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BRIEF ARTICLE

Preoperative predictors of portal vein thrombosis after splenectomy with periesophagogastric devascularization

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Abstract

AIM: To evaluate the predictive value of preoperative predictors for portal vein thrombosis (PVT) after splenectomy with periesophagogastric devascularization.

METHODS: In this prospective study, 69 continuous patients with portal hypertension caused by hepatitis B cirrhosis underwent splenectomy with periesophagogastric devascularization in West China Hospital of Sichuan University from January 2007 to August 2010. The portal vein flow velocity and the diameter of portal vein were measured by Doppler sonography. The hepatic congestion index and the ratio of velocity and diameter were calculated before operation. The prothrombin time (PT) and platelet (PLT) levels were measured before and after operation. The patients' spleens were weighed postoperatively.

RESULTS: The diameter of portal vein was negatively correlated with the portal vein flow velocity ($P < 0.05$). Thirty-three cases (47.83%) suffered from postoperative PVT. There was no statistically significant difference in the Child-Pugh score, the spleen weights, the PT, or PLT levels between patients with PVT and without PVT. Receiver operating characteristic curves showed four variables (portal vein flow velocity, the ratio of velocity and diameter, hepatic congestion index and diameter of portal vein) could be used as preoperative predictors of postoperative portal vein thrombosis. The respective values of the area under the curve were 0.865, 0.893, 0.884 and 0.742, and the respective cut-off values (24.45 cm/s, 19.4333/s, 0.1138 cm/s^{-1} and 13.5 mm) were of diagnostically efficient, generating sensitivity values of 87.9%, 93.9%, 87.9% and 81.8%, respectively, specificities of 75%, 77.8%, 86.1% and 63.9%, respectively.

CONCLUSION: The ratio of velocity and diameter was the most accurate preoperative predictor of portal vein thrombosis after splenectomy with periesophagogastric devascularization in hepatitis B cirrhosis-related portal hypertension.

Key words: Hypertension; Portal; Thrombosis; Splenectomy; Diagnosis

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INTRODUCTION

As a potentially fatal complication, portal vein thrombosis (PVT) can aggravate liver damage and increase the risk of gastrointestinal bleeding[1]. PVT can also increase the difficulty of future liver transplantation[2,3]. The incidence of PVT including splenic vein, superior mesenteric vein, or portal vein thrombosis after splenectomy with periesophagogastric devascularization in hepatitis B cirrhosis-related portal hypertension is 13.4%-43.5%. At present, most of patients with PVT undergo specific treatments after the diagnosis with color Doppler, computed tomography or magnetic resonance imaging. Alternatively, some patients undergo preventive measure, such as antiplatelet and anticoagulation therapy. However, it is not clear in PVT correlates with an increase in blood platelets count. The preventive effect of antiplatelet and anticoagulation therapy on PVT is also not conclusive. The prevention of PVT after splenectomy with periesophagogastric devascularization remains uncertain. A preoperative predictor of PVT is urgently required to guide clinical practice, to assist in the selection of an appropriate surgical procedure, and for considering the success of future liver transplantation.

