MEDICAL SCIENCE MONITOR

CLINICAL RESEARCH

e-ISSN 1643-3750
© Med Sci Monit, 2017; 23: 2788-2795
DOI: 10.12659/MSM.904660

Received: 2017.04.02
Accepted: 2017.05.09
Published: 2017.06.08

Retrospective Study to Compare Selective Decongestive Devascularization and Gastrosplenic Shunt versus Splenectomy with Pericardial Devascularization for the Treatment of Patients with Esophagogastric Varices Due to Cirrhotic Portal Hypertension

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

ABDE Haili Bao*
BCDEF Qikuan He*
BC Ninggao Dai
BC Ruifan Ye
AG Qiyu Zhang

Department of General Surgery, The First Affiliated Hospital of Wenzhou Medical University, Wenzhou, Zhejiang, P.R. China

* Equal contributions to this paper

Corresponding Author: Qiyu Zhang, e-mail: Qiyuz2016@sina.com

Source of support: This work was supported by the National Natural Science Funding of China (grant number 81470874); the Zhejiang Provincial Natural Science Foundation of China (grant number LY13H030009)

Background: For patients with esophagogastric varices secondary to portal hypertension due to liver cirrhosis, portosystemic shunts and devascularization have become the most commonly used treatment methods. We have developed a novel surgical approach for the treatment of patients with cirrhotic portal hypertension, selective decongestive devascularization, and shunt of the gastrosplenic region (SDDS-GSR). This aim of this study was to compare the efficacy and safety of SDDS-GSR with splenectomy with pericardial devascularization (SPD).

Material/Methods: A retrospective study was undertaken between 2006 and 2013 and included 110 patients with cirrhotic portal hypertension, 34 of whom underwent SDDS-GSR; 76 patients underwent SPD. Kaplan-Meier analysis was used to evaluate clinical outcomes, mortality, the incidence of re-bleeding, encephalopathy, and portal venous system thrombosis (PVST).

Results: Postoperatively portal venous pressure decreased by 20% in both groups. The long-term incidence of re-bleeding and PVST was significantly lower in the SDDS-GSR group compared with the SPD group (P=0.018 and P=0.039, respectively).

Conclusions: This preliminary retrospective study has shown that SDDS-GSR was an effective treatment for patients with esophagogastric varices secondary to portal hypertension that may be used as a first-line treatment to prevent variceal bleeding and lower the incidence of PVST.

MeSH Keywords: Hypertension, Portal • Liver Cirrhosis • Postoperative Complications • Splenorenal Shunt, Surgical

Full-text PDF: http://www.medscimonit.com/abstract/index/idArt/904660

2743 5 4 25

Indexed in: [Current Contents/Clinical Medicine] [SCI Expanded] [ISI Alerting System] [ISI Journals Master List] [Index Medicus/MEDLINE] [EMBASE/Excerpta Medica] [Chemical Abstracts/CAS] [Index Copernicus]

Background

Due to the high incidence of chronic hepatitis B and C in Asia, an increasing number of patients now suffer from portal hypertension secondary to liver cirrhosis. Esophagogastric varices and hypersplenism secondary to portal hypertension are common major complications of liver cirrhosis. Patients with large esophagogastric varices have a 30% risk of bleeding over two years, which is a serious, life-threatening complication [1]. When the portal-systemic venous pressure gradient exceeds 12 mmHg in patients with cirrhosis, the risk of variceal bleeding is 30%, and the mortality within 30 days can be 20% [2].

There are several approaches to the treatment of varices and variceal hemorrhage, including medications, endoscopic variceal ligation (EVL), transjugular intrahepatic portosystemic shunt (TIPS), and liver transplantation. Though liver transplantation appears to be the most effective treatment, there remains a lack of liver donors, and the high medical costs limit the use of organ transplantation. Therefore, portosystemic shunting and devascularization have become the most commonly used treatment method [3]. The disadvantage of treatment with a portosystemic shunt is the resultant reduction in liver function, caused by the reduction in portal blood flow.

In contrast, pericardial devascularization does not affect liver function. Also, splenectomy with pericardial devascularization (SPD) has been widely accepted as a surgical treatment for cirrhosis in patients with variceal bleeding and secondary hypersplenism. SPD may be the most appropriate treatment for patients with cirrhotic portal hypertension, particularly when EVL and TIPS are unsuccessful or contraindicated. However, when compared with other treatments, SPD is associated with an increased incidence of postoperative complications, such as recurrent variceal hemorrhage. Therefore, there is a need for a more rational and effective surgical approach to the management of patients with esophagogastric varices secondary to portal hypertension due to liver cirrhosis.

