



PORTAL VENOUS STENTING FOR PORTAL VEIN ANASTOMOTIC STENOSIS AFTER LIVER TRANSPLANTATION: A CASE REPORT

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ABSTRACT

Liver transplantation (LT) is currently one of the most crucial methods for treating end-stage liver disease. The survival rate after transplantation has exceeded 70-80%. Vascular complications after LT are a significant cause of postoperative failure. Therefore, early diagnosis and proactive treatment of vascular complications after LT can lead to a more comprehensive recovery for the patient. This case involves a 55-year-old female patient with liver cirrhosis. Three days after the right lobe of the liver was transplanted, she was found to have elevated blood pressure, increased ascites, and deteriorating liver function. Doppler ultrasound revealed insufficient blood supply to the portal vein (PV), while computed tomography (CT) diagnosed stenosis at the PV anastomosis, resulting in partial thrombosis in the hepatic artery and hepatic vein. The self-expanding stent (Bard® E-LUMINEXX™) was deployed using angiography to widen the vascular anastomotic stenosis. The portal venogram after the stent placement revealed that the stent was well positioned and open, leading to improved PV flow and a significant reduction in PV pressure. Reduce pulse pressure and improve arterial blood flow.

KEYWORDS: Liver Transplantation, Angiography, Liver Cirrhosis, LDLT.

INTRODUCTION

Liver cirrhosis is the result of widespread inflammation and fibrosis in the liver, leading to the formation of nodules. This condition impairs normal liver function, causing obstruction of portal blood flow and an increase in portal pressure. [1] Liver cirrhosis is a chronic condition that impacts the entire body. Common causes include viral hepatitis B and C, alcohol-related damage, and injury from hepatotoxic drugs or toxins. The liver can be considered the organ with the most robust regenerative capacity in the human body, and most diseases can gradually improve over time. However, if the liver is damaged beyond its capacity to recover for an extended period, fibrous tissue may develop. This process is called liver fibrosis, and if the liver continues to undergo fibrosis, it will progress to cirrhosis. [2-4] Cirrhosis is

also a significant risk factor for developing liver cancer. [5] In recent years, liver transplant surgery has become increasingly popular, and the success rate has also significantly improved.

After receiving a liver transplant, patients must overcome numerous challenges, particularly potential complications, and resume their normal lives as soon as possible. Common complications include intraoperative or postoperative intra-abdominal bleeding, risk of infection, and autologous rejection. Other potential issues include stenosis or infarction of bile ducts or vascular anastomoses. [6-8]

In addition to the bile duct, the blood vessels that need to be connected during liver transplantation (LT) include the hepatic artery, hepatic vein, and portal vein.

These blood vessels are undergoing self-repair after surgery, so there is a possibility of thrombosis. [9]

If necessary, drug treatment or surgery will be required to remove the thrombus.

Portal vein thrombosis (PVT) and stenosis are common complications following LT, with incidence rates of 1.0% to 2.0% and 0.6% to 3.0%, respectively. [10-12] The diagnosis of portal vein (PV) complications is typically performed through postoperative Doppler ultrasound as a routine examination. [13] Once insufficient or abnormal blood flow is detected, computed tomography angiography (CTA) is performed to confirm the diagnosis. Vascular anastomosis can be visualized using CTA reconstruction. The presence of stenosis, the extent of thrombosis, and the affected area need to be determined. After confirming the diagnosis, it is essential to assess the recipient's liver function and vital signs to determine their stability before deciding on surgical treatment or interventional examination and treatment via angiography. Angiography usually involves directly puncturing the hepatic vein or the PV through the superior mesenteric artery, or percutaneous transhepatic puncture with ultrasound guidance. This enables the catheter to navigate the vascular anastomotic stenosis using the guide wire for stent placement to relieve the stenosis. Enhance the blood flow to promote the recovery of liver function. [14]

This case involves a patient who underwent a living donor liver transplantation (LDLT). The ultrasound

revealed a decrease in the PV blood flow rate. Computerized tomography (CT) confirmed that the surgical connection of the PV was too narrow, leading to inadequate blood flow and thrombosis. Treated by inserting intravascular stents.

