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

Long Huang
Qingsheng Yu
Jiajia Wang

Corresponding Author: Qing-sheng Yu, e-mail: zapttest163.com
Source of support: Departmental sources

Background: The purpose of this study was to investigate splanchnic hemodynamic changes and determine an optimal cut-off 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

Full-text PDF: https://www.medicmoni.com/abstract/index/idArt/909403

2747 4 1 50
**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, erythrocytopenia, 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 de-compress the portal vein system, which results in esophageal-gastric 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 esophageal-gastric varices, which may contribute to esophageal and gastric variceal bleeding (EGVB) [15,16]. Furthermore, recent studies reported that splenectomy with periesophageal 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, periesophageal 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.

**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 <7.5×10^9/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...
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 (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, periesophagogastriastic 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 7th day after the operation.

Laboratory tests

Preoperative details were collected on admission. Postoperative details, including erythrocyte, leucocyte, thrombocyte, hemoglobin, transaminase, bilirubin, and albumin, were collected on postoperative day 7 and postoperative day 14. Erythrocyte, leucocyte, thrombocyte, and hemoglobin 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 χ² 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
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 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 |                   |                      |
| Slight                     | 26 (43.3)         |                      |
| Moderate                   | 25 (41.7)         |                      |
| Severe                     | 9 (15.0)          |                      |
| Child-pugh                 |                   |                      |
| 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)          | 17.7±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.

**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 (×10¹²/L)        | 4.01±0.47               | 4.22±0.57        | 4.17±0.59         | 0.24                      |
| WBC count (×10⁹/L)         | 2.77±1.36               | 9.08±3.39        | 7.92±3.75         | 0                         |
| Hemoglobin (g/L)           | 112.16±13.40            | 117.59±14.47     | 115.79±13.39      | 0.28                      |
| Platelet count (×10⁹/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       | 34.3±4.00         | 0.17                      |

* Compared with preoperative indicators, P<0.05. EGVB – esophageal and gastric variceal bleeding; SPD – splenectomy with periesophagogastrectomy devascularization; ALT – alanine amino transferase; WBC – white blood cell; RBC – red blood cell.
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

---

**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                 |                                        |
| **Spleenic artery**        |                        |                          |                           |                                        |
| Internal diameter (cm)     | 0.35±0.06              |                          |                           | 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              |                          |                           |                                        |
| **Spleenic vein**          |                        |                          |                           |                                        |
| Internal diameter (cm)     | 0.62±0.02              | 1.24±0.16                |                           | 0                                      |
| Vmax (cm/s)                | 20.13±1.28             | 26.15±1.13               |                           | 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.
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 |
|------------------------------------------------|--------------------|-------------|---------|----------------------------------|
| 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           |         |                                  |
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 splanchic 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 splanchic 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.

**References:**

1. Gangireddy VG, Kanneganti PC, Sridhar S et al: Management of thrombocytopenia in advanced liver disease. Can J Gastroenterol Hepatol, 2014; 28: 558–64
2. Mitchell O, Feldman DM, Diakow M, Sigal SH: The pathophysiology of thrombocytopenia in chronic liver disease. Hepat Med, 2016; 8: 39–50
3. Grossmann RJ, Abraldes JG: Portal hypertension: from bedside to bench. J Clin Gastroenterol, 2005; 39: S125–30
4. La Villa G, Gentilini P: Hemodynamic alterations in liver cirrhosis. Mol Aspects Med, 2008; 29: 112–11
5. Maller S, Bendtsen F, Henriksen JH: Splanchnic and systemic hemodynamic derangement in decompensated cirrhosis. Can J Gastroenterol, 2001; 15: 94–106
6. Yin XY, Lu MD, Huang JF et al: Color Doppler velocity profile assessment of portal hemodynamics in cirrhotic patients with portal hypertension: Correlation with esophageal variceal bleeding. J Clin Ultrasound, 2001; 29: 7–13

7. Chikamori F, Inoue A, Okamoto H et al: Relationships between types of esophagogastric varices and systemic hemodynamics in patients with liver cirrhosis. Hepatogastroenterology, 2011; 58: 909–15

8. Xu KY, Tao CL, Wang HJ et al: The effect of splenic arterial blood flow (SBF) on severity of hypersplenism and analysis of factors associated with SBF. Hepatogastroenterology, 2010; 57: 1360–62

