Turk J Gastroenterol *2016; 27: 354-60*

Effect of coronary-caval shunt combined with partial pericardial devascularisation on oesophageal and gastric variceal bleeding caused by portal hypertension

LIVER

Original Article

Jigang Bai[1], Meng Xu[1]*, Ruitao Wang[1], Yiping Mu[2], Shunbin Dong[1], Zheng Wu[1], Shengli Wu[1], Chang Liu[1]*

[1]Department of Hepatobiliary Surgery, The First Affiliated Hospital of Xi'an Jiaotong University, Xi'an, China
[2]Department of Medical Record Administration, The First Affiliated Hospital of Xi'an Jiaotong University, Xi'an, China

ABSTRACT

Background/Aims: To investigate the effect of coronary-caval shunt combined with partial pericardial devascularisation on oesophageal and gastric variceal bleeding caused by portal hypertension.

Materials and Methods: Between January 2005 and January 2015, coronary-caval shunt operations combined with partial pericardial devascularisation were performed electively on 15 cirrhotic patients with portal hypertension. All of these patients had a history of oesophageal and gastric variceal bleeding. The clinical and follow-up data of these patients were reviewed retrospectively. Another 15 patients receiving non-surgical treatments in a similar follow-up period were used as controls to compare the preventive effects of different treatment strategies on rebleeding.

Results: All of the 15 surgical procedures were performed successfully, and no severe complications occurred. Among these, autogenous splenic veins were used as bridge vessels in 6 cases, whereas the coronary vein and inferior vena cava were anastomosed directly in 9 cases. All surgical patients were followed up from 5 months to 10 years with an average of 63 months; 2 patients died due to liver failure induced by reactivation of hepatitis B virus and oesophageal/gastric variceal rebleeding, respectively. The rebleeding rates for surgical and non-surgical patients were 6.7% and 66.7% ($p<0.05$), respectively, whereas the 5-year survival rates for the two groups were 85.7% and 33.3% ($p<0.05$), respectively.

Conclusion: Patients with oesophageal and gastric variceal bleeding caused by portal hypertension may benefit from a coronary-caval shunt combined with partial pericardial devascularisation due to decreased coronary vein pressure, unaffected hepatic blood inflow, and reduced incidence of rebleeding.

Keywords: Portal hypertension, cirrhosis, oesophageal and gastric variceal, coronary-caval shunt

INTRODUCTION

Oesophageal and gastric variceal bleeding is the most severe complication of portal hypertension due to cirrhosis; its treatment depends to a great degree on the severity of cirrhosis and the damage to liver function. The mortality of oesophageal and gastric variceal bleeding is quite high. Furthermore, rebleeding readily occurs after the first instance of variceal bleeding, and the mortality of rebleeding is even higher (1,2). Therefore, the main treatment strategy is to control bleeding and prevent rebleeding (2,3). Surgical therapy is an option when pharmacotherapy and endoscopic treatment are less effective. The surgical therapies for oesophageal and gastric variceal bleeding caused by portal hypertension include portal-systemic shunt surgery, gastroesophageal devascularisation and liver transplantation. The curative effects of various operative methods are different, as are the changes in postoperative haemodynamics as well as liver function; further investigation is still needed to determine the optimal surgical procedure for this disease (4-7).

The coronary-caval shunt was first designed by Inokuchi (7) in 1967. In the following years, Inokuchi et al. (7-12) continued to observe and evaluate this method and found that its curative effect was distinct. However, the

Address for Correspondence: Chang Liu, Shengli Wu E-mail: eyrechang@126.com, victorywu@163.com
Received: February 23, 2016 **Accepted:** March 31, 2016
 • DOI: 10.5152/tjg.2016.16086

fact that the wall of the coronary vein is quite thin increased the difficulty of this surgery and prevented its extensive application in clinical practice. In recent years, with the development of surgical techniques, especially blood vessel anastomosis and micro-invasive surgery, extensive use of the coronary-caval shunt became possible. Because there were still potential oesophageal and gastric varices after the placement of a sole coronary-caval shunt, partial pericardial devascularisation was added to further improve the curative effect (12). From 2005 to 2015, we performed coronary-caval shunt procedures combined with partial pericardial devascularisation for 15 patients with portal hypertension, and the results were promising. Reported herein are data on 15 patients with a maximal follow-up of 10 years.