MATERIALS AND METHODS

Subjects

From January 2007 to August, 2010, 69 patients with portal hypertension caused by hepatitis B cirrhosis underwent splenectomy with periesophagogastric devascularization in the same medical group in the West China Hospital of Sichuan University. Thirty-three (47.83%) cases suffered from postoperative PVT. The ages of the cases ranged from 35 to 68 years (mean 37.3 ± 10.7 years). The inclusion criteria included clinically diagnosed portal hypertension caused by hepatitis B-induced cirrhosis in patients with a history of upper gastrointestinal hemorrhage or severe hypersplenism [white blood cell counts $< 3 \times 10^3$/dL or/and platelet (PLT) $< 50 \times 10^3$/dL]. The values of the patients' platelet count ranged from 9×10^3/dL to 85×10^3/dL (mean $33.2 \times 10^3 \pm 15.9 \times 10^3$/dL). White blood cell counts ranged from 0.9×10^3/dL to 5.7×10^3/dL (mean $2.4 \times 10^3 \pm 1.0 \times 10^3$/dL). All 69 patients underwent routine preoperative endoscopic examination. The esophageal varices were evaluated by Dagradi classification[4,5]. All patients had endoscopically confirmed esophageal varices, with four mild cases, 15 moderate cases, and 50 severe cases. Sixteen cases were associated with the red-color sign. Thirty-nine cases had gastric fundus varices. Thirty patients had at least one previous instance of upper gastrointestinal bleeding. Before surgery, 33 cases were grade A according to the Child classification. Twenty-four cases were grade B and 12 cases were grade C. According to the Pugh-modified Child's score scale, the patients' scores ranged from 7 to 11 (mean 7.59 ± 1.22). According to the model for end-stage liver disease score scale, the patients' score ranged from 5 to 12 (mean 7.71 ± 2.43).

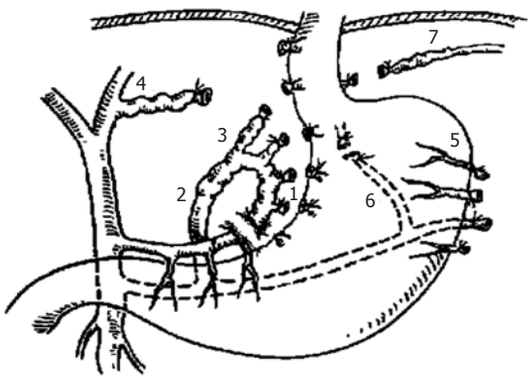

Figure 1 The anatomy of the lower part of the esophagus and periesophagogastric area after surgery. 1: Gastric branch of gastric coronary vein; 2: Esophageal branch of gastric coronary vein; 3: High esophageal branch of gastric coronary vein; 4: Aberrant high esophageal branch of gastric coronary vein; 5: Gastric short vein; 6: Gastric posterior vein; 7: Left subphrenic vein.

Operation

Patients underwent surgery similar to that previously described in detail by Yang and Qiu[6]. In brief, an extended left subcostal incision or incision of the left upper abdomen was used for extreme splenomegaly. Routine splenectomy was an important part of periesophagogastric devascularization. The right gastric vein was disconnected near the gastric angular incisura. Then, the gastric branch of the right gastric vein and 5-8 small branches of the gastric coronary veins were disconnected. The esophageal branch was then disconnected and suture-ligated up to 7-9 cm of the esophageal inferior segment. The high esophageal branch went anteriorly and upward near the left-lateral hepatic lobe, and entered into the esophageal muscular layer at 4-6 cm above the cardia, and this branch should be disconnected. The gastric posterior veins and short gastric veins were ligated with sutures, and then the left subphrenic vein was also ligated. 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. The net weight of the spleens were determined after surgery (Figure 1).

Perioperative treatment

Preoperatively, patients underwent treatment to improve their functional hepatic reserve and blood clotting function [Vitk1 (20 mg), qd; 10% GS (500 mL) + 10% KCl (15 mL) + $MgSO_4$ (5 g) + RI (10 U), qd; BCAA (500 mL), qd]. On the day of surgery, Vitk1 (20 mg) and Reptilase (2000 U) were administered. On postoperative day (POD) 1, only Vitk1 (20 mg) was used. After POD 1, no hemostatic agent was administered. Postoperative

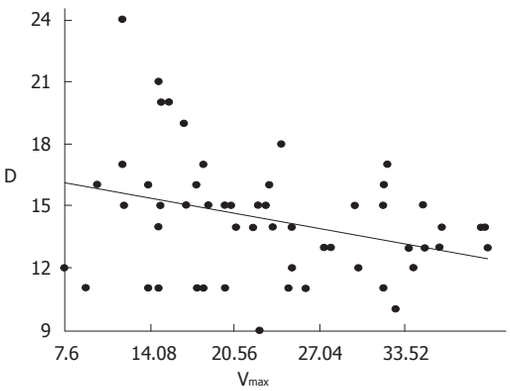

Figure 2 The diameter of the portal vein is negatively correlated with the preoperative maximum portal vein flow velocity. V_{max}: The mean maximum portal blood flow velocity; D: Portal vein diameter.

patients underwent treatment to improve their functional hepatic reserve.