CASE PRESENTATION

A 55-year-old woman underwent LDLT in March due to severe liver cirrhosis resulting from long-term alcoholism and chronic hepatitis C virus (HCV). CT for preoperative evaluation of transplantation recipients. This patient has portal vein type A. [15] The anatomy is normal, with a single right portal vein (RPV) and a single left portal vein (LPV) originating from the main PV. The diameter of the main portal vein is 9.8 mm. No obvious thrombosis was found in the PV, splenic vein, and upper mesenteric vein.

The patient was admitted to the hospital on May 18 and underwent a series of preoperative evaluations and examinations. The patient is scheduled to undergo LDLT on May 20. During the surgery, the PV was severely adhered to the CBD, resulting in damage to the PV. This necessitated a longitudinal incision and transverse sutures for repair during the operation. During the PV anastomosis, a mild stenosis was found, as shown in Figure 1, but the PV flow remained at 40-50 cm/s, and the outflow was also within the acceptable range for segment 5 (V5) and the right hepatic vein at about 15-25 cm/s. Liver flow was closely monitored postoperatively.

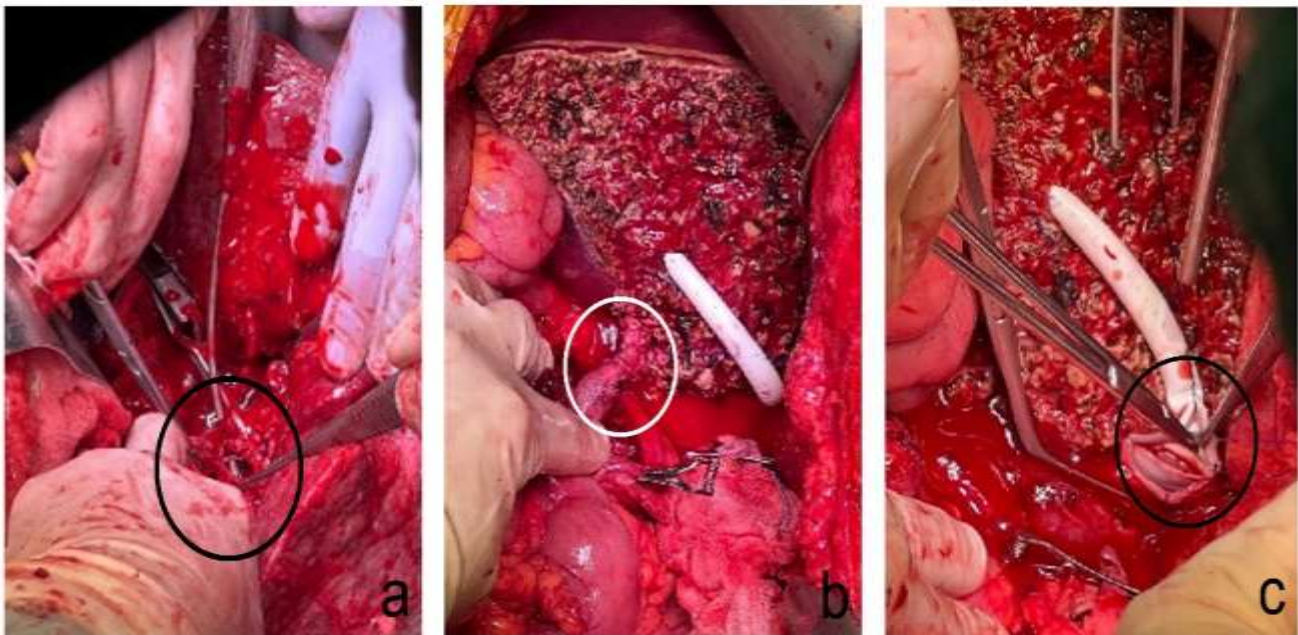


FIGURE 1- (a) PV rupture occurred due to severe adhesion during dissection of the portal vein, followed by repair. (b) End-to-end PV anastomosis. (c) Anastomosis of the right hepatic vein with the inferior vena cava (IVC).