MATERIALS AND METHODS

Ethics statements

All parts of this retrospective study were conducted in accordance with the Declaration of Helsinki of the World Medical Association. This study was approved ethically by The First Affiliated Hospital of Medical College, Xi'an Jiaotong University. All patients provided informed written consent.

Patients

From January 2005 to January 2015, a total of 15 patients underwent coronary-caval shunt procedures combined with partial pericardial devascularisation for portal hypertension due to non-neoplastic liver cirrhosis in our hospital; the patients included 11 males and 4 females with an average age of 43 years (range 29–61). Diagnosis of cirrhosis was based on clinical, laboratory and radiological findings (13). There were 13 cases of posthepatitic cirrhosis, including 10 hepatitis B virus (HBV)-related cirrhosis cases, 2 hepatitis C virus (HCV)-related cirrhosis cases and 1 HBV complicated with HCV-related cirrhosis case, 1 case of alcoholic cirrhosis and 1 case of autoimmune liver cirrhosis. All of these patients had a history of oesophageal and gastric variceal bleeding with manifestations of hematemesis and/or melena; according to the Glasgow-Blatchford scoring system (14), 3 patients were considered to be at high risk and 12 were at low risk. Preoperative endoscopic examination revealed obvious oesophageal and gastric varices (Figure 1). As for liver function grade, Child-Pugh A was found in 7 patients and Child-Pugh B was found in 8 patients. Preoperative colour Doppler ultrasound examination or computed tomography angiography (CTA) scan showed that the diameters of the patients' coronary veins ranged from 0.5 cm to 1.0 cm (Figure 2).

Another 15 patients with oesophageal and gastric variceal bleeding caused by portal hypertension who received non-surgical treatments, including pharmacotherapy and/or endoscopic treatment, in a similar follow-up period were used as controls to compare the preventive effects of different treatment strategies on rebleeding. None of the major clinical parameters were significantly different between the two groups.

Preoperative preparation

Routine laboratory and radiology examinations were performed to evaluate the general conditions, coagulation function, heart, lung and liver functions of the patients. If necessary, corresponding intervention measures were taken to improve the conditions of the patients so that they could safely tolerate the surgery. Specific examinations, including colour Doppler ultrasound examination and CTA scan, were performed to reveal the extent of gastric coronary vein varicose, the diameter and length of the coronary vein and the degree of portal vein dilation as well as the presence of thrombosis and variation.

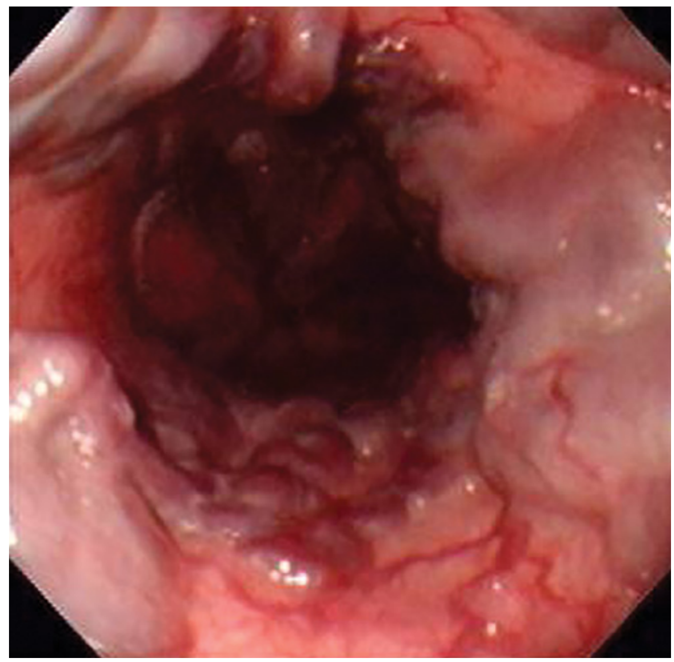

Figure 1. Endoscopic pictures of oesophageal varices before the operation.

Figure 2. Preoperative CTA scan showing the dilation of the coronary vein (black arrow).

The splenic vein, great saphenous vein and internal jugular vein were also evaluated as potential bridge vessels.

