

Observation on the Effect of Interventional Devascularization in the Treatment of Liver Cirrhosis with Portal Hypertension

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Abstract: *Objective:* To analyze the clinical effect of interventional devascularization in the treatment of liver cirrhosis with portal hypertension. *Methods:* 80 patients with liver cirrhosis and portal hypertension admitted between January 2020 and January 2023 were selected as research subjects. They were divided into a control group (surgical devascularization) and an experimental group (interventional devascularization) through the computer grouping method, and the effect of the treatment received by both groups were compared. *Results:* (i) The efficacy of the treatment received in the experimental group was 94.87%, which was significantly higher than that of the control group, which was 76.92% ($P < 0.05$). (ii) There was no difference in the levels of alanine transaminase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP) between the control group and the experimental group before treatment ($P > 0.05$); after treatment, the levels of ALT, AST, and ALP in the experimental group were statistically significantly lower than those in the control group ($P < 0.05$). (iii) Compared with the total complication rate of 28.21% in the control group, the total complication rate of the experimental group was lower at 10.25%, and the statistical significance was established ($P < 0.05$). *Conclusion:* Interventional devascularization has demonstrated positive outcomes in treating liver cirrhosis and portal hypertension. This is evident in the enhancement of liver function and its high safety profile. Consequently, it merits wider adoption and utilization in clinical practice.

Keywords: Interventional devascularization; Liver cirrhosis and portal hypertension; Liver function

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1. Introduction

When liver cirrhosis develops to a certain degree, a series of syndromes such as portal hypertension, hypersplenism, and ascites will occur due to establishment and opening of collateral circulation^[1]. The establishment of collateral circulation usually occurs at the esophagus and gastric fundus, and the main clinical manifestation is varicose veins. Usually, portal hypertension is accompanied by upper gastrointestinal bleeding^[2]. When the portal pressure increases, the collateral circulation between the portal vein and the vena cava expands. The treatment methods for cirrhotic portal hypertension include drugs, surgery, etc. The effect of surgical treatment is more ideal compared to conservative treatment,^[3]. In this study, the efficacy of interventional devascularization in the treatment of cirrhotic portal hypertension was analyzed.

2. Clinical information and methods

2.1. Clinical information

A total of 80 patients with liver cirrhosis and portal hypertension treated between January 2020 and January 2023 were selected as research subjects. The patients were divided into a control group (surgical devascularization) and experimental group by the computer grouping method. Inclusion criteria: (i) diagnosed with liver cirrhosis by laboratory diagnosis, (ii) signed an informed consent, (iii) patency of the main portal vein. Exclusion criteria: (i) gastrointestinal hemorrhage on admission, (ii) infection in other body parts, (iii) has other serious malignant diseases, (iv) history of surgery. In the control group, there were 20 male and 19 female patients, with ages ranging from 33 to 65 years and an average age of 49.00 ± 4.66 years. In the experimental group, there were 20 male and 19 female patients, with ages ranging from 33 to 63 years and an average age of 48.00 ± 4.63 years. Statistical software was employed to compare the gender and age between the two groups of patients, revealing no significant differences ($P > 0.05$), indicating their comparability.

2.2. Methods

The control group was treated by surgical devascularization: the whole spleen was removed, and the blood vessels around the cardia and gastric fundus were blocked. Routine ligation of the gastric tubular vein branch was performed at 6 locations away from the lower end of the esophagus, and the high esophageal branch was cut off at the same time. Antibiotics and hepatoprotective drugs were routinely administered after surgery.