An ultrasound on May 23 revealed a significant increase in resistance to blood flow in the liver. The systolic pressure (V systolic) in the hepatic artery decreased from 65.7 cm/s to 46.8 cm/s, and the resistance index (RI) changed from 0.67 to 0.57. Additionally, the acceleration time decreased from 0.044 seconds to 0.022 seconds (V

systolic >40-60cm/s, RI: 0.5-0.8, and acceleration time ≤ 0.08 sec).

On CTA, a 0.4 cm stenosis of the vascular anastomosis can be observed, but there is still blood flow. Additionally, there are indications of hypodensity along the PV in the liver V5 implant. Shows partial thrombosis, as shown in Figure 2.

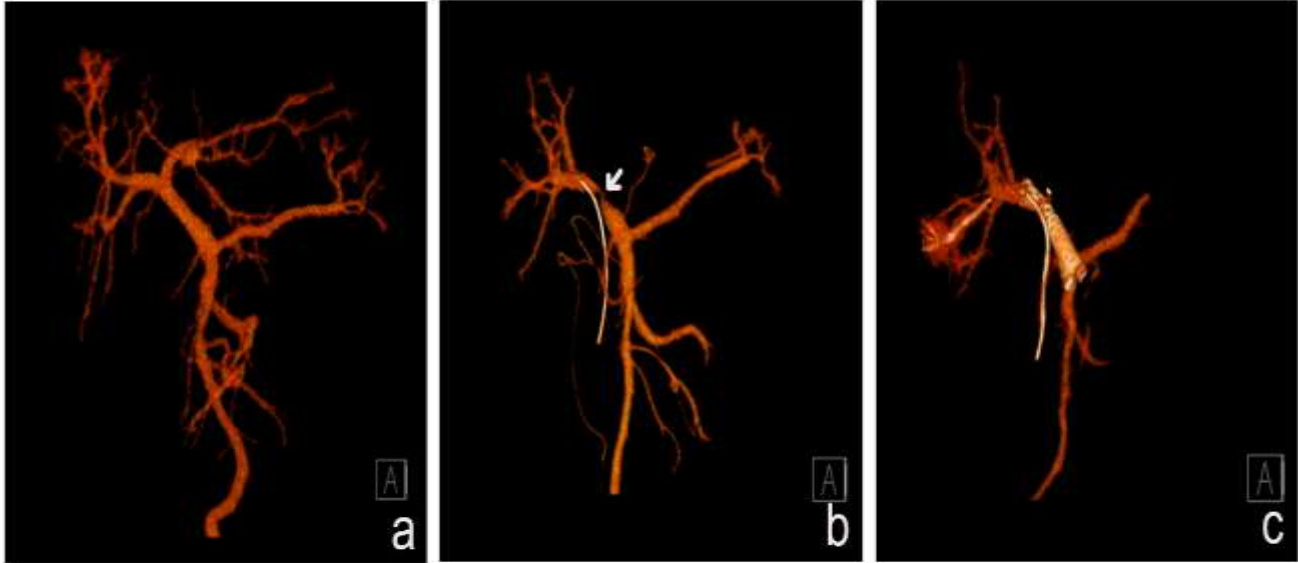


FIGURE 2- (a) CT and reconstructed 3D imaging of liver blood vessels were performed during pre-transplantation evaluation. (b) After transplantation, obvious narrowing of blood vessels can be observed. (c) One week after the vascular stent placement, it can be observed that the stent has not narrowed or deformed again.