Indications for coronary-caval shunt include: liver cirrhosis with portal hypertension; a history of oesophageal and gastric variceal bleeding, for which pharmacotherapy or endoscopic treatment are less effective, or a need to prevent rebleeding; Child-Pugh A or B; gastric coronary vein dilation with a diameter ranging from 0.5 cm to 1.0 cm; a clear anatomic relationship around the inferior vena cava and no obvious inflammatory change. The contraindications include very poor general conditions due to severe heart, lung or kidney dysfunction so that the patient could not tolerate surgery; Child-Pugh C; no obvious gastric coronary vein dilation (diameter less than 0.5 cm) or extreme dilation (diameter more than 1.0 cm) or multiple aneurysmal dilatation and an unclear anatomic relationship around the inferior vena cava or inflammatory change.

Operative technique

The coronary-caval shunt procedure was performed according to the technique described by Inokuchi (7). Briefly, a midline abdominal incision from the xyphoid to approximately 5 cm below the navel was performed with the patient in the supine position. At first, catheters were inserted into the right gastro-omental vein and veins along the lesser curvature to measure the portal vein and gastric coronary vein pressure, respectively. Then, the gastric coronary vein was explored to realize the open position, single or double branch, degree of dilation and tortuosity. If the diameter of the gastric coronary vein ranged from 0.5 cm to 1.0 cm and there was no obvious variation and tortuosity, a coronary-caval shunt procedure could be performed.

Splenectomy was performed first. The spleen pedicle was cut off as close to the splenic hilum as possible to retain a sufficient length of portal vein for later use (Figure 3a). The splenic vein was freed for 5–7 cm, and all its tributaries were ligated on the superior border of the pancreas from the tail of the pancreas to its head. The isolated splenic vein was preserved in saline for later use. The gastric coronary vein was dissected and divided about 0.5 cm away from the portal vein. The distal end was double ligated, and the proximal end was freed as close to the gastric wall as possible (Figure 3b). The left and right gastroepiploic artery and vein, left gastric artery, right gastric vein, posterior gastric vein and gastric short vein were freed and ligated. The right gastric artery was retained. The anterior medial wall of the inferior vena cava was exposed close to the omental foramen above the renal vein. The anterior wall of the inferior vena cava was clamped with a non-invasive vascular clamp for anastomosis (Figure 3c).

If the coronary vein was long enough, it could be anastomosed directly to the inferior vena cava (Figure 3d). If a bridge vessel was needed, end-to-side anastomosis of the isolated splenic vein and the inferior vena cava would be performed first. The anastomotic stoma was performed as close to the left wall of the inferior vena cava as possible to avoid sharp angulation of the bridge vessel as it entered the inferior vena cava. The bridge vessel was taken through the omental foramen to the broken end of the coronary vein, and end-to-end anastomosis of the isolated splenic vein and coronary vein was performed. For both anastomoses, a continuous eversion suture technique was adopted with 6-0 Prolene. The vessels were flushed with heparin saline while the anastomosis was performed. Before the vessel clamp was removed, some blood was released from the anastomotic stoma to expel possible air and thrombus inside. The portal vein and gastric coronary vein pressure were measured again immediately after the shunt operation.

Postoperative management

Expect for regular electrocardiogram monitoring, nutrition support and medications protecting liver function, specific attention should be paid to the following: 1) Maintain stable blood pressure. 2) Monitor bleeding time, clotting time and platelet count to prevent thrombogenesis. When safe, patients should receive intravenous or subcutaneous injections of low molecular weight heparin, 5000–7500 U per 12 h, for 5 days followed by oral therapy with warfarin for 3–6 months to maintain the target prothrombin time/international normalized ratio (PT/INR) at a level between 1.5 and 2.0. 3) Monitor liver function, including the levels of transaminase enzymes, cholesterol, proteins and jaundice index. 4) Perform colour Doppler ultrasound examination or CTA scan regularly to observe the blood flow and thrombogenesis in the shunt vessels. 5) Perform gastroscopy examination regularly to monitor the extent of oesophageal and gastric varices. 6) Monitor the rebleeding rate of varices and the incidence of hepatic encephalopathy.

Statistical analysis

The Statistical Package for the Social Sciences (SPSS) 11.5 statistical software (SPSS Inc.; Chicago, IL, USA) was used for statistical analyses. Continuous data were presented as mean±standard deviation (SD) and compared with the two-tailed non-paired Student's t-test. Categorical data were presented as frequencies and analysed with the Chi-square or Fisher exact test. A p value <0.05 was considered statistically significant.

RESULTS

Surgical results

All of the 15 surgical procedures were performed successfully, and no operation-related death occurred. Among these, autogenous splenic veins were used as bridge vessels in 6 cases, whereas the coronary vein and inferior vena cava were anastomosed directly in 9 cases. No severe postoperative complications, such as hepatic failure or hepatic encephalopathy, occurred. White blood cell and platelet count significantly increased postoperatively (Table 1). All the patients recovered satisfactorily with a mean hospital stay of 11 days.

