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Risk Factors and Management of Portal Vein Thrombosis after Hepatectomy

A Single-Center Experience

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Objective: This study investigated the risk factors and management of portal vein thrombosis (PVT) after hepatectomy.

Background: PVT after hepatectomy can cause liver dysfunction and portal hypertension, and may be fatal. However, it has not been sufficiently investigated.

Methods: The study included 1403 consecutive patients who underwent elective hepatectomy at our department from January 2010 to July 2022. The patients were divided into PVT and non-PVT groups based on the presence or absence of PVT, and relevant risk factors were analyzed. The management and prognosis of patients with PVT were investigated.

Results: Among the 1403 patients, PVT occurred in 33 cases, giving a frequency of 2.4%. In univariate analyses, female sex ($P = 0.03$), portal vein reconstruction ($P = 0.01$), and left lateral sectionectomy ($P < 0.001$) were significant risk factors for PVT. On multivariate analysis, portal vein reconstruction ($P = 0.01$) and left lateral segmentectomy ($P < 0.001$) remained significant risk factors for PVT. The management options for PVT were thrombectomy, antithrombotic therapy, and observation. With antithrombotic therapy, 96.4% of patients achieved PVT resolution. Among patients who underwent hepatectomy with portal vein reconstruction, the PVT site was the main trunk of the portal vein in all 3 cases, and thrombectomy was performed in 2 cases. No perioperative mortality was observed.

Conclusions: In the present study, portal vein reconstruction and left lateral sectionectomy were identified as risk factors for PVT after hepatectomy. As PVT can be fatal, early detection and appropriate treatment according to the status of PVT are important.

Keywords: antithrombotic therapy, hepatectomy, portal vein thrombosis, thrombectomy

INTRODUCTION

Hepatectomy is performed as a treatment for hepatobiliary diseases. Despite efforts to reduce complications through the development of surgical techniques, surgical devices, and postoperative management, hepatectomy still has a high incidence of postoperative complications and a high mortality rate compared with other surgical procedures. The main complications after hepatectomy include postoperative bleeding, bile leakage, liver failure, respiratory problems, and venous thrombosis, with portal vein thrombosis (PVT) being a frequent complication.[1–4] PVT occurs in 10% to 25% of patients with cirrhosis and is known to cause liver dysfunction and portal hypertension.[5] Although the pathogenesis and management of PVT associated with cirrhosis have been established, PVT after hepatectomy has not been sufficiently investigated. Posthepatectomy PVT is a complication that can be fatal if not properly treated, and it is necessary to elucidate its pathogenesis and establish methods for treatment and management. The present study investigated the risk factors and management of PVT after hepatectomy in a large cohort of patients.

METHODS

Patients

This was a retrospective single-center study approved by the Institutional Review Board of Hokkaido University Hospital (approval number 022-0172) and performed in compliance with the Declaration of Helsinki. Consecutive patients who underwent elective hepatectomy, excluding liver transplant donor surgery, at Hokkaido University Hospital from January 2010 to July 2022 were included in the study. Relevant patient data were collected from the hospital's database, which contains information on age, sex, diagnosis, preoperative Child-Pugh classification, surgical method, operative time, blood loss, and postoperative complications.

Surgical Procedure and Perioperative Management

The clinical indications and surgical methods for the patients were determined using the Hokkaido University Algorithm for hepatectomy, as described in our previous report.[6] Absence of uncontrolled ascites and serum total bilirubin ≤2 mg/dL were the criteria for hepatectomy, and indocyanine green retention rate at 15 minutes was used as an indicator for the acceptable

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range of hepatectomy. In cases requiring extensive hepatectomy, the remnant liver volume and effective liver resection rate were calculated based on preoperative volumetric computed tomography (CT), and percutaneous transhepatic portal vein embolization was performed preoperatively if the remnant liver volume was <400 mL or the effective liver resection rate was >60%.

Transection of the liver parenchyma was essentially performed using a Harmonic Synergy Hook Blade (Ethicon, San Angelo, TX) and a DS3.0 Dissection Sealer (Medtronic, Minneapolis, MN) for open hepatectomy or the clamp-crushing method with a Harmonic Shears (Ethicon) for laparoscopic hepatectomy. In cases where the Pringle maneuver was feasible, transection of the liver parenchyma was performed under this maneuver with intermittent inflow occlusion for 15 minutes and reperfusion for 5 minutes.