The spleen is an important component of the immune system, and so splenectomy may leave the patient in an immunocompromised state [4]. Splenic resection may also be complicated by the development of pancreatic fistulas [3,5], systemic bacterial infection [6], and portal venous system thrombosis (PVST) [7]. Moreover, the destruction of the communicating perisplenic portosystemic branches may further increase portal venous pressure.

To reduce the incidence of PVST and to improve patient prognosis, in 2000, under the leadership of Professor Qi-Yu Zhang, our surgical team developed a novel surgical approach, namely selective decongestive devascularization and shunt of the gastrosplenic region (SDDS-GSR). This aim of this study was to compare the efficacy and safety of SDDS-GSR with SPD in patients with cirrhotic portal hypertension.

Material and Methods

Patients

Between January 2006 and December 2013, a total of 110 patients underwent either selective decongestive devascularization and shunt of the gastrosplenic region (SDDS-GSR) or splenectomy with pericardial devascularization (SPD). All patients had been diagnosed with liver cirrhosis, portal hypertension, and secondary hypersplenism. All procedures were performed by the same team of surgeons from the general surgery department of our hospital.

Before proceeding with surgery, all patients were informed that SDDS-GSR appeared to be a more effective procedure in comparison with SPD. The surgical procedure conducted was based on the patient's preference and their written informed consent.

Exclusion criteria were as follows: 1) patients who had hepatocellular carcinoma or any other malignancy; 2) patients in whom the splenic vein was not suitable for a shunt; 3) patients with a history of portal vein thrombosis; 4) patients with hypersplenism as a result of any other disease; 5) patients who were classified as Child–Pugh C; and 6) patients receiving anticoagulant or antiplatelet agents.

The general clinical patient details analyzed including patient age, sex, etiology of cirrhosis, and Child–Pugh classification. Data on platelet and white blood cell counts, liver function, and blood coagulation status were collected on pre-operative day 1 and postoperative day 7. The thickness of the spleen was measured by B-mode ultrasonography on pre-operative day 7 and postoperative month 1.

Intraoperative data collected included operation time, pre-operative and postoperative free portal pressure (FPP), and estimated intraoperative blood loss. Persistent hyperbilirubinemia (total bilirubin level >5 mg/dL) for more than 5 days after surgery was defined as postoperative hepatic insufficiency [8]. Patients were followed-up via telephone or in the outpatient clinic.

Operative procedures

SDDS-GSR was performed through a left rectus muscle incision or a left subcostal incision. The FPP was measured through the venous branch of the right gastroepiploic vein by a pressure sensor.

Indexed in: [Current Contents/Clinical Medicine] [SCI Expanded] [ISI Alerting System] [ISI Journals Master List] [Index Medicus/MEDLINE] [EMBASE/Excerpta Medica] [Chemical Abstracts/CAS] [Index Copernicus]

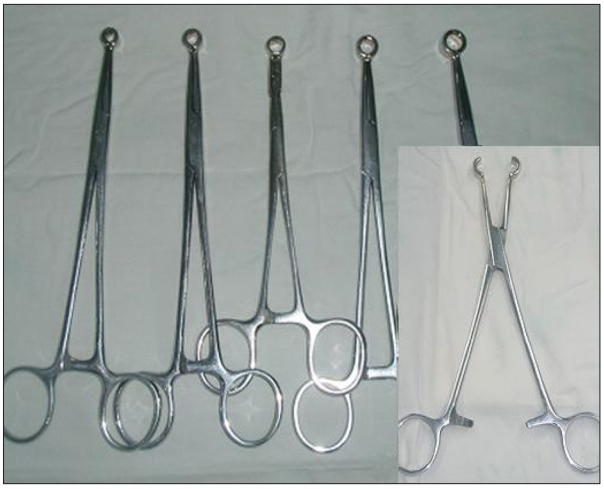

Figure 1. The coarctation forceps.