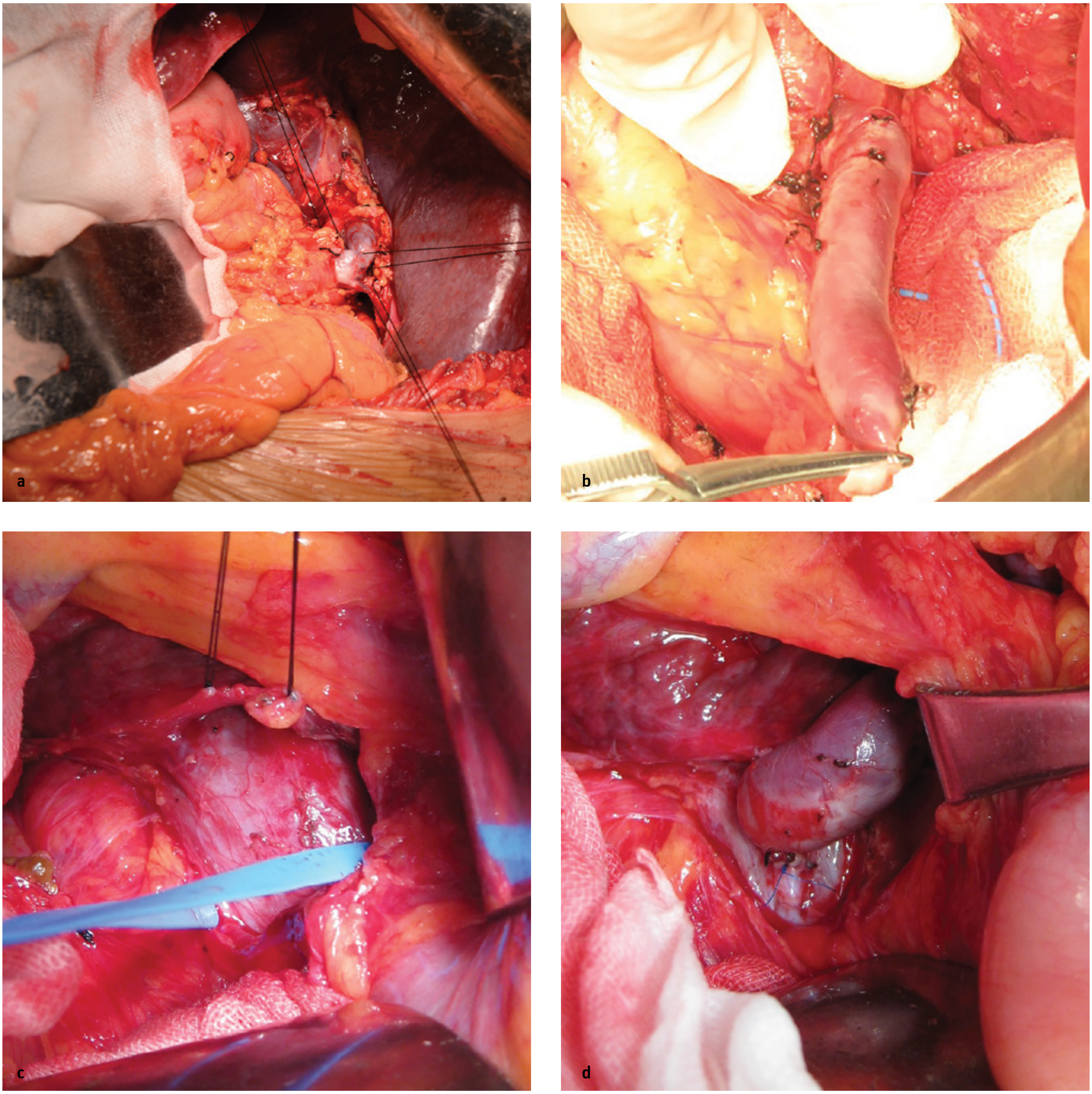

Figure 3. a-d. Pattern of coronary-caval shunt. Cut off the spleen pedicle close to the splenic hilum (a); free gastric coronary vein as long as possible (b); expose the anterior medial wall of the inferior vena cava (c); coronary vein (or antilogous blood veins) and inferior vena cava end-to-side anastomosis (d)