During digital subtraction angiography (DSA), a percutaneous transhepatic cholangiography drainage (PTCD) set is utilized to percutaneously puncture the liver directly into the right hepatic vein under ultrasound and

fluoroscopic guidance, and then replaced with a 7Fr. catheter. Thread the sheath along the guide wire and insert it into the catheters (4 Fr. RC1, 5 Fr. Omnio, 5 Fr. Pigtail), as shown in Figure 3.

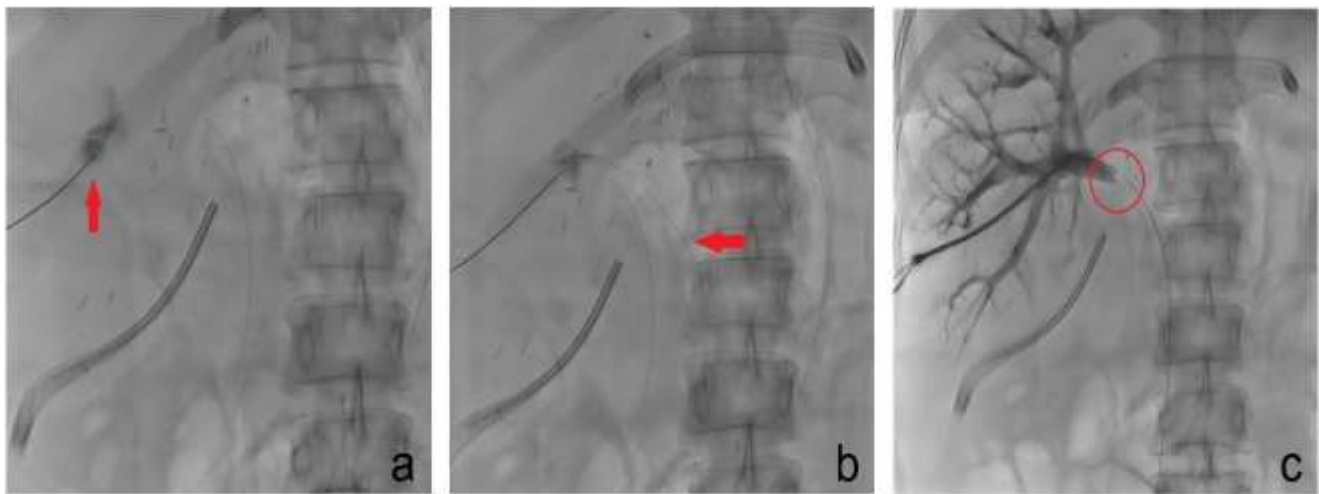


FIGURE 3- (a) The arrow indicates a PTC D 21G needle that percutaneously penetrates the liver and enters the right hepatic vein under ultrasound guidance. (b) Insert the 0.018 guide wire (indicated by the arrow) from the PTC D set and navigate it into the portal vein through the right hepatic vein. (c) Replace with 7Fr. Insert the sheath and 0.035 guide wire, and then inject the contrast agent.

Under fluoroscopy, it is evident that both the left hepatic vein and the right hepatic vein are fully filled with contrast agent. The circle represents stenosis at the surgical anastomosis, which obstructs the smooth downward flow of the contrast agent.

Please indicate the location of the PV graft. Venography of the superior mesenteric vein (SMV) shows venous reflux, resulting in ileal varices and bilateral pelvic congestion. Portal venography shows a concentrated high-grade stenosis at the anastomosis site, as shown in Figure 4.