9. Sikuier E, Grossmann RI: Interaction of flow and resistance in maintenance of portal hypertension in a rat model. Am J Physiol, 1986; 250: G205–12

10. Aoki H, Hasumi A, Hashizume M et al: Hemodynamic analysis of findings in patients with portal hypertension: Multicenter analysis in Japan. Japan Portal Hypertension Study Group. Hepatogastroenterology, 1995; 42: 1030–38

11. Yamauchi H, Suda Y, Yamamoto K, Sato T: Angiographic studies of splenic and hepatic arteries in portal hypertension. Tohoku J Exp Med, 1970; 101: 363–74

12. Toni R, Bolondi L, Gaiani S et al: Accessory ultrasonographic findings in chronic liver disease: Diagnosis of splenic arterial collaterals, fasting gall bladder volume, and course of left portal vein. J Clin Ultrasound, 1985; 13: 611–18

13. Liu Q, Ma K, Song Y et al: Two-year follow-up of radio frequency ablation for patients with cirrhotic hypersplenism: Does increased hepatic arterial flow induce liver regeneration? Surgery, 2008; 143: 509–18

14. Liu Q, Ma K, He Z et al: Radiofrequency ablation for hypersplenism in patients with liver cirrhosis: A pilot study. J Gastrointest Surg, 2005; 9: 648–57

15. Lee E, Kim YJ, Goo DE et al: Comparison of hepatic venous pressure gradient and endoscopic grading of esophageal varices. World J Gastroenterol, 2016; 22: 1212–19

16. Kumar S, Aravani SK, Kamath PS: Epidemiology, diagnosis and early patient management of esophageal hemorrhage. Gastroenterol Clin North Am, 2014; 43: 765–82

17. Yu H, Guo S, Wang L et al: Laparoscopic splenectomy and esophagogastroduodenal devascularization for liver cirrhosis and portal hypertension is a safe, effective, and minimally invasive operation. J Laparoendosc Adv Surg Tech A, 2014; 24: 612–16

18. Wang WI, Tang Y, Zhang Y, Chen Q: Prevention and treatment of hemorrhage during laparoscopic splenectomy and devascularization for portal hypertension. J Huazhong Univ Sci Technolog Med Sci, 2015; 35: 99–104

19. Cheng Z, Li JW, Chen J et al: Therapeutic effects of laparoscopic splenectomy and esophagogastroduodenal devascularization on liver cirrhosis and portal hypertension in 204 cases. J Laparoendosc Adv Surg Tech A, 2014; 24: 612–16

20. Hong D, Cheng J, Wang Z et al: Comparison of two laparoscopic splenectomy plus pericardial devascularization techniques for management of portal hypertension and hypersplenism. Surg Endosc, 2015; 29: 3819–26

21. Ushitora Y, Tashiro H, Takahashi S et al: Splenectomy in chronic hepatic disorders: Portal vein thrombosis and improvement of liver function. Dig Surg, 2011; 28: 9–14

22. Kodama T, Takeda H, Takita H et al: Thrombocytopenia exacerbates cholesterol-induced liver fibrosis in mice. Gastroenterology, 2010; 138: 2487–98

23. Kim J, Yi NL, Shin WY et al: Platelet transfusion can be related to liver regeneration after living donor liver transplantation. World J Surg, 2010; 34: 1052–38

24. Akkozi EM, Nijsen NW, de Jong KP et al: Immediate postoperative low platelet count is associated with delayed liver function recovery after partial liver resection. Ann Surg, 2010; 251: 300–6

25. Zhang Y, Wen T, Yan L et al: The changes of hepatic hemodynamics and functional hepatic reserve after splenectomy with periesophagogastric devascularization. Hepatogastroenterology, 2009; 56: 835–39

26. Cao H, Hua R, Wu ZY: Effects of combined splenorenal shunt devascularization and devascularization only on hemodynamics of the portal venous system in patients with portal hypertension. Hepatobiliary Pancreat Dis Int, 2005; 4: 385–88

27. Takenaka H, Nakao K, Miyata M et al: Hemodynamic study after devascularization procedure in patients with esophageal varices. Surgery, 1990; 107: 55–62

28. Zhang X, Wang Y, Yu M et al: Effective prevention for portal vein system thrombosis after splenectomy: A meta-analysis. J Laparoendosc Adv Surg Tech A, 2017; 27: 247–52