When portal vein resection was required, we selected the reconstruction method based on the extent of the resection, choosing from suture closure, patch reconstruction, end-to-end anastomosis, or graft reconstruction. We prefer to use an autologous left renal vein graft as the first choice. In this study, portal vein reconstruction was defined as cases where end-to-end anastomosis was performed. No cases in this study required the use of a graft.

Blood tests were performed on postoperative days (PODs) 1, 2, 3, 5, and 7. Abdominal Doppler ultrasound was performed as needed, and contrast-enhanced CT was performed when there was concern for the portal vein blood flow. If no problems were encountered during the postoperative course, routine contrast-enhanced CT was performed on POD 7 for open hepatectomy and POD 5 for laparoscopic hepatectomy. The contrast-enhanced CT was performed earlier on POD 5 for laparoscopic hepatectomy due to the minimally invasive nature of the procedure, which generally allows for a faster recovery and earlier assessment.

Diagnosis and Management of PVT

PVT was diagnosed on the contrast-enhanced CT examinations described above. In this study, PVT was classified as main type, hilar type, or peripheral type according to the classification proposed by Onda et al.[7] If PVT was judged to have no effect on the postoperative course, the patient was followed up without treatment. Specifically, this includes cases where the thrombus was a small peripheral thrombus or where the thrombus did not expand over time. If therapeutic intervention was deemed necessary, thrombectomy or antithrombotic therapy was selected. Thrombectomy was indicated for cases where the thrombus was located in the main trunk of the portal vein, with complete obstruction or the expectation of progression to complete obstruction. Drugs used for antithrombotic therapy included heparin, low-molecular-weight heparin, warfarin, direct-acting oral anticoagulant (DOAC), antiplatelet agent, and antithrombin III concentrate, and were selected at the discretion of the treating physician. Contrast-enhanced CT was performed periodically during treatment, and antithrombotic therapy was discontinued when the PVT had resolved or become organized and unchanging.

Statistical Analysis

The patients were divided into PVT and non-PVT groups according to the presence or absence of PVT, and relevant risk factors were compared. Statistical analyses were performed using EZR version 1.35 (Saitama Medical Center, Jichi Medical University, Saitama, Japan).[8] Fisher exact test was used for comparisons of categorical variables. Continuous variables were expressed as median with interquartile range (IQR), and compared using the Mann–Whitney *U* test. To identify the risk factors for PVT after hepatectomy, logistic regression models were used to conduct univariate and multivariate analyses. Values of $P \leq 0.05$ were considered to indicate statistical significance.

RESULTS

The database contained 1403 patients who underwent hepatectomy. Among these patients, there were 33 cases with PVT, giving a frequency of 2.4%.

Diagnoses of Hepatobiliary Tumors

The hepatobiliary tumors diagnosed in the study cohort are shown in Table 1. The main indications for hepatectomy were primary liver cancer ($n = 825$; 58.8%) and metastatic liver cancer ($n = 303$; 21.6%). The primary liver cancers were hepatocellular carcinoma in 678 patients, intrahepatic cholangiocarcinoma in 108 patients, combined hepatocellular cholangiocarcinoma in 18 patients, and other malignant tumors in 21 patients. The primary sources of metastatic liver cancer were colorectal cancer in 248 patients and other cancers in 55 patients. Biliary cancers included hilar cholangiocarcinoma in 94 patients and gallbladder carcinoma in 10 patients. The study included 104 patients with hepatic echinococcosis because our hospital is located in an area with a high incidence of the disease. The frequencies of PVT were 2.2%, 2.6%, 1.9%, 1.9%, and 4.8% for primary liver cancer, metastatic liver cancer, biliary cancer, alveolar echinococcosis, and benign liver tumor, respectively, with no significant difference by hepatobiliary tumor diagnoses ($P = 0.72$, $P = 0.67$, $P > 0.99$, $P > 0.99$, and $P = 0.20$, respectively).