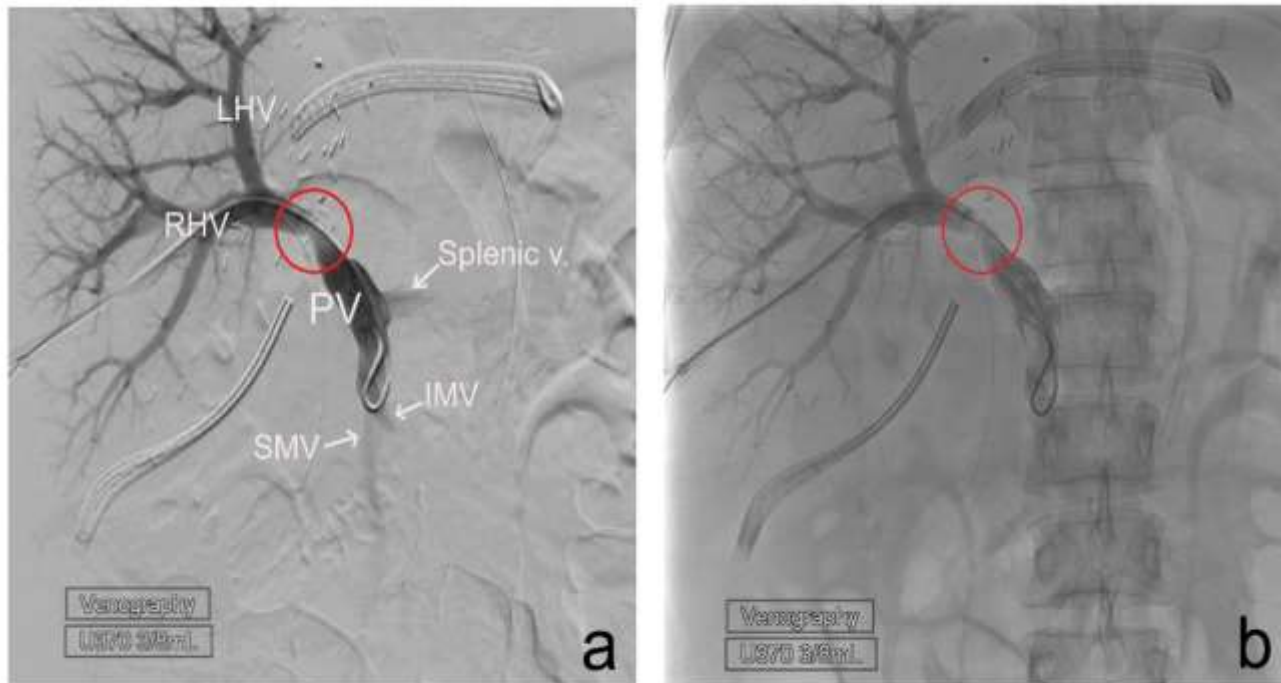


FIGURE 4- Insert a 5 Fr. Ominio hydrophilic catheter into the distal end of the portal vein and perform a series of venous photographs. The red circle clearly indicates the stenosis of the blood vessel. (a)(b) The picture illustrates the comparison before and after weight loss.

RESULT

The intravascular pressure of the PV was measured. It was 26 mmHg before the stenosis (proximal end) and 23 mmHg after the stenosis (distal end). The venous pressure in the liver was 23 mmHg, and the splenic vein pressure was 26 mmHg. The normal portal pressure should be 5-10 mmHg, but in this case, the portal pressure difference was less than 3 mmHg. This has adverse effects on liver

function and blood circulation, and it will cause the transplanted liver to fail.

A self-expandable stent (Bard® E-LUMINEXX™ 10mm) is used to expand the PV stenosis. After the stent was placed, the diameter of the vessel at the anastomotic stenosis of the PV significantly increased. Glue embolization (NBCA) is utilized to seal the entire path of the catheter through the liver (intervention tract) in order to prevent infection and the risk of re-bleeding, as shown in Figure 5.