Color Doppler ultrasound detection

Color Doppler ultrasound detection was performed by a color Doppler ultrasound system (Biosound AU 4, Esaote, Italy) on preoperative day 1 and postoperative day 7, respectively. For each measurement, at least three reproducible patterns were created to calculate the mean maximum portal blood flow velocity (V_{max}) at the midposition of the main portal vein. The mean portal blood velocity (V_{mean}) was calculated using the formula "$V_{mean} = 0.57 \times V_{max}$" as described by Moriyasu *et al*[7]. The portal vein diameter was also measured at the midposition of the main portal vein. Hepatic congestion index (CI) and the ratio of velocity and diameter (V_{max}/D) were calculated before surgery.

CI = portal vein cross sectional area/portal vein mean flow velocity = $(\pi \times D^2/4)/ V_{mean} = (\pi \times D^2/4)/0.57 \times V_{max}$

Ratio of velocity and diameter= the maximum portal blood flow velocity/ the diameter of the portal vein = V_{max}/D

All patients underwent routine PLT and prothrombin time (PT) tests on preoperative day 1 and postoperative day 7.

Statistical analysis

Numeration data: patients were divided into two groups according the presence or absence of postoperative PVT, or were divided into two groups respectively according to the respective cut-off values of V_{max}, V_{max}/D, CI and D. Numeration data was analyzed by χ^2 tests. Measurement data: results were expressed as mean ± SD and were analyzed by paired-sample *t* test and by receiver operating characteristic (ROC) curves. All calculations were performed using the SPSS 12.0 statistical software. Results with *P* values < 0.05 (paired-tailed test) were considered statistically significant.

The Ethics Committee of our hospital approved the study, and all patients gave their informed consent prior to their inclusion into this investigation.

RESULTS

Postoperative complications

After surgery, 33 cases suffered from PVT, including splenic vein, superior mesenteric vein, or portal vein thrombosis. One case suffered from main portal vein complete obstruction, superior mesenteric vein, and splenic vein thrombosis and died on POD 7. The patient had suffered from hepatic encephalopathy and upper gastrointestinal hemorrhage before surgery, and had no opportunity to undergo liver transplantation. No patient suffered from hepatic encephalopathy after surgery. The remaining 68 patients have remained well postoperatively.

Correlation between preoperative maximum portal blood flow velocity and the diameter of portal vein

The preoperative maximum portal blood flow velocity of the 69 cases ranged from 7.6 cm/s to 40.0 cm/s, and the mean value was (24.18 ± 9.08) cm/s. The diameters of their portal veins ranged from 9mm to 24mm (mean value 14.22 ± 2.86 mm). The diameter of portal vein was negatively correlated with the portal vein flow velocity, with the linear regression equation being Y = 1.6955 - 0.0113X ($F = 9.88$, $P < 0.05$) (Figure 2).

Analytic results of the differences between the portal vein thrombosis and non-portal vein thrombosis groups

The preoperative maximum portal blood velocity of the group with PVT was 18.06 ± 5.97 cm/s (7.6-32.3 cm/s), the preoperative maximum portal blood velocity of the group without PVT was 29.79 ± 7.75 cm/s (14.0-40.0 cm/s). The diameter of portal vein of the group with PVT was 15.39 ± 2.97 mm (11-24 mm), the diameter of portal vein of the group without PVT was 13.17 ± 2.31 mm (9-21mm). The hepatic congestion index of the group with PVT was 0.2126 ± 0.1243 cm/s^{-1} (0.0641-0.6614 cm/s^{-1}), hepatic congestion index of the group without PVT was 0.0942 ± 0.0702 cm/s^{-1} (0.041881-0.410575 cm/s^{-1}). The ratio of velocity and diameter of the group with PVT was 12.1774 ± 4.7493/s (5-23.63636/s), the ratio of velocity and diameter of the group without PVT was 23.3167 ± 6.7956 cm/s^{-1} (7.047619-32.9/s). All of the above-mentioned four variables showed the statistically significant difference between the two groups ($P < 0.05$). There was no statistically significant difference in the Child-Pugh score, the net weight of the patients' spleens, the value of PT and PLT count between the two groups (Table 1).