Patient Characteristics and PVT After Hepatectomy

The background characteristics and surgical factors in the study cohort are shown in Table 2. The patients comprised 996 men (71.0%) and 407 women (29.0%) with a median age of 67 years (IQR, 60–74 years). The Child-Pugh classification was A in 1369 patients (97.6%) and B in 34 patients (2.4%). The surgical factors included laparoscopic surgery in 206 patients (14.7%), portal vein reconstruction in 33 patients (2.4%), and biliary reconstruction in 134 patients (9.6%). The median operation time was 306 minutes (IQR, 240–388 min), and the median blood loss was 270 mL (IQR, 100–582.5 mL). Postoperative complications of Clavien–Dindo classification IIIb or higher occurred in 19 cases (1.4%). In comparisons between the 2 groups, the frequency of PVT was significantly higher in women than in men (3.7% vs. 1.8%; $P = 0.05$). The

TABLE 1.

Diagnosis and PVT After Hepatectomy

Diagnosis	Overall (n = 1403)	PVT (n = 33)	Non-PVT (n = 1370)	*P*
Primary liver cancer	825 (58.8)	18 (2.2)	807 (97.8)	0.72
Metastatic liver cancer	303 (21.6)	8 (2.6)	295 (97.4)	0.67
Biliary cancer	104 (7.4)	2 (1.9)	102 (98.1)	>0.99
Alveolar echinococcosis	104 (7.4)	2 (1.9)	102 (98.1)	>0.99
Benign liver tumor	67 (4.8)	3 (4.5)	64 (95.5)	0.20

Data are presented as n (%).

TABLE 2.

Patient Characteristics and PVT After Hepatectomy

Variables		Overall (n = 1403)	PVT (n = 33)	non-PVT (n = 1370)	*P*
Age (years)		67 (60–74)	65 (59–72)	68 (60–74)	0.53
Sex	Male	996 (71.0)	18 (1.8)	978 (98.2)	0.05*
	Female	407 (29.0)	15 (3.7)	392 (96.3)	
Child-Pugh classification	A	1369 (97.6)	32 (2.3)	1337 (97.7)	0.55
	B	34 (2.4)	1 (2.9)	33 (97.1)	
Laparoscopic surgery	Yes	206 (14.7)	7 (3.4)	199 (96.6)	0.31
	No	1197 (85.3)	26 (2.2)	1171 (97.8)	
Portal vein reconstruction	Yes	33 (2.4)	3 (9.1)	30 (90.9)	0.04*
	No	1370 (97.6)	30 (2.2)	1340 (97.8)	
Biliary reconstruction	Yes	134 (9.6)	3 (2.2)	131 (97.8)	>0.99
	No	1269 (90.4)	30 (2.4)	1239 (97.6)	
Operation time (minutes)		306 (240–388)	306 (185–370)	306 (241–388)	0.47
Blood loss (mL)		270 (100–582.5)	170 (70–560)	277.5 (100–583.75)	0.28
Postoperative complication†	CD class≥IIIb	19 (1.4)	1 (3.0)	18 (1.3)	0.36

Data are presented as n (%) or median [IQR].

*P <0.05.

†Excluding PVT.

TABLE 3.

Surgical Methods and PVT After Hepatectomy

Surgical Methods	Overall (n = 1403)	PVT (n = 33)	non-PVT (n=1370)	*P*
Partial resection	349 (24.9)	4 (1.1)	345 (98.9)	0.28
Right lobectomy	298 (21.2)	4 (1.3)	294 (98.7)	0.28
Left lobectomy	199 (14.2)	0 (0)	199 (100)	0.009*
Segmentectomy	194 (13.8)	8 (4.1)	186 (95.9)	0.66
Right posterior sectionectomy	100 (7.1)	5 (5.0)	95 (95.0)	0.07
Right anterior sectionectomy	74 (5.3)	0 (0)	74 (100)	0.41
Left lateral sectionectomy	43 (3.1)	7 (16.3)	36 (83.7)	<0.001*
Right tresectionectomy	43 (3.1)	0 (0)	43 (100)	0.62
Left median sectionectomy	42 (3.0)	3 (7.1)	39 (92.9)	0.31
Central bisegmentectomy	35 (2.5)	2 (5.7)	33 (94.3)	0.19
Left trisectionectomy	26 (1.9)	0 (0)	26 (100)	>0.99

Data are presented as n (%).

*P <0.05.

frequency of PVT was also significantly higher in the group with portal vein reconstruction than in the group without portal vein reconstruction (9.4% vs. 2.2%; P = 0.04). Other factors did not differ significantly between the 2 groups.