Hemodynamic changes

The preshunt average portal vein and gastric coronary vein pressures were 37.73±5.38 cm H_2O and 33.16±4.36 cm H_2O, respectively, while those after the shunt operation were 35.61±4.70 cm H_2O and 15.26±3.91 cm H_2O, respectively (Figure 4). The gastric coronary vein pressure after the shunt operation was obviously lower than that before the operation ($p<0.01$), while the pressure of the portal vein changed only slightly before and after the shunt operation ($p>0.05$). Pulsed Doppler ultrasound examination showed that the preshunt average portal vein flow was 741±213 mL/min, while that after the shunt procedure was 690±187 mL/min; no statistical difference was found between them ($p>0.05$).

Follow-up results

All surgical patients were followed up regularly from 5 months to 10 years, with an average of 63 months. Thrombosis was found in the shunt vessel on the 4th day after operation in one patient and disappeared after timely thrombolytic therapy. Two patients died during the follow-up. One death was due to liver failure induced by reactivation of hepatitis B virus in the 37th month after operation, and the other was due to oesopha-

Table 1 . Laboratory and imaging data of the 15 patients before and after the operation

	Preoperative 24 h	Postoperative 24 h	Discharge
RBC ($\times 10^{12}$/L)	3.21 (1.63)	3.51 (1.72)	4.12 (1.80)
WBC ($\times 10^{9}$/L)	2.54 (1.33)	7.36 (3.24)	6.68 (3.01)*
PLT ($\times 10^{9}$/L)	53.3 (31.2)	75.8 (39.3)	329.3 (113.2)*
ALT (U/L)	26.6 (11.2)	35.2 (7.7)	22.8 (6.3)
AST (U/L)	19.9 (8.3)	34.6 (12.4)	26.5 (10.1)
TP (g/L)	65.6 (13.2)	63.2 (15.5)	78/6 (17.4)
ALB (g/L)	31.3 (5.1)	30.3 (4.3)	37.0 (6.5)
TBIL (μmol/L)	24.2 (10.0)	27.3 (8.9)	19.8 (11.4)
CHOL (mmol/L)	3.2 (1.7)	2.7 (2.0)	3.9 (2.1)
NH3 (μmol/L)	24.7 (11.2)	32.6 (13.6)	15.1 (16.5)
Diameter of PV (mm)	15.2 (2.3)	14.3 (2.1)	13.6 (2.0)
Flow of PV (mL/min)	741 (213)	702 (190)	667 (230)

Data are expressed as mean±SD.
*p<0.05 vs. preoperative 24 h.
RBC: red blood cell; WBC: white blood cell; PLT: platelets; ALT: alanine aminotransferase; AST: aspartate aminotransferase; TP: total protein; ALB: serum albumin; TBIL: total bilirubin; CHOL: total cholesterol; NH3: blood ammonia; PV: portal vein

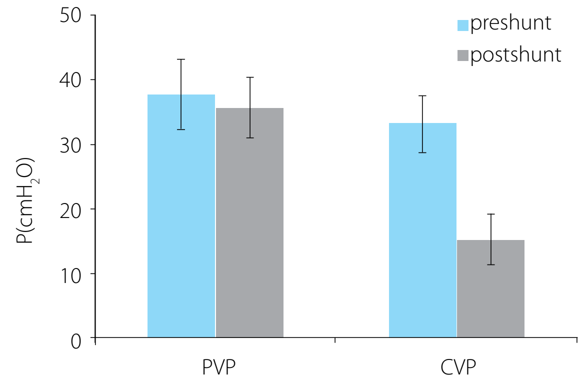

Figure 4. Changes in the portal vein pressure (PVP) and coronary vein pressure (CVP) before and after the shunt