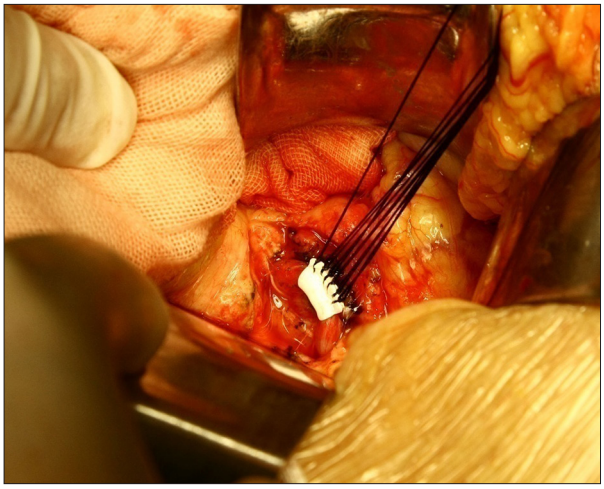

Figure 2. The splenic artery coarctated with the artificial artery.

After the initial incision, a 1.0–1.5 cm length of the middle segment of the main splenic artery was exposed on the upper edge of the pancreas. A special surgical instrument, termed coarctation forceps (Figure 1), was used to coarctate the isolated splenic artery and reduce its diameter by one-half to two-thirds. Using these forceps, the splenic artery blood flow was decreased effectively to the point where the splenic arterial output was normal or the arterial pulse was weakened, without causing a thrill. Then, based on the size of the coarctation forceps used, an adequate length (about 1 cm) of vascular prosthesis material was secured circumferentially around the splenic artery with the use of sutures (Figure 2).

Following splenic artery coarctation, as much of the splenic vein as possible was exposed and isolated from the posterior pancreas along the lower pancreatic margin, and all pancreatic branches of the splenic vein were ligated to ensure that pancreatic blood flowed to the liver. The left renal vein was then isolated for the splenorenal anastomosis. The distal splenic vein and left renal vein were connected in an end-to-side anastomosis, without distortion or tension. The anastomotic stoma was recommended to be more than 1.0 cm to ensure patency of the shunt and to provide sufficient decompression of the gastrosplenic region. Finally, ligatures or sutures were carefully placed in the posterior peritoneal tissue to prevent ascites or chylous leakage.

Next, the branches of the coronary vein together with the posterior peritoneal tissue were removed en bloc around the lesser curvature of the stomach, including gastric branches and the high esophageal branch, to prevent or reduce the likelihood of re-canalization of the coronary vein. The left gastric artery and the posterior gastric vein were also ligated. To achieve a more effective decompression effect of the stomach area, the collateral circulation between the coronary vein and azygous vein was preserved, as were the short gastric vessels. However, if there was no natural connection between the coronary vein and azygous vein detected by the routine magnetic resonance venogram (MRV) before operation, then the trunk and all branches of the coronary vein were removed. The left gastric artery and the right gastric artery around the greater curvature were disconnected, followed by suturing the posterior peritoneal tissue to prevent bleeding and chylous leakage. After surgery, the effect was detected by the pressure measurement of the spleen and stomach area and mesenteric area, respectively.

Among patients undergoing SPD, splenectomy with pericardial devascularization was carried out through either a left subcostal incision or a midline laparotomy, using a modified Hassab procedure [9]. Splenectomy was performed before pericardial devascularization.

Following the two types of surgical procedure, all patients were hospitalized and then followed-up by physical examination, laboratory tests, and radiologic examination in the short and long term, respectively.

Statistical analysis

Data were compared using the t-test and chi-squared test, where applicable, and presented as the mean ± standard deviation (SD). The Kaplan-Meier method was used to analyze long-term data when required. P values <0.05 were considered to be statistically significant. All statistical analysis was performed using SPSS 19.0 software (SPSS, Chicago, IL, USA).

Results

Between January 2006 to December 2013, 34 patients were treated with selective decongestive devascularization, and shunt

Indexed in: [Current Contents/Clinical Medicine] [SCI Expanded] [ISI Alerting System] [ISI Journals Master List] [Index Medicus/MEDLINE] [EMBASE/Excerpta Medica] [Chemical Abstracts/CAS] [Index Copernicus]

Table 1. Clinical characteristics of patients.