Surgical Methods and PVT After Hepatectomy

The surgical methods used in the study cohort are shown in Table 3. The frequencies of PVT were compared among the methods. The table is ordered by the number of samples for each surgical method. For left lobectomy, PVT occurred in 0 of 199 cases (0%), and this frequency was significantly lower than those in the other surgical methods (P = 0.009). For left lateral sectionectomy, PVT occurred in 7 of 43 cases (16.3%), and this frequency was significantly higher than those in the other surgical methods (P < 0.001). The frequencies of PVT for left median sectionectomy, central bisegmentectomy, and right posterior sectionectomy tended to be slightly higher at 7.1%, 5.7%, and 5.0%, respectively, but the differences were not significant (P = 0.31, P = 0.19, and P = 0.07, respectively).

Analysis of Risk Factors for PVT After Hepatectomy

The above findings suggested that female sex, portal vein reconstruction, and left lateral sectionectomy were risk factors for PVT, and logistic regression analyses were performed on these factors (Table 4). In the univariate analyses, female sex [odds ratio (OR), 2.08; 95% confidence interval (CI) = 1.04–4.17; P=0.03], portal vein reconstruction (OR, 4.47; 95% CI = 1.29–15.40; P = 0.01), and left lateral sectionectomy (OR, 9.98; 95% CI = 4.06–24.50; P < 0.001) were confirmed as significant risk factors. In the multivariate analysis, portal vein reconstruction (OR, 5.18; 95% CI = 1.46–18.40; P = 0.01) and left lateral segmentectomy (OR, 10.70; 95% CI = 4.29–26.70; P < 0.001) remained significant factors, while female sex did not (OR, 1.93; 95% CI = 0.94–9.93; P = 0.06).

Management of PVT

The treatments for the patients with PVT and their outcomes are shown in Table 5. Two patients underwent thrombectomy, 28 received antithrombotic therapy, and 3 were followed up without therapeutic intervention. Regarding thrombectomy, 1 patient underwent thrombectomy on POD 5 and achieved resolution on POD 6, while the other had complete occlusion of the portal vein but did not experience any complications owing to the development of collateral flow. In the antithrombotic therapy group, PVT resolution was achieved in 27 of 28 patients (96.4%), and the median time to resolution was 45 days. The breakdown of antithrombotic therapy included 2cases with antiplatelet therapy, 24 with anticoagulant therapy, and 1 with antithrombin III concentrate. The improvement rates were 2/2 (100%), 24/25 (96%), and 1/1 (100%), respectively. In the 3 patients who were followed up without therapeutic intervention, the PVT resolved in 1 case.

TABLE 4.

Univariate and Multivariate Analysis of Risk Factors for PVT After Hepatectomy

	Univariate Analysis			Multivariate Analysis		
Variables	**Odds Ratio**	**95% CI**	***P***	**Odds Ratio**	**95% CI**	***P***
Female	2.08	1.04–4.17	0.03*	1.93	0.94–9.93	0.06
Portal vein reconstruction	4.47	1.29–15.40	0.01*	5.18	1.46–18.40	0.01*
Left lateral sectionectomy	9.98	4.06–24.50	<0.001*	10.70	4.29–26.70	<0.001*

*P <0.05.

All patients were discharged from the hospital after surgery, including the 4 cases without PVT resolution, and no perioperative mortality was observed. No postoperative bleeding was observed in the antithrombotic therapy group. Regarding the classification of PVT by site of occurrence, 5 cases were main type, 22 were hilar type, and 6 were peripheral type (Table 6). All 3 patients who underwent portal vein reconstruction were main type, and all 7 patients who underwent left lateral sectionectomy were hilar type, including the umbilical portion of the portal vein. The PODs for PVT diagnosis in the 3 types are shown in Supplemental Figure S1, http://links.lww.com/AOSO/A435. Among the 33 patients, 17 (51.5%) were diagnosed with PVT on routine contrast-enhanced CT conducted on POD 7. The 2 cases diagnosed with PVT on POD 1 were both main type with portal vein reconstruction. In all cases, there were no specific findings on physical examination or blood tests other than abdominal Doppler ultrasound and contrast-enhanced CT.