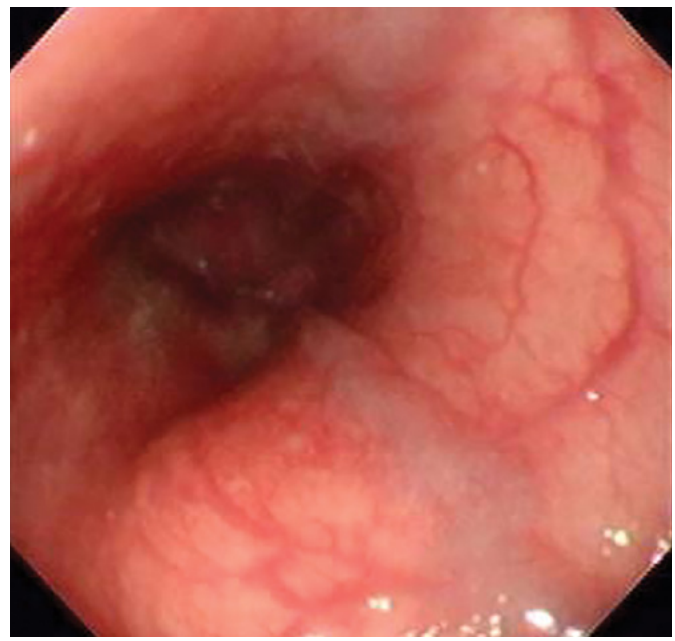

Figure 5. Endoscopic pictures of oesophageal varices 3 months after operation.

Figure 6. Postoperative CTA scan showing the patency of the shunt vessel (white arrow)

geal and gastric variceal rebleeding in the 63rd month after operation. The rebleeding rate was 6.7% and the five-year survival rate was 85.7%. No long-term complications occurred for the remaining 13 patients, and hypersplenism was rectified completely (Table 1). Gastroscopy re-examination three months after operation revealed that the oesophageal and gastric varices had almost disappeared (Figure 5). Portal hypertension gastric disease was greatly relieved. Colour Doppler ultrasound examination or CTA scan showed that the patency rate of the shunt vessels was 100% (Figure 6).

For the non-surgical group, the rebleeding rate was 66.7% (10/15), which was significantly higher than that in the surgical group ($p<0.05$). In line with this, the 5-year survival rate of the non-surgical group was obviously decreased compared to that of the surgical group (33.3% vs. 85.7%, $p<0.05$).

DISCUSSION

Pharmacotherapy combined with endoscopic therapy has been regarded as the first-line treatment of oesophageal and gastric variceal bleeding. However, when these treatments are less effective, surgery is the best option (1-3). Currently, the surgical strategy for oesophageal and gastric variceal bleeding includes devascularisation and shunt operations (15). Devascularisation is an effective way to control bleeding and assure blood supply to the liver; however, this surgery may lead to increased postoperative portal pressure and induce portal hypertensive gastropathy and rebleeding (16). Therefore, it has been almost abandoned in all countries except for China. There are three types of shunting procedures for the treatment of oesophageal and gastric variceal bleeding: total shunt, limited shunt and selective shunt (17). Total shunt and limited shunt operations could relieve portal hypertension effectively. However, these operations decreased hepatic blood inflow, aggravated liver damage and could induce hepatic encephalopathy (18). Selective shunts include distal splenorenal shunt, splenocaval shunt and coronary-caval shunt (19). Among these, coronary-caval shunt is theoretically considered to be the best.

The portal vein blood flow can essentially be divided into two basins, namely the spleen and stomach area and the mesenteric blood flow area. There are 'barriers' between the two areas. Blood flow from the portal vein via the coronary vein to the systemic circulation is the anatomical basis of oesophageal and gastric varices caused by portal hypertension (7). Thus, coronary-caval shunt has the following advantages: 1) It effectively relieves the pressure in oesophageal and gastric varices. Inokuchi (7) reported that the pressure of the coronary vein decreased from 14 to 39 cm H_2O to 14 to 20 cm H_2O after the shunt operation. In this study, the preshunt average gastric coronary vein pressure was 33.16±4.36 cm H_2O, while that after the shunt procedure was 15.26±3.91 cm H_2O. The drop was 16.82±3.16 cm H_2O. Gastroscopy re-examination 3 months after the operation revealed that the oesophageal and gastric varices almost disappeared. 2) The communication between the coronary vein and the portal or splenic vein was disconnected, and the branches from the portal or splenic vein to the oesophagus and the bottom of the stomach were ligated, which played a partial devascularisation role. 3) Venous pressure in the spleen and stomach area decreased after the shunt operation, which can relieve portal hypertensive gastropathy. 4) This surgical method had little effect on portal vein flow and therefore could reduce the incidence of hepatic encephalopathy. Inokuchi et al. (10) reported that the preshunt average portal vein flow was 713±193 mL/min, while that after the shunt was 756 mL/min; the shunt had little effect on the portal vein flow. In agreement with the report by Inokuchi et al. (10), our results showed that the preshunt average portal vein pressure was 37.73 5.38 cm H_2O, while that after the shunt operation was 35.61±4.70 cm H_2O; the drop was only 2.23±1.52 cm H_2O. Additionally, the preshunt average portal vein flow was 741±213 mL/min, while that after the shunt operation was 690±187 mL/min. No statistical difference was found between pre- and post-shunt operation for either portal pressure or portal flow.

