**Original Article**

**Adult to pediatric living donor liver transplantation for portal cavernoma**

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**Aim:** Portal cavernoma (PC) is an important cause of non-cirrhotic portal hypertension with severe complications, such as variceal hemorrhage in pediatric patients. With the development of new surgical techniques, living donor liver transplantation (LDLT) has recently been recognized as a viable but challenging treatment option for PC. The purpose of the present study was to summarize the efficacy of LDLT in PC patients and to carry out a follow-up study of pediatric recipients.

**Methods:** The primary indication for LDLT in our research was PC with severe variceal bleeding and liver function decompensation. Three patients were diagnosed with PC following evaluation with computed tomography angiography and abdominal color Doppler ultrasonography (CDU).

**Results:** Various surgical techniques, including jump bypass grafting for portal vein anastomosis, were carried out according to the range and degree of cavernous transformation within the splenic vein and superior mesenteric vein. Postoperative CDU confirmed the early integrity of the portal vein (PV) in each patient. PV rethrombosis occurred in one patient 7 days after LDLT, despite anticoagulation therapy with coumadin. Two of the three patients had no further episodes of variceal hemorrhage during the 2-year follow-up period.

**Conclusions:** The present study is the first report of the successful use of LDLT to treat pediatric PC patients. We conclude that LDLT is effective for the majority of pediatric patients with PC.

**Key words:** computed tomography angiography, indication, living donor liver transplantation, portal cavernoma

**INTRODUCTION**

PORTAL CAVERNOMA (PC) or the cavernomatous transformation of the portal vein, which can occur as early as 6–20 days after birth, is an important cause of gastrointestinal bleeding among children. Variceal bleeding is the most common manifestation of PC. Approximately 80% of children diagnosed with PC have at least one episode of upper gastrointestinal bleeding caused by esophageal varices in their lifetime. Other symptoms include thrombocytopenia, abdominal pain, transient or persistent ascites, anorexia, fatigue, body-weight loss and so on. Many symptoms are associated with splenomegaly and hypersplenism. The bleeding episodes are characterized by high morbidity, frequent hospital admissions, and consequent high healthcare expenses. Therefore, it is necessary to approach these patients appropriately so that the quality of life of children with PC can be improved.

PC usually results from a portal vein (PV) thrombotic event. Within a few days after thrombosis, an extended network of small and extremely sinuous vessels develops within the hepatoduodenal and hepatocolic ligaments as a means of compensating for an impaired hepatofugal flow. Eventually, this leads to the formation of a concentrated vascular system of the PV or its tributaries, which ultimately extends into the liver (intrahepatic cavernoma). Slow blood flow through a tortuous network of veins, permanent periportal fibrosis, redistribution of the blood and new thromboses all contribute to an increase in blood pressure. As a result of this pathophysiological condition, the majority of patients with PC will develop portal hypertension or esophagogastric varices, splenomegaly, or even biliary
abnormalities. The development of esophagogastric varices and variceal bleeding from PC poses a difficult therapeutic problem and the optimal therapeutic strategy is multifaceted and controversial.\textsuperscript{1,2} A variety of surgical techniques have been proposed to manage these patients, such as various portosystemic shunts and gastroesophageal decompression, but when these fail, surgical options are limited, especially for intrahepatic cavernoma. Splenectomy should be avoided as a response to hypersplenism, because splenectomy itself is an etiological factor in portal vein thrombosis (PVT). Rapid progress in the surgical technique of orthotopic liver transplantation in the past few years have shown that patients with PC can be safely and successfully transplanted.\textsuperscript{3,4} Given severe organ shortages, living donor liver transplantation (LDLT) has recently become more common. This approach is well suited for use with pediatric recipients because younger patients require a lower weight of graft. It is also associated with reduced ischemic time as compared with grafts taken from deceased donors.\textsuperscript{5}

During liver transplantation, the need to restore PV integrity in portal vein thrombotic patients led to the development of various specialized surgical techniques, each of which is suited to different circumstances. These include vascular reconstruction using venous graft, thrombectomy, or cavoportal hemitransposition.\textsuperscript{6–10} At the liver transplantation center of Chongqing Medical University, almost all patients with PC (even those with advanced disease) can elect to undergo a liver transplantation. The purpose of the present investigation was to retrospectively review our experience of carrying out LDLT in pediatric patients with PC and to evaluate long-term results. Our goal was to determine the feasibility and indications of LDLT in pediatric PC patients.

\section*{PATIENTS}

THREE PATIENTS WERE admitted to our facility presenting with severe variceal bleeding. After a diagnosis of PC, they were managed with LDLT at the Children’s Hospital of Chongqing Medical University between July 2006 and March 2007. These patients were retrospectively reviewed after appropriate institutional review board approvals had been obtained. The preoperative medical histories are summarized in Table 1 and pertinent details of each patient are described below.