Receiver operating characteristic curve analysis

The ROC curve (Figure 3) showed that the two variables V_{max} and V_{max}/D could be used as preoperative predictors of postoperative portal vein thrombosis. The respective values of the area under the curve were 0.865 (asymptotic 95% confidence interval: 0.780-0.950) and 0.893 (asymptotic 95% confidence interval: 0.815-0.970), and the respective cut-off values (24.45 cm/s and 19.4333 /s) were diagnostically efficient, with sensitivities of 87.9% and 93.9%, respectively, specificities of 75% and 77.8%, respectively (Figure 3).

Table 1 Differences between patients with and without portal vein thrombosis

Group	Group with PVT (n = 33)	Group without PVT (n = 36)	Tort' value	P value
Preoperative maximum portal blood velocity (cm/s)	18.06 ± 5.97	29.79 ± 7.75	6.9978	< 0.05
The diameter of portal vein (mm)	15.39 ± 2.97	13.17 ± 2.31	3.4935	< 0.05
Hepatic congestion index (cm/s^{-1})	0.2126 ± 0.1243	0.0942 ± 0.0702	4.812	< 0.05
The ratio of velocity and diameter (/s)	12.1774 ± 4.7493	23.3167 ± 6.7956	7.9439	< 0.05
The net weight of spleen (g)	619.4 ± 132.6	636.4 ± 235.3	0.3736	> 0.05
Preoperative PLT count (× 10^9/L)	31.5 ± 14.3	34.8 ± 17.2	0.8622	> 0.05
Postoperative PLT count (× 10^9/L)	237.8 ± 84.4	267.7 ± 137.6	1.0978	> 0.05
Preoperative PT value (s)	15.6 ± 1.4	15.9 ± 1.3	0.923	> 0.05
Postoperative PT value (s)	16.0 ± 1.8	16.4 ± 1.6	0.9772	> 0.05
Child-Pugh score	7.54 ± 1.24	7.63 ± 1.22	0.3037	> 0.05

PVT: Portal vein thrombosis; PLT: Platelet; PT: Prothrombin time.

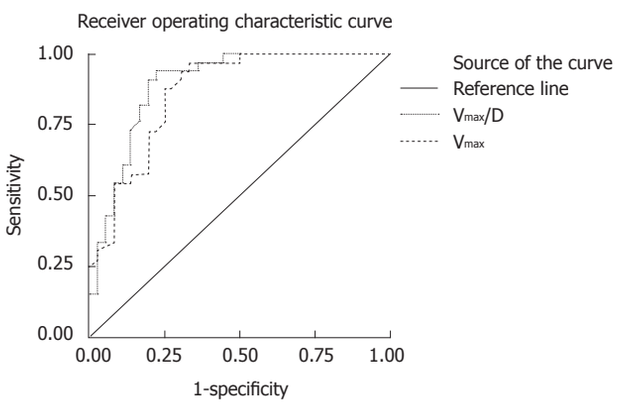

Figure 3 Receiver operating characteristic curve of the mean maximum portal blood flow velocity and Vmax/portal vein diameter. Vmax: The mean maximum portal blood flow velocity; D: Portal vein diameter.