Characteristics	SDDS-GSR group (n=34)	SWPD group (n=76)	P value
Age (yr)	46.9±8.8	48.7±11.0	0.414
Sex			
Male	25	58	
Female	9	18	
Child–Pugh score	6.1±1.2	5.7±0.8	0.080
Child–Pugh classification			
A	23	64	
B	11	9	
Etiology of cirrhosis			
Hepatitis B virus	26	60	
Hepatitis C virus	0	1	
Alcohol	3	12	
Biliary cirrhosis	1	0	
Autoimmune hepatitis	1	1	
Cryptogenic cirrhosis	3	1	
Platelet count (×10^9/L)	63.1±31.9	50.1±24.3	**0.020**
White blood cells (×10^9/L)	3.2±2.0	2.8±2.6	0.394
ALT (U/L)	35.4±16.1	38.4±55.8	0.753
AST (U/L)	43.1±20.3	42.3±26.1	0.877
Total bilirubin (μmol/L)	16.7±7.3	19.4±11.6	0.209
Albumin (g/L)	36.9±4.6	36.5±5.0	0.734
Prothrombin time (s)	16.6±2.2	16.4±1.7	0.603
PTA (%)	67.4±12.7	67.5±11.6	0.940
INR	1.4±0.2	1.3±0.2	0.563

SDDS-GSR – selective decongestive devascularization shunt of the gastrosplenic region; SWPD – splenectomy with pericardial devascularization; ALT – alanine aminotransferase; AST – aspartamine aminotransferase; PTA – prothrombin time activity; INR – international normalized ratio.

of the gastrosplenic region (SDDS-GSR), and 76 patients underwent splenectomy with pericardial devascularization (SPD). The platelet count was greater in the SDDS-GSR group, but there were no other significant differences in other patient characteristics, including white blood cell count, hepatic function, and coagulation functions between the two groups (Table 1).

The postoperative free portal pressure (FPP) was reduced by almost 20% in both groups without difference between the two groups. However, in the SDDS-GSR group, the postoperative FPP was significantly decreased compared with the pre-operative value. Moreover, in the SDDS-GSR group, the postoperative spleen thickness was considerably reduced compared with the pre-operative thickness. The operation time was reduced in the SPD group compared with the SDDS-GSR group. There was no significant difference in intraoperative blood loss between the SDDS-GSR and SPD groups (Tables 2, 3).

The postoperative platelet and white blood cell counts of patients in the SDDS-GSR group were lower than those of

Indexed in: [Current Contents/Clinical Medicine] [SCI Expanded] [ISI Alerting System] [ISI Journals Master List] [Index Medicus/MEDLINE] [EMBASE/Excerpta Medica] [Chemical Abstracts/CAS] [Index Copernicus]

Table 2. Perioperative parameters.

Parameters	SDDS-GSR group (n=34)	SWPD group (n=76)	P value
Preoperative FPP (cm H_2O)	38.6±5.9	39.7±6.4	0.418
Postoperative FPP (cm H_2O)	31.6±5.6	32.6±4.9	0.422
Postoperative FPP/Preoperative FPP	0.8±0.1	0.8±0.1	0.764
Operation time (min)	267.1±61.6	184.0±66.2	**<0.001**
Intraoperative blood loss (mL)	463.6±474.9	542.1±630.7	0.524

SDDS-GSR – selective decongestive devascularization shunt of the gastrosplenic region; SWPD – splenectomy with pericardial devascularization; FPP – free portal venous pressure.

Table 3. Postoperative characteristics.

Parameters	SDDS-GSR group (n=34)	SWPD group (n=76)	P value
Platelet count (×10^9/L)	122.2±57.9	264.4±129.4	**<0.001**
White blood cells (×10^9/L)	6.7±3.2	9.9±4.0	**<0.001**
ALT (U/L)	35.9±26.3	54.7±74.0	0.154
AST (U/L)	51.7±26.4	59.3±56.6	0.457
Total bilirubin (μmol/L)	25.3±11.4	21.7±10.5	0.111
Albumin (g/L)	35.4±3.8	33.8±4.1	0.053
Prothrombin time (s)	16.6±2.4	16.2±1.6	0.409
PTA (%)	67.9±15.2	60.3±10.0	0.891
INR	1.4±0.3	1.3±0.2	0.412

SDDS-GSR – selective decongestive devascularization shunt of the gastrosplenic region; SWPD – splenectomy with pericardial devascularization; ALT – alanine aminotransferase; AST – aspartamine aminotransferase; PTA – prothrombin time activity; INR – international normalized ratio.

Table 4. Changes of some parameters in the SDDS-GSR group.

Parameters	Preoperative	Postoperative	P value
Platelet count (×10^9/L)	63.1±31.9	122.2±57.9	**<0.001**
Spleen thickness (cm)	55.6±9.1	47.2±8.1	**<0.001**
FPP (cm H_2O)	38.6±5.9	31.6±5.5	**<0.001**

SDDS-GSR – selective decongestive devascularization shunt of the gastrosplenic region; FPP – free portal venous pressure.

patients in the SPD group. Postoperatively, there were no significant differences in liver and coagulation function between the two groups (Table 4). The platelet count increased significantly when compared with the pre-operative count in the SDDS-GSR group (Table 3).