DISCUSSION

In this study, we retrospectively investigated the incidence, risk factors, treatment, and prognosis of PVT after hepatectomy in a large cohort of patients. The key findings were as follows: the frequency of PVT was 2.4%. Portal vein reconstruction and left lateral sectionectomy were identified as independent risk factors for PVT after hepatectomy. The management options for PVT were thrombectomy, antithrombotic therapy, and observation. With antithrombotic therapy, 96.4% of patients achieved PVT resolution, and the median time to resolution was 45 days. In hepatectomy with portal vein reconstruction, the PVT site was the main trunk of the portal vein in all 3 cases, and thrombectomy was performed in 2 cases. Other than Doppler ultrasound and contrast-enhanced CT, there were no findings specific for the PVT group, including blood test findings, and most cases were diagnosed on routine postoperative contrast-enhanced CT. No perioperative mortality was observed.

In previous studies, the incidence of PVT after hepatectomy has ranged from 2.1% to 14.3%.[7,9–15] Reported risk factors for PVT include age,[13] right lobectomy,[9,10] left lateral sectionectomy,[13] hepatectomy with caudate lobectomy,[10] portal vein reconstruction,[11,14] portal vein diameter ratio and angle,[12,14] liver resection volume,[9] operation time,[9] duration of Pringle maneuver,[7,9,11] and postoperative bile leakage.[10] Treatment options for PVT after hepatectomy include thrombectomy, thrombolysis therapy, antithrombotic therapy, and follow-up without intervention.[10,16] These options are selected according to the clinical symptoms and PVT status. If complete occlusion of the main trunk of the portal vein is observed, thrombectomy and/or thrombolysis therapy are indicated immediately. Recently, there have been several reports on the effectiveness of thrombolysis therapy via the ileocolic mesenteric vein, in which heparin and urokinase are directly administered to the PVT.[17–19] If the obstruction is incomplete and the above treatments are not indicated, antithrombotic therapy is recommended. Drugs used for antithrombotic therapy include heparin, warfarin, antithrombin III, enoxaparin, and danaparoid.[13,20] If the patient is judged to be clinically normal, such as the presence of a thrombus in the periphery, follow-up without intervention is indicated.

A few reports have described the prevention and predictive markers of PVT after hepatectomy. Yamashita et al.[20] found that postoperative antithrombotic therapy with enoxaparin could prevent PVT after hepatic resection for liver cancers. For cases of right lobectomy with caudate lobectomy in particular, Kuboki et al.[10] indicated that the portal vein may be morphologically folded, and that reconstruction to straighten the portal vein should be considered by suturing the posterior wall of the portal vein to the anterior wall of the inferior vena cava, and anchoring the left lobe of the liver to the abdominal wall. Okuno et al.[21] reported that a low postoperative level of plasma antithrombin III was associated with PVT after liver surgery. There are no other reported symptoms or serum markers specific for PVT after hepatectomy, and diagnosis is made by Doppler ultrasound and contrast-enhanced CT.

TABLE 5.

PVT Treatment and Results

Treatment	**n**	**Disappearance of PVT**	**Time to Resolved (days)**
Thrombectomy	2	1 (50.0)	6 [6, 6]
Antithrombotic therapy	28	27 (96.4)	45 [7, 144]
Observation	3	1 (33.3)	34 [34, 34]

Data are presented as n (%) or median [minimum, maximum].

TABLE 6.

Treatment and Results for Each PVT Classification

PVT Type	**N**	**Treatment**	**n**	**Disappearance of PVT**	**Time to Resolved (days)**
Main	5	Thrombectomy	2	1 (50.0)	6 [6, 6]
		Antithrombotic therapy	3	3 (100.0)	31 [7, 67]
Hilar	22	Antithrombotic therapy	20	19 (95.0)	47 [36, 66]
		Observation	2	1 (50.0)	34 [34, 34]
Peripheral	6	Antithrombotic therapy	5	5 (100.0)	45 [12, 144]
		Observation	1	0 (0.0)	N/A

Data are presented as n (%) or median [minimum, maximum].

PVT classification: Type 0, main trunk; Type 1, first branch; Type 2, second branch; Type 3, peripheral.

TABLE 7.

