine position and using a paramedian straight incision in the left upper abdomen. The splenic artery was firstly ligated, and then splenectomy was performed. After routine splenectomy, periesophagogastric devascularization was performed. The right gastric vein and small branches of the gastric coronary veins were disconnected. Then, the esophageal branch was disconnected and suture-ligated up to 7–9 cm of the esophageal inferior segment. The gastric posterior veins and short gastric veins were ligated by suturing, and then the left subphrenic vein was ligated as well. In addition, the arteries accompanied by the veins including the left gastric artery, left gastroepiploic artery, gastric posterior artery, and left subphrenic artery were disconnected. One latex drainage tube was inserted beside the splenic fossa, and intermittent suction of the drainage fluid was performed in principle. The latex drainage tube was pulled out when the drain left with slight output. Five staff surgeons performed all of the operations. Patients were thoroughly observed for possible complications, including bleeding, abdominal infection, abdominal collection, pulmonary infection, PVST, hepatic failure, and peritonitis, within 2 weeks and were assessed for need for a secondary intervention.

Color Doppler ultrasound detection

Color Doppler ultrasound detection was performed by an experienced examiner with a color Doppler ultrasound system (ACUSON S2000, Siemens, USA) and a broadband convex array probe (3 to 5 Mhz). The ultrasound examination encompassed internal diameter and blood flow of the proper hepatic artery, splenic vessels, and portal vein. The peak systolic velocity (PSV) of proper hepatic artery and splenic artery, the maximum blood flow velocity (Vmax) of portal vein and splenic vein and RI were also measured. For each measurement, at least 3 reproducible patterns were created to ensure the measurement accuracy. Generally, routine ultrasound was performed in all patients on admission and on the 7^{th} day after the operation.

Laboratory tests

Preoperative details were collected on admission. Postoperative details, including erythrocyte, leucocyte, thrombocyte, hematoglobin, transaminase, bilirubin, and albumin, were collected on postoperative day 7 and postoperative day 14. Erythrocyte, leucocyte, thrombocyte, and hematoglobin levels were detected using an automatic 5-classification blood cell analyzer (Sysmex XT-2000i). Transaminase, bilirubin, and albumin were detected using a fully automatic biochemical analyzer (HITACHI 7600, Japan).

Data analysis

Receiver operating characteristic (ROC) curve analysis was performed to determine the optimal cutoff values of each hemodynamic indicator. Associations between the hemodynamic indicators and the incidence of PVST were assessed using univariate analyses, and those variables showing statistical significance ($P<0.05$) were evaluated by multivariate logistic analyses to discover the main independent risk factors of PVST. Statistical analysis was done using the Statistical Package for the Social Sciences (SPSS, Version 13.0, Chicago, IL, USA). Continuous variables are reported as means ± standard deviation (SD) or ranges. Comparison between groups was carried out using the *t* test for measurement data and the χ^2 test with or without Fisher's exact test for categorical variables. Statistical significance was accepted at the 5% level by a two-tailed test.

Results

A total of 60 included cases (40 males, 20 females; mean age 42.7±10.2 years) who underwent SPD and 30 healthy controls (20 males, 10 females; mean age 46.1±8.5 years) were enrolled. Characteristics between the 2 groups regarding age and sex revealed no significant difference. All clinical data are summarized in Table 1.

Outcomes and complications

SPD was performed successfully in 60 cirrhotic patients with EGVB and PH. Mean operative time was 225.5±61.5 min and intraoperative blood loss was 243.5±150.5 ml. The mean postoperative length of hospital stay was 19.7±11.2 days. No patient died after the operation. Overall, complications occurred

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Table 1. Clinical characteristics of included patients, n(%).