No perioperative mortality was observed in either of the two groups. No patients developed hepatic encephalopathy during hospitalization in both groups. Two patients in the SPD group experienced recurrent hemorrhage during hospitalization, and one patient developed hepatic insufficiency. The rates of portal venous system thrombosis (PVST), recurrent hemorrhage, and morbidity were relatively higher in the SPD group than in the SDDS-GSR group. There was no difference in the rate of hepatic encephalopathy (Table 5).

Twenty-nine of the 34 patients (85.2%) in the SDDS-GSR group were followed-up for a mean duration of 69.4 months. The

Indexed in: [Current Contents/Clinical Medicine] [SCI Expanded] [ISI Alerting System] [ISI Journals Master List] [Index Medicus/MEDLINE] [EMBASE/Excerpta Medica] [Chemical Abstracts/CAS] [Index Copernicus]

Table 5. Postoperative complications.

Complications	SDDS-GSR group (n=34)	SWPD group (n=76)	P value
PVST	6	18	**0.039**
Postoperative hemorrhage	0	2	0.475
Hepatic encephalopathy	3	9	0.890
Long-term rebleeding	2	19	**0.018**

SDDS-GSR – selective decongestive devascularization shunt of the gastrosplenic region; SWPD – splenectomy with pericardial devascularization; PVST – portal venous system thrombosis.

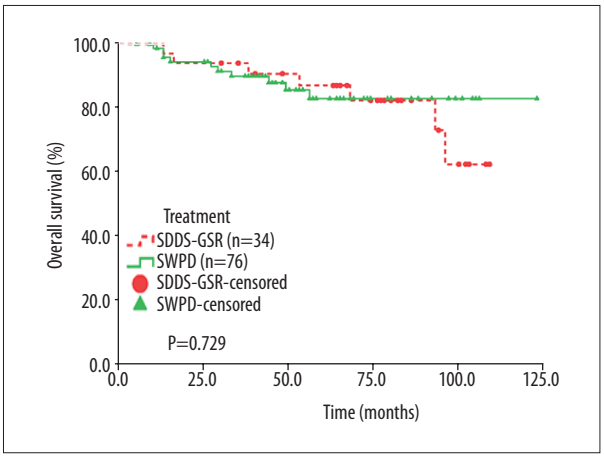

Figure 3. Kaplan-Meier curve for overall survival (OS) rate.

survival was 100% at one year and 87.1% (27/31) at three years in the SDDS-GSR group; one patient died of liver failure, and one patient died due to multiple system organ failure.

In the SPD group, the follow-up rate was 81.5% (61/76), with a mean follow-up duration of 53.3 months; patient survival was 98.7% at one year (one died of variceal re-bleeding) and 80.1% (55/68) at three years in the SPD group; one patient died from cerebral hemorrhage, two patients died from variceal re-bleeding, two patients died from primary hepatic cancer, and one patient died from renal failure.

The median overall survival (OS) time was 107.8 months (95% CI: 99–116 months) in the SPD group, with one-year and three-year OS rates of 100% and 81.5%, respectively. In the SDDS-GSR group, the median OS was 94.4 months (95% CI: 85–104 months), with one-year and three-year survival rates of 100% and 93.1%, respectively (Figure 3); the difference was not significant between the SPD and SDDS-GSR groups (P=0.729).

Discussion

Portal hypertension is a consequence of liver cirrhosis, but the mechanisms by which portal hypertension develops are complicated and are associated with changes in the vascular architecture of the liver, due to fibrosis and regenerative nodules [10]. Increased resistance of the portal venous system to blood flow from the viscera to the liver leads to the development of collaterals at sites of venous communication between the portal and systemic circulation [11]. The main aim of treatment for portal hypertension is to control variceal bleeding and to prevent re-bleeding.

Surgical treatment is considered to be a primary method for patients with portal hypertension [12,13]. Surgical treatments, including ligation of portosystemic collaterals, have been designed to prevent esophagogastric variceal bleeding [14]. However, the availability of many treatment modalities suggests that no single therapeutic modality brings entirely satisfactory outcomes for all patients or in all clinical situations. Currently, distal splenorenal shunt and devascularization surgeries are the two most commonly used methods. In 1981, a modified pericardial devascularization procedure was developed by Qiu Fazu [15] that is now widely used in China, where splenectomy is routinely performed with this procedure. However, a high rate of re-bleeding and a high rate of residual varices occurs, together with changes in the gastric mucosa following SPD [16–18]. Also, hepatic encephalopathy has been reported following this procedure, even though this procedure can preserve hepatic blood flow to maintain liver function and improve the microcirculation of the gastric mucosa [19,20]. A high rate of PVST has also been observed following splenectomy [17,18].