The experimental group was treated by interventional devascularization: the patient was placed in the supine position on the angiography table, and the 21G Chiba needle was used to puncture the branch of the intrahepatic portal vein or the branch of the intrasplenic splenic vein through ultrasound guidance. The guide wire was inserted, and the puncture tract was properly expanded along the guide wire. Then, the catheter sheath was introduced into the portal vein. The blood pressure of the patient was then measured. A catheter was placed at the splenic hilum at the beginning of the splenic vein and the main trunk of the superior mesenteric vein, and digital subtraction angiography (DSA) was performed to observe the status of the portal vein and its collaterals. The femoral artery was punctured by the Seldinger technique, and the catheter was placed in the main trunk of the splenic artery near the splenic hilum, and splenic arteriography was performed. A suspension of contrast medium, gelatin sponge particles, and antibiotics were injected into the main trunk of the splenic artery through fluoroscopy until the blood flow of the splenic artery disappears. The embolism area was controlled between 50–80%. Angiography was performed again, and the embolism area was recorded. A guide was inserted into the proximal main trunk of the portal vein collateral vessel, and then DSA was performed to determine the direction and velocity of blood flow, followed by embolization treatment. If the blood flow velocity was high and the varicose veins had thickened with obvious branches, 5% morrhuate sodium or 99% absolute alcohol was injected through the catheter. Simultaneously, a spring steel ring was placed to embolize the proximal main trunk of the side branch vessels. After about 3 minutes, a contrast agent was injected to determine the degree of embolism until the distal end of the embolized varicose vein is no longer visible. Next, a catheter was inserted into the vein, and the portography was re-examined, and the free portal vein pressure was measured again after the intervention was cut off. The puncture channel was blocked by a spring steel coil or gelatin sponge, and the bleeding was stopped by extubation. Anti-infection, fluid replacement and other symptomatic treatment were routinely performed postoperatively.

2.3. Efficacy and observation indicators

2.3.1. Evaluation of efficacy

(i) Cured: symptoms and signs of the disease disappeared, imaging results were normal, and no varicose veins

were found by endoscopic examination. (ii) Markedly effective: symptoms and signs of the disease disappeared, imaging examinations indicated improvements, and no varicose veins were found during endoscopic examination. (iii) Effective: the signs and symptoms of the disease improved, but varicose veins were still visible through endoscopy. (iv) Ineffective: disease symptoms, signs, and imaging results only improved slightly or worsened, and endoscopic results showed obvious varicose veins ^[4,5].

2.3.2. Observation indicators

(1) Liver function indicators: 3 mL of venous blood was drawn in the morning on an empty stomach, centrifuged for 10 minutes, and the upper layer was extracted for testing. The levels of glutamic acid aminotransferase (ALT), aspartate aminotransferase (AST) and alkaline phosphatase (ALP) were detected by an automatic biochemical analyzer. (2) Complications: fever, rebleeding, infection of the surgical site, and lung infection.

2.4. Statistical analysis

Statistical analysis was performed using SPSS 22.0. data related to efficacy and rate of complications of both groups were compared using a χ^2 test, while the liver functions were compared using a t-test. $P < 0.05$ indicated statistical significance.

3. Results

3.1. Treatment efficacy

The treatment received in the experimental group demonstrated significantly higher total efficacy at 94.87% compared to the control group, which achieved 76.92% efficacy ($P < 0.005$). See **Table 1** for further details.

Table 1. compares the clinical curative effect of different groups of patients [n (%)]

Group	Cured	Markedly effective	Effective	Ineffective	Total effective rate
Control group	11 (28.20)	9 (23.08)	10 (25.64)	9 (23.08)	30 (76.9)
Experimental group	21 (53.84)	10 (25.64)	6 (15.39)	2 (5.13)	37 (94.87)
χ^2					5.185
P					< 0.05

3.2. Changes in liver function

Before treatment, there was no difference in the ALT, AST, and ALP levels between both groups of patients ($P > 0.05$). After treatment, the ALT, AST, and ALP levels in the experimental group were significantly lower than those in the control group ($P < 0.005$). See **Table 2** for further details.