Analytic results for the difference between cases with Vmax under 24.45 cm/s (Vmax ≤ 24.45 cm/s) and cases with Vmax above 24.45 cm/s (Vmax > 24.45 cm/s)

The mean preoperative maximum portal blood velocity of the group with Vmax under 24.45 cm/s (n = 38) was 17.10 ± 4.60 cm/s (7.6-24.2 cm/s). The mean preoperative maximum portal blood velocity of the group with Vmax above 24.45 cm/s (n = 31) was 32.85 ± 4.46 cm/s (24.7-40.0 cm/s). Twenty-nine cases suffered from PVT in the group with Vmax under 24.45 cm/s (29/38, 76.32%); only four cases suffered from PVT in the group with Vmax above 24.45 cm/s (4/31, 12.90%). The incidence of PVT in the cases with Vmax under 24.45 cm/s was significantly higher than in the cases with Vmax above 24.45 cm/s (χ^2 = 27.51, P < 0.05) (Table 2).

Analytic results for the differences between cases with Vmax/D under 19.43/s (Vmax/D ≤ 19.43/s) and cases with Vmax/D above 19.43/s (Vmax/D > 19.43/s)

The mean value of cases with Vmax/D under 19.43/s (n = 39) was (11.79 ± 3.99)/s (5.00-19.00/s), the mean value of cases with Vmax/D above 19.43/s (n = 30) was (26.05 ± 3.82)/s (19.87-32.90/s). Thirty-one cases suffered from PVT in the group with Vmax/D under 19.43/s (31/39, 79.49%); only two cases suffered from PVT in the group with Vmax/D above 19.43/s (2/30, 6.67%). The incidence of PVT in the cases with Vmax/D under 19.43/s was significantly higher in than the cases with Vmax/D above 19.43/s (χ^2 = 36.04, P < 0.05) (Table 2).

Receiver operating characteristic curve analysis of variable congestion index and D

This ROC curve (Figure 4) showed that the two variables (CI and D) could also be used as preoperative predictors of postoperative portal vein thrombosis. The respective values of the area under the curve were 0.884 (asymptotic 95% confidence interval: 0.799-0.970) and 0.742 (asymptotic 95% confidence interval: 0.624-0.861), and the respective cut-off values (0.1138 cm/s^{-1} and 13.5 mm) were diagnostically efficient, with sensitivities of 87.9% and 81.8%, respectively, and specificities of 86.1% and 63.9%, respectively (Figure 4).

Analytic results for the differences between cases with congestion index under 0.1138 cm/s^{-1} and cases with congestion index above 0.1138 cm/s^{-1}

The mean CI of cases with CI under 0.1138 cm/s^{-1} (n = 35) was 0.0733 ± 0.0190 cm/s^{-1} (0.041 881-0.112 652 cm/s^{-1}). The mean CI of cases with CI above 0.1138 cm/s^{-1} was 0.2306 ± 0.1193 cm/s^{-1} (0.114 922-0.661 389 cm/s^{-1}). Twenty-nine cases suffered from PVT in the group with CI above 0.1138 cm/s^{-1} (29/34, 85.29%); four cases suffered from PVT in the group with CI under 0.1138 cm/s^{-1} (4/35, 11.43%). The incidence PVT in the cases with CI above 0.1138 cm/s^{-1} was significantly higher than in the cases with CI under 0.1138 cm/s^{-1}(χ^2 = 37.71, P < 0.05) (Table 2).

Analytic results for the differences between cases with D under 13.5 mm and cases with D above 13.5 mm

The mean diameter of cases with D under 13.5 mm (n = 40) was 11.79 ± 1.21 mm (9-13 mm). The mean diameter of cases with D above 13.5mm was 16.00 ± 2.35 mm (14-24 mm). Twenty-seven cases suffered from PVT in the group with Vmax/D above 13.5 mm (27/40, 67.5%). Six cases suffered from PVT in the group with D under 13.5 mm (6/29, 20.69%). The incidence of PVT in the cases with D above 13.5mm was significantly higher than in the cases with D under13.5 mm (χ^2 = 14.76, P < 0.05) (Table 2).