Therefore, in 2000, a novel procedure for treating patients with portal hypertension, selective decongestive devascularization, and shunt of the gastrosplenic region (SDDS-GSR), was developed at our institution by Professor Zhang. This new procedure allows preservation of the spleen and decreases splenic arterial flow volume by splenic artery coarctation and splenorenal shunting. SDDS-GSR can relieve splenomegaly and hypersplenism effectively and permanently, even in patients with severe splenomegaly and hypersplenism. In this study, we analyzed clinical data to determine the clinical safety and efficacy of the SDDS-GSR procedure.

Indexed in: [Current Contents/Clinical Medicine] [SCI Expanded] [ISI Alerting System] [ISI Journals Master List] [Index Medicus/MEDLINE] [EMBASE/Excerpta Medica] [Chemical Abstracts/CAS] [Index Copernicus]

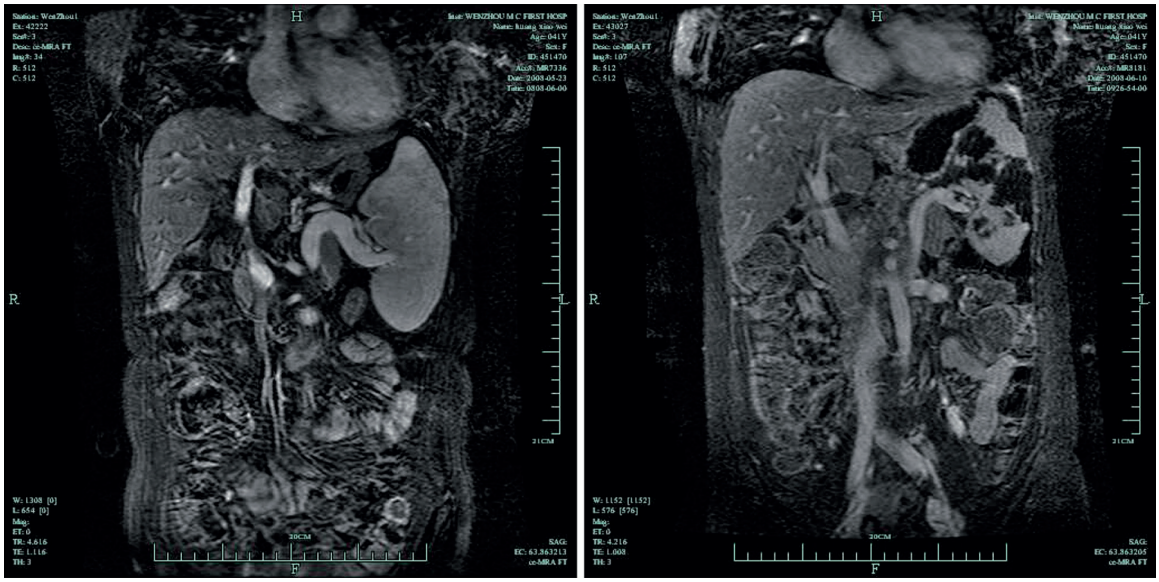

Figure 4. The abdominal magnetic resonance imaging (MRI) before and after selective decongestive devascularization and shunt of the gastrosplenic region (SDDS-GSR).

Previous studies have reported a re-bleeding rate of 7.1–37% in patients undergoing devascularization [21,22], while the re-bleeding rate after undergoing a distal splenorenal shunt is reported to be between 5–15% [1]. The incidence of recurrent variceal bleeding in the SDDS-GSR group in our study was significantly lower than that in the splenectomy with pericardial devascularization (SPD) group. This lower re-bleeding rate in the SDDS-GSR group is possibly the result of the following mechanisms. First, this procedure uses en bloc resection of the coronary vein branch to prevent postoperative venous recanalization. This procedure also reduces the risk of congestive gastropathy, which is an important cause of postoperative re-bleeding [22]. Second, the splenorenal shunt relieves portal hypertension to some extent and might delay the recurrence of gastroesophageal varices. Our study found that, among patients in the SDDS-GSR group, the postoperative free portal pressure (FPP) was reduced by almost 20% and was significantly lower than the pre-operative FPP. Third, because of the dilatation of the hepatic artery following ligation of the left gastric artery and coarctation of the splenic artery, liver blood flow was maintained, which helps to prevent hepatic failure and improves liver function, thereby reducing the rate of re-bleeding.