Previous Reports on PTV After Hepatectomy Outcomes

Author	Total Number	PVT Number	Resolved Number			Postoperative Death
			Thrombectomy	Antithrombotic Therapy	Observation	
Yoshiya et al[7]	708	19	N/A	9/9 (100)	6/10 (60)	0
Kuboki et al[8]	1193	25	9/11 (81.8)	12/14 (85.7)	N/A	2
Han et al[9]	534	19	2/2 (100)	9/9 (100)	7/8 (87.5)	0
Uchida et al[10]	81	9	1/1 (100)	5/6 (83.3)	2/2 (100)	0
Mori et al[11]	622	21	0/1 (0)	12/15 (80.0)	5/5 (100)	1
Onda et al[12]	398	57	2/2 (100)	29/34 (85.2)	21/21 (100)	0
Terasaki et al[13]	247	20	4/4 (100)	16/16 (100)	N/A	2
Lemaire et al[14]	86	7	5/5 (100)	2/2 (100)	N/A	1
Our study	1403	33	1/2 (50.0)	27/28 (96.4)	1/3 (33.3)	0

Data are presented as n (%).

The outcomes of PVT after hepatectomy in previous reports and the present study are summarized in Table 7. Thrombectomy is often indicated for complete occlusion of the main trunk of the portal vein, and the improvement rate is high, but there have been reports of postoperative death resulting from failure to achieve resolution. Kuboki et al.[10] found that patients who underwent thrombectomy on or after POD 6 had poorer outcomes than those who underwent thrombectomy before POD 6. These findings demonstrate the importance of early detection and appropriate treatment of PVT. The resolution rate with antithrombotic therapy is 80% to 100%, and the treatment outcomes are generally good. Even when the treatment decision is observation, the spontaneous resolution rate is relatively good at 60% to 100%. These outcomes provide further evidence for the importance of early detection and appropriate treatment of PVT, especially in cases with complete occlusion of the main trunk of the portal vein, which can be fatal if treatment is delayed.

The etiology of postoperative PVT can be categorized based on Virchow's triad of venous stasis, hypercoagulable state, and endothelial injury.[16] The interactive and combined effects of these factors may cause PVT. In the present study, portal vein reconstruction and left lateral sectionectomy were identified as risk factors for PVT, consistent with previous reports. In portal vein reconstruction, narrowing and endothelial damage may increase the risk of PVT. In left lateral sectionectomy, the umbilical portion of the portal vein is exposed, and thermal injury to this portion is thought to increase the risk of thrombosis.

As shown in Table 7, the outcomes for antithrombotic therapy in the present study were comparable to those in previous reports. DOAC drugs have recently been introduced and were used in 10 of the 28 patients who received antithrombotic therapy in the present study. Although there is no evidence regarding the use of these drugs for PVT after hepatectomy, DOAC use has been reported for PVT in patients with cirrhosis, and efficacy and safety have been demonstrated.[22] Thrombectomy achieved rapid improvement in 1 case but did not lead to resolution in the other case, resulting in complete occlusion. The latter patient did not develop postoperative liver failure because collateral flow developed before the occlusion was complete. In all cases, early therapeutic intervention was considered to have resulted in a favorable prognosis. However, the spontaneous resolution rate was low compared with those in other reports, and more aggressive antithrombotic therapy may need to be considered.

To our knowledge, this is the largest case–control study of PVT after hepatectomy. However, it was a single-center study, and biases in patient background characteristics, surgical techniques, and postoperative management may have influenced the results.

The present findings suggest that early detection and appropriate therapeutic intervention are important, given that PVT after hepatectomy can be fatal. Thrombectomy or thrombolysis therapy should be considered immediately, especially if there is a complete occlusion of the main trunk of the portal vein. Careful postoperative management is necessary in high-risk cases, such as those with portal vein reconstruction and left lateral sectionectomy. Since there are no specific findings for diagnosis of PVT in tests other than Doppler ultrasound and contrast-enhanced CT, it is important not to hesitate to perform these examinations if there is concern. DOACs are considered effective for antithrombotic therapy, but there is no evidence regarding their use for PVT after hepatectomy, and this issue should be investigated in future studies.

In conclusion, the risk factors for PVT after hepatectomy identified in the present study were portal vein reconstruction and left lateral sectionectomy. As PVT can be fatal, early detection and appropriate treatment according to the status of PVT are important.

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AUTHOR CONTRIBUTIONS

K.W. contributed to the conception and design, analysis and interpretation of data, and drafting the article. S.S., T.A., Y.A., A.N., T.O., and T.K. contributed to the acquisition of data and revising the article. A.T. contributed to the conception and design and revising the article. All authors gave final approval of the article.

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