Table 2 Analysis of respective variables

Variable(s)	Area under the curve	Cut-off value	Sensitivity (%)	Specificity (%)	Cases above the value	Cases with PVT	Cases under the value	Cases with PVT	χ^2 value	*P* value
V_{max}	0.865	24.45 cm/s	87.9	75	31	4	38	29	27.51	< 0.05
V_{max}/D	0.893	19.4333/s	93.9	77.8	30	2	39	31	36.04	< 0.05
CI	0.884	0.1138 cm/s^{-1}	87.9	86.1	34	29	35	4	37.71	< 0.05
D	0.742	13.5 mm	81.8	63.9	40	27	29	6	14.76	< 0.05

PVT: Portal vein thrombosis; CI: Congestion index; V_{max}: The mean maximum portal blood flow velocity; D: Portal vein diameter.

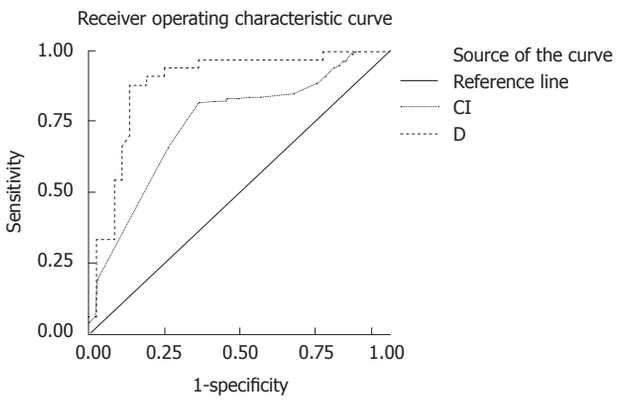

Figure 4 Receiver operating characteristic curve of hepatic congestion index and portal vein diameter. CI: Congestion index; D: Portal vein diameter.

DISCUSSION

The causes of PVT after splenectomy with periesophagogastric devascularization are disputed. Extrahepatic portal vein thrombosis frequently results from multiple concurrent factors, including procoagulant states and underlying myeloproliferative disorders (MPDs). The JAK2 V617F mutation is a point mutation in the Janus kinase 2 (JAK2) tyrosine kinase that is variably present in MPDs. The role of screening for the JAK2 V617F mutation in patients presenting with thrombosis without overt MPD is unclear, but appears justified in cases of idiopathic splanchnic vein thrombosis[8]. Silvia's research showed that pre-operative splenic vein diameter is a risk factor for portal-splenic vein thrombosis after laparoscopic splenectomy[9]. Shetty's research showed that among the acquired thrombophilias, MPD are the most frequent cause, while antiphospholipid antibodies and hyperhomocysteinemia have not shown very strong association with PVT[10]. Many scholars think that the rebound in PLT count post splenectomy and the hypercoagulable state cause postoperative PVTs in hepatitis B cirrhosis-related portal hypertension. Similar to Roberto's research[11], our research showed that there was no statistically significant difference in the Child-Pugh score, the net weight of the patients' spleens, the value of PT and the PLT count between the group with PVT and group without a PVT. However, our research did show a statistically significant difference in the preoperative maximum portal blood velocity and the diameter of the portal vein between the two groups. This indicated that the rebound in PLT count was not the main cause of postoperative PVTs. The occurrence of postoperative PVTs also did not show any correlation with the Child-Pugh score or the net weight of spleens.

Our research showed that preoperative maximum portal vein flow velocity in patients with postoperative PVT was significantly lower than in patients without postoperative PVT. The diameter of the portal vein in patients with a PVT was significantly wider than in patients without a PVT. Thus, the preoperative portal vein flow velocity and the diameter of portal vein were the important factors influencing the incidence of postoperative PVT. Our study showed that the diameter of the portal vein was negatively correlated with the preoperative maximum portal vein flow velocity. Considering to that result, the hepatic CI and the ratio of velocity and diameter (V_{max}/D) were both calculated before surgery.