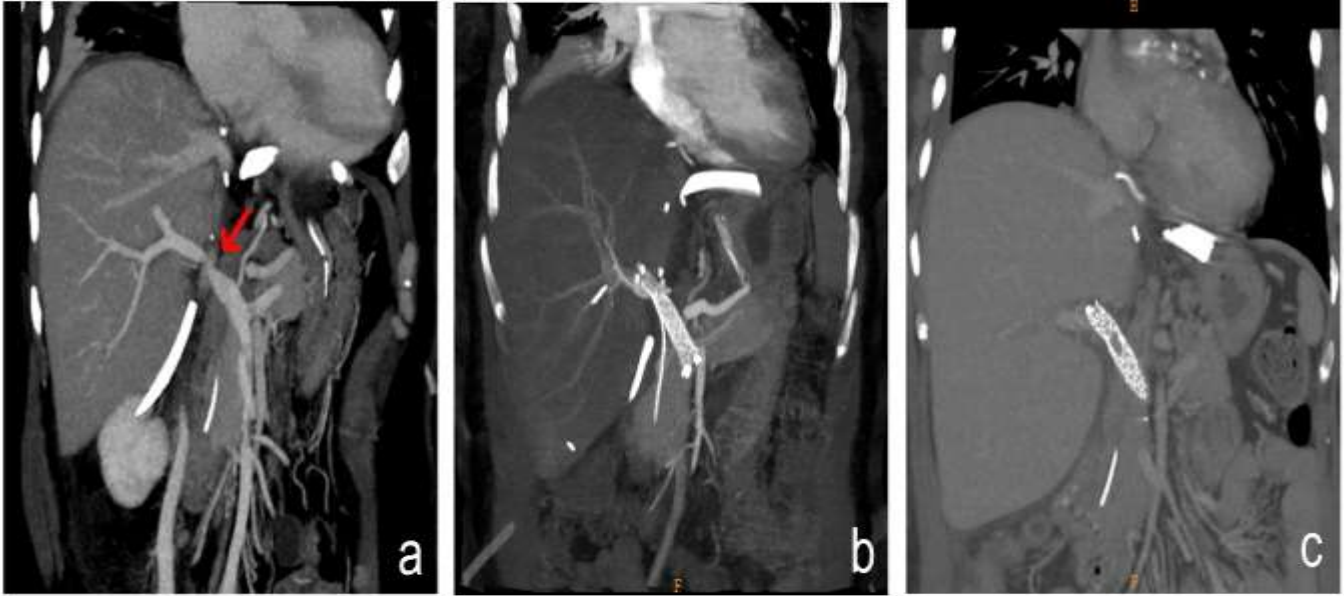


FIGURE 5- (a) This picture shows a CT MIP coronal view. After transplantation, obvious narrowing of blood vessels can be observed. (b) One week after the self-expanding stent was placed, the stenosis has significantly increased. (c) Three weeks after the self-expanding stent was placed, there was no restenosis in the blood vessel, but slight thrombosis occurred. The patient continued to take anticoagulants. Then use ultrasound for ongoing tracking.

Portal venography after stent placement revealed that the stent was fully expanded, positioned as planned, and resulted in a significant increase in portal blood flow, as shown in Figure 6.