The coronary vein can communicate with the vena cava via a bridge vessel. The bridge vessel can be an artificial blood vessel or autogenous vessels such as the splenic vein, great saphenous vein or internal jugular vein (10-12). With the development of surgical techniques, the extensive use of coronary-caval shunts became possible. If the coronary vein was sufficiently long, it could be anastomosed directly to the inferior vena cava. In this study, the coronary vein and inferior vena cava were anastomosed directly in 9 cases, while autogenous splenic veins were used as bridge vessels in the other 6 cases. To prevent postoperative thrombosis in the shunt vessel, patients should receive prophylatic anticoagulants early as long as it is safe. If thrombosis is found, thrombolytic therapy should be given in a timely fashion. All of the 15 surgical patients in this study received intravenous or subcutaneous injection of low molecular weight heparin, 5000 to 7500 U per 12 h, from post-operative day (POD) 1 to POD 5, followed by oral warfarin therapy for 3 to 6 months to keep the target PT/INR at a level between 1.5 and 2.0. Thrombosis was found in the shunt vessel on POD 4 in only one patient; the thrombosis disappeared after timely thrombolytic therapy.

Potential oesophageal and gastric varices may remain after a coronary-caval shunt operation, which may be related to the limited shunt volume caused by small anastomotic stoma or the reflux of portal vein flow via the right gastro-omental vein or posterior gastric vein. Saeki et al. (20) reported a female patient with sudden haematemesis and melena 17 years after a coronary-caval shunt operation. Gastroscopic examination revealed aneurysmal dilated varicose veins that were bleeding on both the anterior and posterior walls of the stomach body. Percutaneous hepatic portal vein angiography revealed a stealing of the portal blood flow to the inferior vena cava through both a dilated right gastroepiploic vein and a right gastric vein via the left gastric venous caval shunt. In this study, during follow-up, only one incident of rebleeding occurred and the rebleeding rate was 6.7%, which was significantly lower than that in the non-surgical group. Compared with the shunt operation alone, coronary-caval shunt combined with partial devascularisation and splenectomy may increase the risks for patients. To guarantee patient safety, we established a series of preoperative risk assessment criteria, such as a preoperative liver assessment (POLA) checklist (21), to mitigate perioperative complications for these patients. In this study, all of the 15 surgical procedures were performed successfully, and no operation-related death or severe postoperative complications such as hepatic failure or hepatic encephalopathy occurred. No postoperative portal vein system thrombosis was found in this study, and we speculate that the reason might be the early regular use of anticoagulants for preventing thrombosis in the shunt vessels. Therefore, we concluded that coronary-caval shunt combined with partial devascularisation could be a practicable treatment for patients with oesophageal and gastric varices to reduce the rebleeding rate and rebleeding-related mortality. However, due to the limited number of cases and the short follow-up period for some cases, the long term results of this surgical treatment need further investigation.

In general, each of the surgical strategies for cirrhotic portal hypertension has its own characteristics, and adopting personalized treatment programs to achieve the best curative effect will be the trend in the future (22,23). Coronary-caval shunt combined with partial devascularisation could decrease pressure in the coronary vein and relieve oesophageal and gastric varices effectively. At the same time, this surgical technique has little effect on blood flow in the mesenteric area and can assure portal pressure and hepatic blood inflow, thus preserving liver function well. Theoretically, this is the best shunt operation for patients with severe oesophageal and gastric varices who have poor liver function and who are not suitable for or unwilling to undergo liver transplantation.

Ethics Committee Approval: Ethics committee approval was received for this study from the ethics committee of Xi'an Jiaotong University.

Informed Consent: Written informed consent was obtained from patients who participated in this study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept - C.L.; Design - J.B., C.L.; Supervision - S.W., C.L.; Funding - C.L.; Materials - M.X., R.W.; Data Collection and/or Processing - Y.M., S.D.; Analysis and/or Interpretation - Z.W., S.W.; Literature Review - J.B., C.L.; Writer - J.B., M.X.; Critical Review - C.L.

*JB and MX contributed equally to this paper.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study has received financial support from National Natural Science Foundation of China (No. 81101872).