ROC curves showed that four variables (V_{max}, V_{max}/D, CI and D) could be used as preoperative predictors of postoperative portal vein thrombosis. The area under the curve of V_{max}/D was the largest (0.893); therefore, V_{max}/D was the most accurate preoperative predictor of portal vein thrombosis after splenectomy with periesophagogastric devascularization in hepatitis B cirrhosis-related portal hypertension.

After surgery, four (21.90%) cases suffered from PVT in the group with V_{max} above 24.45 cm/s and only two (6.67%) cases suffered from PVT in the group with V_{max}/D above 19.43/s. Four cases (11.43%) suffered from PVT in the group with CI under 0.1138 cm/s^{-1} and six cases (20.69%) suffered from PVT in the group with D under 13.5 mm. Okuda *et al*[12] reported that the natural incidence of PVT was 6.6% in patients with hepatitis B cirrhosis-related portal hypertension that have not undergone surgery. We also showed that the incidence of PVT was very low in the patients with V_{max} above 24.45 cm/s, V_{max}/D above 19.43/s, CI under 0.1138 cm/s^{-1}, or D under 13.5 mm.

Reports concerning the incidence of portal vein thrombosis splenectomy with periesophagogastric devascularization are very uniform. Our study showed that postoperative portal vein thrombosis is mainly due to the change of portal vein blood flow dynamics, rather than a change in the value of PT or the PLT count. The incidence of postoperative PVT did not show any correlation with the Child-Pugh score or the net weight of the patients' spleens. The change of portal vein blood flow dynamics in patients with portal hypertension included

decreased portal vein velocity and increased portal vein diameter[13,14]. The decreased blood flow velocity can lead to the development of thrombus, and even the formation of eddy currents; increased portal vein diameter would lead to a vortex, causing venous intimal damage and "atherosclerosis-like" changes. In part, endothelial cells' detachment and collagen exposure would lead to blood cell adhesion and thus thrombosis. When V_{max}/D was above 19.43/s, postoperative thrombosis very unlikely to occur. According to the report of Deng *et al*[15], thrombosis mainly occurs in the perioperative period (within about one month after surgery). Thus, these patients with V_{max}/D above 19.43/s should have a relatively good prognosis, but it still require long-term follow-up. When the V_{max}/D is under 19.43/s, it is necessary to pay special attention to the prevention of a potential PVT after splenectomy with periesophagogastric devascularization. Further study is required to determine whether such patients require liver transplantation, but not splenectomy with periesophagogastric devascularization.

COMMENTS

Background

As a potentially fatal complication, portal vein thrombosis (PVT) can aggravate liver damage and increase the risk of gastrointestinal bleeding. PVT can also increase the difficulty of the future liver transplantation. The incidence of PVT, including splenic vein, superior mesenteric vein or portal vein thrombosis after splenectomy with periesophagogastric devascularization in hepatitis B cirrhosis-related portal hypertension, is 13.4%-43.5%.

Research frontiers

The preventive effect of antiplatelet and anticoagulation therapy on PVT is not conclusive. It is still unknown as to how to prevent PVT after splenectomy with periesophagogastric devascularization. A preoperative predictor of PVT is urgently required to guide clinical practice.

Innovations and breakthroughs

In this prospective study, 69 patients with portal hypertension caused by hepatitis B cirrhosis underwent splenectomy with periesophagogastric devascularization in West China Hospital of Sichuan University from January 2007 to August 2010. The portal vein flow velocity and diameter of portal vein were measured by Doppler sonography. Hepatic congestion index and the ratio of velocity and diameter were calculated before surgery, and the prothrombin time values and platelet levels were detected before and after surgery. The patients' spleens were weighed after surgery.

Applications

The ratio of velocity and diameter was most accurate as a preoperative predictor of portal vein thrombosis after splenectomy with periesophagogastric devascularization in hepatitis B cirrhosis-related portal hypertension.

Peer review

This is a prospective study of preoperative predictors for the risk of portal vein thrombosis after splenectomy with periesophagastric devascularization.

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