Variables	Study group (n=60)	Control group (n=30)
Sex		
Male	40 (66.7)	20 (66.7)
Female	20 (33.3)	10 (33.3)
Age (year, mean ±SD)	42.7±10.2	46.1±8.5
The degree of splenomegaly		NA
Slight	26 (43.3)	
Moderate	25 (41.7)	
Severe	9 (15.0)	
Child-pugh		NA
Child A	25 (41.7)	
Child B	32 (53.3)	
Child C	3 (5.0)	
Operation time (min)	225.5±61.5	NA
Blood loss (ml)	243.5±150.5	NA
Hospital stay (d)	19.7±11.2	NA
Complication	22 (36.7)	NA
Bleeding	0	
Ascites	6 (10.0)	
Encephalopathy	0	
PVST	11 (18.3)	
Intestinal obstruction	2 (3.3)	
Pulmonary infection	0	
Incisional infection	3 (5.0)	

PVST – portal venous system thrombosis.

in 22 cases after surgical intervention. Six patients with ascites received diuretics therapy and 2 patients had intestinal obstruction. Three patients developed incisional infection. No recurrence of EGVB occurred perioperatively. After the operations, 11 patients (18.3%) had PVST during the first 7 days postoperatively, and they received heparin therapy. All patients fully recovered after medical treatment. Outcomes and complications are listed in Table 1.

Clinical laboratory tests

Preoperative and postoperative clinical laboratory test of erythrocytes, leucocytes, and thrombocytes are contrasted in Table 2. In comparison to preoperative results, results 1 week after the operation also revealed significant differences with regard to leucocytes, thrombocytes, and transaminase (P<0.05). Results at 2 weeks after the operation revealed significant differences with respect to leucocytes, thrombocytes, bilirubin, and transaminase compared to preoperative statistics (P<0.05).

Hemodynamic indexes

In our study, there was no significant differences regarding the internal diameter and RI of the proper hepatic artery between the 2 groups (P>0.05). The PSV and blood flow of proper hepatic artery were significantly lower in the study group (P<0.05). The internal diameter, blood flow, and PSV of the splenic artery were much higher in the study group (P<0.05). The internal diameter and blood flow of the portal vein were much higher in the study group, but the Vmax of the portal vein was lower in the study group. There were significant differences between the 2 groups in these parameters (P<0.05).

Compared with preoperative values, the PSV and blood flow of the proper hepatic artery after the operation were significantly increased (P<0.05), and the Vmax of the portal vein

Table 2. Comparisons of preoperative and postoperative clinical laboratory test results in cirrhotic patients with EGVB and portal hypertension who received SPD (n=60).

Variables	Preoperative indicators	7 days after SPD	14 days after SPD	P-value (Pre- *vs.* 14 days)
RBC count ($\times 10^{12}$/L)	4.01±0.47	4.22±0.57	4.17±0.59	0.24
WBC count ($\times 10^{9}$/L)	2.77±1.36	9.08±3.35$^{\#}$	7.92±3.75	0
Hemoglobin (g/L)	112.16±13.40	117.59±14.47	115.79±13.39	0.28
Platelet count ($\times 10^{9}$/L)	47.44±16.03	389.65±156.27$^{\#}$	491.29±194.89	0
ALT (U/L)	43.37±16.59	33.78±15.99$^{\#}$	32.94±8.57	0.02
Total bilirubin (µmol/L)	24.36±13.85	21.80±18.43	14.97±10.56	0.01
Albumin (g/L)	36.95±4.67	35.84±4.01	34.43±4.00	0.17

$^{\#}$ Compared with preoperative indicators, P<0.05. EGVB – esophageal and gastric variceal bleeding; SPD – splenectomy with periesophagogastric devascularization; ALT – alanine amino transferase; WBC – white blood cell; RBC – red blood cell.

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Table 3. Hemodynamic indexes in included patients.

Variables	Control group	Preoperative study group	Postoperative study group	P-value (Study group *vs.* Control group)
Proper hepatic artery				
Internal diameter (cm)	0.35±0.07	0.33±0.02	0.35±0.02	0.195
PSV (cm/s)	53.05±7.02	36.11±3.52	60.71±11.85#	0
Blood flow (ml/min)	297.04±48.33	175.44±18.27	388.77±79.59#	0
RI	0.67±0.08	0.77±0.08	0.68±0.07	
Splenic artery			NA	
Internal diameter (cm)	0.35±0.06	0.63±0.07		0
PSV (cm/s)	38.43±2.01	77.67±3.32		0
Blood flow (ml/min)	212.35±127.34	809.03±117.84		0
RI	0.54±0.08	0.63±0.07		
Splenic vein			NA	
Internal diameter (cm)	0.62±0.02	1.24±0.16		0
Vmax (cm/s)	20.13±1.28	26.15±1.38		0
Blood flow (ml/min)	270.25±85.33	1085.54±52.23		0
Portal vein				
Internal diameter (cm)	1.02±0.14	1.44±0.21	1.39±0.19	0
Vmax (cm/s)	22.33±2.21	11.42±0.79	10.11±1.12#	0
Blood flow (ml/min)	1066.53±98.26	1741.32±178.64	1516.14±489.98	0