REFERENCES

1. Chen YI, Ghali P. Prevention and management of gastroesophageal varices in cirrhosis. Int J Hepatol 2012; 2012: 750150.
2. Garcia-Tsao G, Bosch J. Management of varices and variceal hemorrhage in cirrhosis. N Engl J Med 2010; 362: 823-32. [CrossRef]
3. Coelho FF, Perini MV, Kruger JA, et al. Management of variceal hemorrhage: current concepts. Arq Bras Cir Dig 2014; 27: 138-44. [CrossRef]
4. Huang Y, Wang W, Wang J, Bai C. Surgical treatment of portal hypertension: 45 year experience. Zhonghua Wai Ke Za Zhi 2000; 38: 85-8.
5. Orloff MJ. Fifty-three years' experience with randomized clinical trials of emergency portacaval shunt for bleeding esophageal varices in Cirrhosis: 1958-2011. JAMA Surg 2014; 149: 155-69. [CrossRef]
6. Lo GH. The use of transjugular intrahepatic portosystemic stent shunt (TIPS) in the management of portal hypertensive bleeding. J Chin Med Assoc 2014; 77: 395-402. [CrossRef]
7. Inokuchi K. A selective portacaval shunt. Lancet 1968; 2: 51-2. [CrossRef]
8. Inokuchi K, Kobayashi M, Kusaba A, Ogawa Y, Saku M, Shiizaki T. New selective decompression of esophageal varices. By a left gastric venous-caval shunt. Arch Surg 1970; 100: 157-62. [CrossRef]
9. Inokuchi K, Kobayashi M, Ogawa Y, Saku M, Nagasue N. Results of left gastric vena caval shunt for esophageal varices: Analysis of one hundred clinical cases. Surgery 1975; 78: 628-36.
10. Inokuchi K, Beppu K, Koyanagi N, et al. Fifteen years' experience with left gastric venous caval shunt for esophageal varices. World J Surg 1984; 8: 716-21. [CrossRef]
11. Inokuchi K, Sugimachi K. The selective shunt for variceal bleeding: a personal perspective. Am J Surg 1990; 160: 48-53. [CrossRef]
12. Sato Y, Hatakeyama K. Left gastric venous caval direct shunt in esophagogastric varices. Hepatogastroenterology 2002; 49: 1251-2.
13. Schuppan D, Afdhal NH. Liver cirrhosis. Lancet 2008; 371: 838-51. [CrossRef]
14. Bozkurt S, Köse A, Arslan ED, et al. Validity of modified early warning, Glasgow Blatchford, and pre-endoscopic Rockall scores in predicting prognosis of patients presenting to emergency department with upper gastrointestinal bleeding. Scand J Trauma Resusc Emerg Med 2015; 23: 109. [CrossRef]
15. Qiu FZ. Evaluation of the pericardial devascularization in portal hypertension. Zhonghua Wai Ke Za Zhi 1983; 21: 275-7.
16. Sarfeh IJ, Rypins EB, Mason GR. A systematic appraisal of portacaval H-graft diameters. Clinical and hemodynamic perspectives. Ann Surg 1986; 204: 356-63. [CrossRef]
17. Stipa S, Balducci G, Ziparo V, Stipa F, Lucandri G. Total shunting and elective management of variceal bleeding. World J Surg 1994; 18: 200-4. [CrossRef]
18. Collins JC, Rypins EB, Sarfeh IJ. Narrow-diameter portacaval shunts for management of variceal bleeding. World J Surg 1994; 18: 211-5. [CrossRef]
19. Henderson JM. The distal splenorenal shunt. Surg Clin North Am 1990; 70: 405-23.
20. Saeki H, Hashizume M, Yanaga K, et al. Giant gastric varices after a left gastric venous caval shunt operation for esophageal varices: a case report. Hepatogastroenterology 1998; 45: 700-4.
21. Im GY, Lubezky N, Facciuto ME, Schiano TD. Surgery in patients with portal hypertension: a preoperative checklist and strategies for attenuating risk. Clin Liver Dis 2014; 18: 477-505. [CrossRef]
22. Tripathi D, Helmy A, Macbeth K, et al. Ten years' follow-up of 472 patients following transjugular intrahepatic portosystemic stent-shunt insertion at a single centre. Eur J Gastroenterol Hepatol 2004; 16: 9-18. [CrossRef]
23. Sauerbruch T, Trebicka J. Future therapy of portal hypertension in liver cirrhosis - a guess. F1000Prime Rep 2014; 6: 95. [CrossRef]