Table 2. Comparison of changes in liver function levels in different groups of patients (mean \pm standard deviation, U/L)

Group	ALT		AST		ALP	
	Before treatment	After treatment	Before treatment	After treatment	Before treatment	After treatment
Control group	60.65 \pm 5.39	42.30 \pm 3.62	62.43 \pm 5.57	44.55 \pm 3.95	135.60 \pm 9.77	72.06 \pm 6.46
Experimental group	60.33 \pm 5.36	34.52 \pm 4.55	62.46 \pm 5.60	36.41 \pm 4.80	135.63 \pm 9.81	54.60 \pm 5.01
t	0.263	8.356	0.024	8.178	0.014	13.338
P	> 0.05	< 0.05	> 0.05	< 0.05	> 0.05	< 0.05

3.3. Complication rate

The experimental group recorded a significantly lower total complication rate of 10.25% compared to the control group, where the total complication rate stood at 28.21% ($P < 0.005$). Refer to **Table 3** for details.

Table 3. Comparison of total complication rates among patients in each group [n (%)]

Group	Fever	Rebleeding	Incision infection	Lung infection	Total
Control group	4 (10.26)	2 (5.13)	3 (7.69)	2 (5.13)	11 (28.21)
Experimental group	2 (5.13)	0 (0.00)	1 (2.56)	1 (2.56)	4 (10.25)
χ^2					4.044
P					< 0.05

4. Discussion

Cirrhotic portal hypertension is relatively difficult to treat, and the disease manifests as splenomegaly, portal vein collateral circulation, etc., accompanied by spontaneous peritonitis, gastrointestinal bleeding, and other symptoms [6,7]. The cause of portal hypertension remains not well-understood, but it is generally associated with increased portal vein resistance (“backward-flow” theory) and increased portal vein blood flow (“forward-flow” theory) [8,9]. The esophageal and gastric varices formed by the opening of portal vein collaterals are the main cause of death due to rupture and bleeding. Studies show that most patients with liver cirrhosis and portal hypertension are accompanied by symptoms of upper gastrointestinal bleeding, and the mortality rate is as high as 58%. The key to the treatment of portal hypertension in liver cirrhosis is to control bleeding and eliminate hypersplenism [10,11].

In this study, the effects of surgical devascularization (control group) and interventional devascularization (experimental group) in the treatment of liver cirrhosis and portal hypertension were compared. Better improvement was seen in the functional indicators of the patients in the experimental compared to the control group, and complication rate of the experimental group was also lower than that of the control group. Therefore, it is clear that interventional devascularization is more effective and safer than surgical devascularization in treating cirrhotic portal hypertension. The treatment methods for cirrhotic portal hypertension include drug therapy, surgery, endoscopic treatment, etc., which are all effective but each comes with certain shortcomings [12,13]. Interventional devascularization is superior over other treatment methods because medication is less effective, and surgical intervention has a higher mortality rate, and liver transplantation a very demanding procedure. Partial splenic embolization can achieve good results by effectively inhibiting hypersplenism and reducing the size of an overly large spleen, so as to improve immune hemocytopenia and correct hypersplenism. Previously, it was believed that the portal vein blood flow will not change after splenectomy or devascularization, and the blood flow may increase [14]. However, some studies showed that the portal vein blood flow decreased significantly after splenectomy or devascularization, which was mainly due to the increase of portal vein blood flow caused by the splenic vein [15]. The selection of the most appropriate portal vein branch for the procedure is crucial. Achieving super-selective intubation for all varicose veins is essential, and employing a slow and intermittent injection of embolic agents helps prevent embolic agent reflux and shunts from entering the vein. The combined use of spring steel ring, gelatin sponge, and absolute ethanol can enhance the embolization effect. Precise positioning by DSA can reduce the occurrence of postoperative complications and reduce the risk of rebleeding. In interventional devascularization, the risk of rebleeding is significantly reduced as it ensures comprehensive coverage of the main portal vein and ectopic collaterals. The procedure involves direct portal vein angiography during which collateral veins are

fully exposed, eliminating the possibility of missing any critical issues.

5. Conclusion

In conclusion, interventional devascularization is effective in treating cirrhotic portal hypertension, which is reflected in the improvement of liver function and high safety, and it is worthy of promotion and application.

Disclosure statement

The authors declare no conflict of interest.

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