Compared with preoperative study group, P<0.05. PSV – the peak systolic velocity; Vmax – the maximum blood flow velocity.

after the operation was significantly decreased (P<0.05). The comparison between the preoperative and postoperative hemodynamic indexes of portal vein and proper hepatic artery in included patients are shown in Table 3. We also demonstrated that the proper hepatic artery RI decreased in compensation after the operation.

Risk factors of PVST

A total of 11 (18.3%) patients were diagnosed with PVST after surgical intervention. The internal diameter of the splenic vein >1.37 cm and the portal vein >1.56 cm was determined as the optimal cutoff values by ROC curve analysis. The sensitivity and 1-specificity of these indicators were 63.6%, 18.4%, 72.7%, and 24.5%, respectively. The optimal cutoff value of abnormal portal vein flow was >1822.32 ml/min with 72.7% sensitivity and 28.6% 1-specificity. When the hemodynamic indexes were assessed by univariate analysis to determine the relationship with PVST, there was statistical significance detected for the internal diameter of splenic vessels and portal vein, the Vmax of portal vein, and the blood flow of the portal vein and splenic artery (P<0.05). The internal diameter of the splenic vein, as well as the portal vein and portal vein flow, were determined as independent risk factors of PVST by multivariate logistic analysis. The univariate and multivariate logistic analysis of risk factors of PVST in included patients are shown in Table 4.

Discussion

Cirrhosis caused by chronic hepatitis B frequently combines with PH, which results from both an increase in resistance to portal flow and an increase in portal venous inflow [9,36]. Since the portal vein flow and the hepatic artery flow comprise the hepatic blood supply together, there is a significant correlation among hepatic artery, splenic artery, and portal vein because the hepatic artery and splenic artery all originate from the celiac trunk. Hemodynamic changes caused by PH are usually sustained for a long time, and progressively aggravated hemodynamic changes may contribute to the increasing incidence of complications, especially for EGVB and PVST, and significantly decrease survival [37]. PVST, which may be followed by the amplified risk of upper gastrointestinal bleeding and bowel infarction, may further enhance portal venous pressure and deteriorate liver function, and even lead to death [38]. Therefore, we provide a comprehensive hemodynamic change in cirrhotic patients with concomitant EGVB and PH to investigate the

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Table 4. Univariate and multivariate analysis of the risk factors for PVST after SPD.

Variables	Study group (n=60)	PVST (n=11)	P-value	Multivariate logistic regression				
				B	S.E	Wals	P	Exp(B)
Internal diameter of proper hepatic artery (cm)			0.14					
<0.355	48	7						
≥0.355	12	4						
PSV of proper hepatic artery (cm/s)			0.25					
<39.865	48	8						
≥39.865	12	3						
Proper hepatic artery flow (ml/min)			0.25					
<194.045	48	8						
≥194.045	12	3						
Internal diameter of splenic artery (cm)			0.03	1.73	1.46	1.4	0.24	5.635
<0.675	42	4						
≥0.675	18	7						
PSV of splenic artery (cm/s)			0.09					
<78.79	36	4						
≥78.79	24	7						
Splenic artery flow (ml/min)			0.02	2.69	1.6	2.82	0.09	14.746
<902.865	44	4						
≥902.865	16	7						
Internal diameter of splenic vein (cm)			0.02	2.63	1.27	4.3	0.04	13.925
<1.365	44	4						
≥1.365	16	7						
Vmax of splenic vein (cm/s)			0.06					
<27.245	44	5						
≥27.245	16	6						
Splenic vein flow (ml/min)			0.07					
<1109.215	38	4						
≥1109.215	22	7						
Internal diameter of portal vein (cm)			0.01	2.99	1.47	4.13	0.04	19.846
<1.555	40	3						
≥1.555	20	8						
Vmax of portal vein (cm/s)			0.03	−1.6	2	0.64	0.42	0.202
<11.955	42	4						
≥11.955	18	7						
Portal vein flow (ml/min)			0.04	2.69	1.25	4.6	0.03	14.662
<1822.32	36	3						
≥1822.32	24	8						

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associations between risk factors of PVST after SPD and hemodynamic indicators to better prevent the occurrence of PVST.