29. Qi X, Bai M, Guo X, Fan D: Pharmacologic prophylaxis of portal vein system thrombosis after splenectomy: A meta-analysis. Gastroenterol Res Pract, 2014; 2014: 292689

30. He S, He F: Predictive model of portal vein system thrombosis in cirrhotic portal hypertensive patients after splenectomy. Int J Clin Exp Med, 2015; 8: 4236–42

31. Li MX, Zhang XF, Liu ZW, Ly Y: Risk factors and clinical characteristics of portal vein thrombosis after splenectomy in patients with liver cirrhosis. Hepatobiliary Pancreat Dis Int, 2013; 12: 512–19

32. Ni YB, Gao P, Wang D et al: Esophagogastroduodenal devascularization without splenectomy in portal hypertension: Safe and effective? Hepatobiliary Pancreat Dis Int, 2015; 14: 276–80

33. Bao H, He Q, Dali N et al: Retrospective study to compare selective decongestive devascularization and gastroplenic shunt versus splenectomy with pericardial devascularization for the treatment of patients with esophago-gastric varices due to cirrhotic portal hypertension. Med Sci Monit, 2017; 23: 2788–95

34. Zeng DB, Dai CZ, Lu SC et al: Abnormal splenic artery diameter/hepatic artery diameter ratio in cirrhosis-induced portal hypertension. World J Gastroenterol, 2013; 19: 1292–98

35. Ikegami T, Soejima Y, Taketomi A et al: Hypersplenism after living donor liver transplantation. Hepatogastroenterology, 2009; 56: 778–82

36. Moriyasu F, Nishida O, Ban N et al: Measurement of portal vascular resistance in patients with portal hypertension. Gastroenterology, 1986; 90: 710–17

37. Kim MY, Baik SK, Lee SS: Hemodynamic alterations in cirrhosis and portal hypertension. Korean J Hepatol, 2010; 16: 347–52

38. D’Amico G, De Franchis R, Cooperative Study Group: Upper digestive bleeding in cirrhosis. Post-therapeutic outcome and prognostic indicators. Hepatology, 2003; 38: 599–612

39. Villanueva C, López-Balaguer JM, Aracil C et al: Maintenance of hemodynamic response to treatment for portal hypertension and influence on complications of cirrhosis. J Hepatol, 2004; 40: 757–65

40. Baik SK, Lee MG, Jeong PH et al: Relationship of hemodynamic indices and prognosis in patients with liver cirrhosis. Korean J Intern Med, 2004; 19: 165–70

41. García-Tsao G: Current management of the complications of cirrhosis and portal hypertension: Variceal hemorrhage, ascites, and spontaneous bacterial peritonitis. Gastroenterology, 2001; 120: 726–48

42. Grace ND, Groszmann RJ, Garcia-Tsao G et al: Portal hypertension and variceal bleeding: An AASLD single topic symposium. Hepatology, 1998; 28: 868–80

43. Otol P, Smaya T, Bureau C et al: Preliminary results of a new expanded-polytetrafluoroethylene-covered stentgraft for transjugular intrahepatic portosystemic shunt procedures. Am J Roentgenol, 2002; 178: 141–47

44. Richter S, Mucke I, Menger MD et al: Impact of intrinsic blood flow regulation in cirrhosis: Maintenance hepatic arterial buffer response. Am J Physiol, 2000; 279: G454–62

45. Byers JM 3rd: Rudolf Virchow – father of cellular pathology. Am J Clin Pathol, 1989; 92: 52–8

46. Kinjo N, Kawanaka H, Akahoshi T et al: Risk factors for portal vein thrombosis after splenectomy in patients with cirrhosis and portal hypertension. Br J Surg, 2010; 97: 910–16

47. Winslow ER, Brunt LM, Drebin JA et al: Portal vein thrombosis after splenectomy. Am J Surg, 2002; 184: 631–36

48. Fujita F, Lyass S, Otsuka K et al: Portal vein thrombosis following splenectomy for haemochromatosis: Identification of risk factors. Am J Surg, 2003; 69: 951–56

49. Danno K, Ikeda M, Sekimoto M et al: Diameter of splenic vein is a risk factor for portal vein thrombosis after splenectomy. J Gastroenterol Hepatol, 2010; 25: 457–66

50. Cheng Z1, Yu F, Tian J et al: A comparative study of two anti-coagulation plans on the prevention of PVST after laparoscopic splenectomy and esophagogastroduodenal devascularization. J Thromb Thrombolysis, 2015; 40: 294–301