Previous studies have questioned whether splenectomy is an essential component of esophagogastric devascularization in the treatment of patients with portal hypertension. Wang et al. [23] proposed that the spleen in these patients is fibrotic and non-functional. However, postoperative complications, including portal venous system thrombosis (PVST), are often related to splenectomy. Some investigators have suggested that splenectomy should only be used in patients with portal hypertension with severe hypersplenism, PVST, or a markedly enlarged spleen. Other studies have found that splenectomy may aggravate immune dysfunction in patients with portal hypertension [23,24]. Therefore, we considered that only a limited number of patients with severe hypersplenism required splenectomy in our study, which showed that the incidence of PVST was significantly lower in patients undergoing SDDS-GSR compared with those undergoing SPD.

PVST is a major complication of pericardial devascularization surgery, and splenic vein thrombosis may extend to the portal and superior mesenteric veins, increasing the risk of re-bleeding. It has been suggested that the incidence of PVST after SPD in patients with hepatitis B cirrhosis-related portal hypertension is 13.4–43.5% [8]. One study found that an increase in the platelet count by >650×10^9/L at one week after surgery directly correlated to the occurrence of PVST [25]. A platelet count of more than 300×10^9/L was also found to be a risk factor for PVST, and in our study, the platelet and white blood cell counts both increased postoperatively in the SDDS-GSR group. Moreover, the spleen significantly decreased in size and infarcted without infection postoperatively (Figure 4). All of these postoperative changes indicated that hypersplenism had partially remitted, and rapid elevation of post-operative platelet count, which may cause PVST, was avoided. In conclusion, SDDS-GSR reduced the incidence of PVST and reduced the risk of recurrent esophagogastric variceal bleeding.

Indexed in: [Current Contents/Clinical Medicine] [SCI Expanded] [ISI Alerting System] [ISI Journals Master List] [Index Medicus/MEDLINE] [EMBASE/Excerpta Medica] [Chemical Abstracts/CAS] [Index Copernicus]

This study had several limitations. First, we did not evaluate the flow index, including the velocity of the portal venous blood stream, which is an important factor in predicting the outcome of SDDS-GSR. Second, while we believe that immune function may improve following SDDS-GSR, we did not assess any parameters of immunity in this study; this will require further studies. Finally, the number of the patients included in the study was limited. Additional larger studies should be conducted to confirm the benefits of this procedure.

Based on our results, we conclude that SDDS-GSR is a safe and effective treatment for patients with esophagogastric varices and hypersplenism secondary to portal hypertension. Moreover, SDDS-GSR is associated with a low risk of postoperative complications, especially PVST and recurrent esophagogastric variceal bleeding. Further studies of this surgical approach are warranted.

Conclusions

This preliminary retrospective study has shown that selective decongestive devascularization and shunt of the gastrosplenic region (SDDS-GSR) was an effective treatment for patients with esophagogastric varices secondary to portal hypertension that may be used as a first-line treatment to prevent variceal bleeding and lower the incidence of portal venous system thrombosis (PVST).

Acknowledgments

The authors would like to thank Huajun Yu for technical assistance.

References:

1. Henderson JM, Boyer TD, Kutner MH et al: Distal splenorenal shunt versus transjugular intrahepatic portal systematic shunt for variceal bleeding: A randomized trial. Gastroenterology, 2006; 130: 1643–51
2. Wright AS, Rikkers LF: Current management of portal hypertension. J Gastrointest Surg, 2005; 9: 992–1005
3. Wu J, Li Z, Wang Z et al: Surgical and endovascular treatment of severe complications secondary to noncirrhotic portal hypertension: Experience of 56 cases. Ann Vasc Surg, 2013; 27: 441–46
4. Lima Kde M, Negro-Dellacqua M, Dos Santos VE et al. Post-splenectomy infections in chronic schistosomiasis as a consequence of bacterial translocation. Rev Soc Bras Med Trop, 2015; 48: 314–20
5. Cheng Z, Li JW, Chen J et al. Therapeutic effects of laparoscopic splenectomy and esophagogastric devascularization on liver cirrhosis and portal hypertension in 204 cases. J Laparoendosc Adv Sur Tech A, 2014; 24: 612–16
6. Leone G, Pizzigallo E: Bacterial infections following splenectomy for malignant and nonmalignant hematologic diseases. Mediterr J Hematol Infect Dis, 2015; 7: e2015057
7. Ikeda M, Sekimoto M, Takiguchi S et al: High incidence of thrombosis of the portal venous system after laparoscopic splenectomy: A prospective study with contrast-enhanced ct scan. Ann Surg, 2005; 241: 208–16
8. Kim SU, Ahn SH, Park JY et al: Prediction of postoperative hepatic insufficiency by liver stiffness measurement (fibroscan((r))) before curative resection of hepatocellular carcinoma: A pilot study. Hepatol Int, 2008; 2: 471–77
9. Wu YK, Wang YH, Tsai CH et al. Modified Hassab procedure in the management of bleeding esophageal varices – a two-year experience. Hepatogastroenterology, 2002; 49: 205–7
10. Iwakiri Y, Groszmann RJ: Vascular endothelial dysfunction in cirrhosis. J Hepatol, 2007; 46: 927–34
11. Groszmann RJ, Garcia-Tsao G, Bosch J et al: Beta-blockers to prevent gastroesophageal varices in patients with cirrhosis. New Engl J Med, 2005; 353: 2254–61
12. Choy TY, Simoens C, Thill V et al. Results of surgical treatment of uncontrollable upper gastrointestinal hemorrhage using endoscopy. Hepatogastroenterology, 2011; 58: 89–95
13. Wang WM, Wang XG: [Effects of medicine in prevention and treatment of upper gastrointestinal hemorrhage in portal hypertension]. Zhonghua wai ke za zhi, 2007; 45: 865–67 [in Chinese]
14. de la Mora-Levy JG, Tamayo-de la Cuesta JL, Castaneda-del Rio AG et al: [Clinical guidelines for the diagnosis and treatment of nonvariceal upper gastrointestinal hemorrhage. Definition, epidemiology, etiology and physiopathology]. Rev Gastroenterol Mex, 2007; 72: 397–98 [in Spanish]
15. Qiu FZ: [Evaluation of the pericardial devascularization in portal hypertension]. Zhonghua wai ke za zhi, 1983; 21: 275–77 [in Chinees]
16. Chen FM, Wang JY, Huang TJ et al: The effect of portal hypertension on the glycoprotein biosynthesis of rat gastric mucosa. J Invest Surg, 2002; 15: 311–17
17. Ohta M, Yamaguchi S, Gotoh N et al: Pathogenesis of portal hypertensive gastropathy: A clinical and experimental review. Surgery, 2002; 131: S165–70
18. Thuluvath PJ, Yoo HY. Portal hypertensive gastropathy. Am J Gastroenterol, 2002; 97: 2973–78
19. Leng X, Zhu J, Du R: [Portacaval shunt with h-grafts of small diameter in treating cirrhotic patients with portal hypertension]. Zhonghua wai ke za zhi, 1998; 36: 330–32 [in Chinese]
20. Triger DR: Portal hypertensive gastropathy. Bailliere's Clinical Gastroenterology, 1992; 6: 481–95
21. Gouge TH, Ranson JH: Esophageal transection and paraesophagogastric devascularization for bleeding esophageal varices. Am J Surg, 1986; 151: 47–54
22. Johnson M, Rajendran S, Balachandar TG et al: Transabdominal modified devascularization procedure with or without esophageal stapler transection – an operation adequate for effective control of a variceal bleed. Is esophageal stapler transection necessary? World J Surg, 2006; 30: 1507–18; discussion 1519
23. Yu W: [Matters of right and wrong in splenectomy for portal hypertension.] Chinese Journal of Hepatobiliary Surgery, 2006; 12: 577–78 [in Chinese]
24. Suisheng X: [Careful selection of splenectomy should be emphasized for cirrhotic portal hypertension.] Chinese Journal of Hepatobiliary Surgery, 2006; 12: 583–84 [in Chinese]
25. Stamou KM, Toutouzas KG, Kekis PB et al: Prospective study of the incidence and risk factors of postsplenectomy thrombosis of the portal, mesenteric, and splenic veins. Arch Surg, 2006; 141: 663–69

Indexed in: [Current Contents/Clinical Medicine] [SCI Expanded] [ISI Alerting System] [ISI Journals Master List] [Index Medicus/MEDLINE] [EMBASE/Excerpta Medica] [Chemical Abstracts/CAS] [Index Copernicus]