Severe cirrhosis and PH can give rise to hepatic artery hypoperfusion and a shift of hepatic blood flow into the splenic arteries, which consequently result in hypersplenism, liver hypoxic injury, elevated liver enzyme levels, and splanchnic hemodynamic disorders [4,39,40]. Recurrent upper gastrointestinal bleeding is a severe complication due to esophageal and gastric varices in cirrhotic patients, which can increase the mortality rate as well. The incidence of bleeding varies from 10% to 30% of patients with liver cirrhosis within 1 year, depending on the degree of liver insufficiency [41–43]. As an optimal treatment for cirrhotic patients with concomitant EGVB and hypersplenism, SPD has been performed clinically for many years. Recent studies also reported that splenectomy procedures in cirrhotic patients could improve liver function through converting splanchnic hemodynamics [25–27]. In our study, we also demonstrated that SPD can significantly reduce portal venous flow and velocities because of the disconnected splenic vein collateral circulations, and increase the blood flow and velocity of the hepatic artery due to maintenance hepatic arterial buffer response or the disconnected splenic artery shunting from the celiac trunk [44]. Our results revealed that postoperative laboratory indicators, including leucocytes and thrombocytes, were significantly increased, which indicated that these parameters could recover to normal levels ($P<0.05$). Our study also demonstrated a decreasing tendency towards portal vein flow and increased proper hepatic flow, as well as velocities, after the operation ($P<0.05$). Furthermore, SPD procedures break the splenic-to-portal circulation and have been shown to improve hepatic arterial flow. In our study, ultrasound provided diagnosis and monitoring of hemodynamic changes in cirrhotic patients with concomitant EGVB and PH. We found that SPD procedures performed in cirrhotic patients with EGVB and PH not only recover hepatic artery perfusion but also cure hypersplenism and produce satisfactory outcomes, suggesting the necessity of surgical treatment in cirrhotic patients with EGVB.

A wider preoperative internal diameter of the portal vein and splenic vein, and high preoperative portal vein flow were shown to be independent risk factors for PVST after SPD in our study ($P<0.05$). The independent risk factors of PVST can also be illustrated in light of the Virchow triad [45]. First, the wider internal diameter of the portal vein and high portal vein flow usually indicate higher portal pressure and reduced blood flow velocity towards the liver, which favors thrombosis formation. Second, a few studies suggested that blood turbulence of the splenic vein after splenectomy resulted in increased coagulation ability, leading to the development of PVST, because the diameter of the splenic vein was correlated with the change ratio of portal venous flow, which favors PVST formation [46–49]. In our study, the internal diameter of the splenic vein >1.37 cm and portal vein >1.56 cm were determined to be the optimal cutoff values by ROC curve analysis, and the portal vein flow was identified to be >1822.32 ml/min. According to the indicators above, 26.7%, 33.3%, and 40% of cirrhotic patients with concomitant EGVB and PH had a higher risk of postoperative PVST. The incidence of PVST, which is measured in patients with wider internal diameter of the splenic vein, as well as portal vein and increased portal vein flow, remains high, indicating that these indicators can be used as markers of PVST prediction in cirrhotic patients with PH and EGVB. Early heparin therapy should be recommended in patients who have these factors after the operation [50].

There are quite a few limitations of the present study. First, this study was a retrospective and single-center investigation, meaning it had a limited number of patients and contains accidental errors and biases. Second, PVST was diagnosed only by ultrasonography but not by CT angiography, and early-stage PVST may not be detected in time. Third, a few discharged patients refused follow up or were reexamined in local hospitals, so we lacked related data in the following period. These limitations will be taken into consideration in our future prospective studies. A randomized controlled trial is needed, and more centers could join the study to provide more evidence for further research.

Conclusions

In conclusion, SPD is an effective treatment for decreasing portal venous flow and increasing proper hepatic artery flow in cirrhotic patients with concomitant EGVB and PH. Wider preoperative internal diameter of the portal vein and splenic vein, and high preoperative portal vein flow, were independently associated with the formation of PVST after SPD in our study.

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