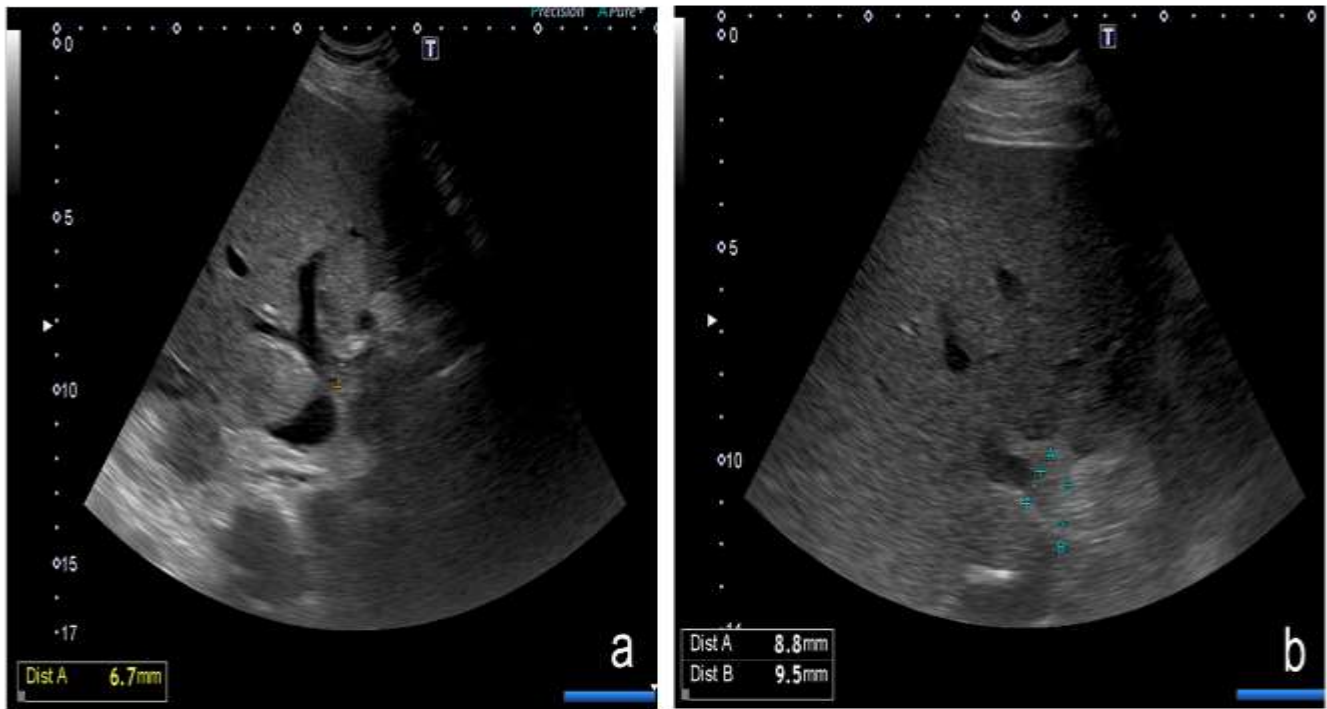


FIGURE 6- (a) Before stent placement. (b) After stent placement, ultrasound shows that the diameter of the blood vessel has significantly increased.

DISCUSSION

In this case, the PV was damaged during LDLT. It is possible that the inner diameter of the blood vessel was reduced after the repair, leading to postoperative stenosis of the anastomosis. After LT, the most common complications arising from the anastomosis are bile duct stenosis or bile leakage.

Followed by vascular stenosis or embolism: (1) Hepatic artery stenosis or embolism. The factors that contribute to hepatic artery thrombosis or anastomotic stenosis are primarily associated with hepatic artery anastomosis surgery or the vascular tension of the anastomosis. These factors can lead to jaundice, abdominal pain, and hypotension in patients. Confusion or even coma can occur when liver function is damaged. (2) PV stenosis or embolism may be caused by surgical factors, preoperative portal hypertension, or thrombosis. Symptoms include severe ascites, portal hypertension, and esophageal variceal bleeding. Severe cases may require transplantation. The liver loses its function.

CONCLUSION

After the successful surgery, the liver enzymes alanine transaminase (ALT) and aspartate transaminase (AST) continued to increase, and a large amount of ascites was produced. A PV stent was placed, although balloon percutaneous transluminal angioplasty (PTA) was not performed due to the incomplete repair of postoperative blood vessel fragility. A follow-up CT three months later revealed improvement in the stenosis, absence of PV restenosis, and a significant rebound in the liver index. Compared to another surgery, the placement of a stent in the blood vessel greatly reduces the risk, and it can also lead to a positive prognosis for the patient. It can be used as a recommended treatment for complications of vascular stenosis following transplantation.

Consent for Publication

All authors agree to the journal rules for publications.

Availability of Data and Material

Not applicable.

Competing Interests

The authors declare no conflict of interest.

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Authors' Contributions

C.-I. S., S.-Y. H., H.-M. C., and C.-T. C contributed equally to this work.

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