Patient 1 was admitted to our hospital with acute gastrointestinal bleeding and shock. Emergency medical treatment was required. The patient’s medical history showed that the child had chronic portal hypertension that manifested as multiple episodes of hematemesis, chills, fever, fatigue and melena requiring blood transfusion for 5 years. The patient underwent a course of endoscopic band ligation at 5 years of age but progressively worsened with recurrent variceal hemorrhage. Emergency esophagogastroduodenoscopy showed large esophageal varices with oozing blood. Results of a liver function test are shown in Table 1. Color Doppler sonography clearly identified a sponge-like mass of collateral vessels around the main PV, and this was an indicator of cavernous transformation of the PV. Because of their deteriorating condition and uncontrolled bleeding, this patients was listed for liver transplant and underwent a living orthotopic liver transplantation from her father.

\begin{table}[h]
\centering
\caption{Preoperative summary of portal cavernoma cases}
\begin{tabular}{lll}
\hline
& Patient 1 & Patient 2 & Patient 3 \\
\hline
Age (years) & 9 & 7 & 10 \\
Diagnoses & PC hypersplenism & PC hypersplenism & PC hypersplenism hepatic encephalopathy \\
Previous operation & Endoscopic band ligation & No & splenorenal shunt \\
Melena & + & + & + \\
Preoperative liver function & & & \\
TB (2.0–20) & 17.2 µmol/L & 46.6 µmol/L & 217.3 µmol/L \\
DB (0–4) & 6.9 µmol/L & 34.5 µmol/L & 167.4 µmol/L \\
ALT (0–20) & 65 U/L & 84 U/L & 124 U/L \\
γGT (0–50) & 125 U/L & 75 U/L & 182 U/L \\
Albumin (33–52) & 31.9 g/L & 25.8 g/L & 28.2 g/L \\
PT (9–16) & 16 s & 19 s & 25.9 s \\
APTT (28–39) & 38 s & 45 s & 56 s \\
\hline
\end{tabular}
\end{table}

ALT, alanine aminotransferase; APTT, activated partial thromboplastin time; DB, direct bilirubin; PC, portal cavernoma; PT, prothrombin time; TB, total bilirubin.

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Patient 2 initially presented with hematemesis at 6 years-of-age. At that time, an upper gastrointestinal endoscopy showed esophageal varices, and an abdominal ultrasound documented chronic portal hypertension with periportal enlarged collateral vessels and splenomegaly, suggestive of partial portal venous thrombosis and cavernomatous transformation. The patient was given drugs to treat the symptoms (analgesics, antispasmyotics) and proton pump inhibitors, resulting in a general improvement. In 2006, the patient experienced several episodes of hematemesis and the child’s condition further deteriorated. Initial tests showed liver function decompensation as shown in Table 1. Given the extensive intrahepatic cavernous transformations, varices, liver function decompensation and concern regarding the expected low effectiveness of a surgical portosystemic shunt, it was decided to proceed with liver transplantation directly.

Patient 3 was a previously healthy patient that presented at 5 years-of-age with hematemesis requiring blood transfusion. Imaging showed cavernous transformation of the PV as well as splenomegaly and esophageal varices. The patient commenced thrombolytic therapy, but progressively worsened with recurrent variceal bleeding. In 2002 and 2003, the patient received a proximal splenorenal shunt and splenectomy in succession. However, just recently, the child had developed jaundice and hepatic encephalopathy. His liver function tests were markedly increased at admission. The routine blood test results from the three cases are presented in Table 1.

METHODS AND RESULTS

Imaging check

Imaging evaluation included both color Doppler ultrasonography (CDU) and computed tomography angiography (CTA) in all cases. PC was suspected based on the tortuous aspect of the PV in the hepatic hilum and the presence of an echoic, inhomogeneous image inside the portal lumen without Doppler signal and splenomegaly (Fig. 1). In patient 3, a dilated common bile duct (8 mm) was also shown. We measured the maximum diameter of the main portal, splenic, and superior mesenteric veins with calipers from wall to wall in a segment of the extrahepatic PV (Fig. 1). All CTA images were obtained at our institution with a helical CT scanner (LightSpeed VCT, GE Medical Systems, Milwaukee, WI, USA). Cavernous transformations were recorded in all three patients (Fig. 1), with loss of the normal architecture of the portal system. In all three patients, numerous vascular channels in the porta hepatis extended into the intrahepatic portal lobar branches, including bilateral lobar spread in patient 2 and protrusion into the right lobar branch without further extension in patients 1 and 3. Thrombosis of the involved portal venous branch was seen as a small, hypodense vessel within the cavernoma. The portal cavernous transformation extended to the splenomesenteric confluence in patients 1 and 2, and to the superior mesenteric vein in patient 3. A splenorenal shunt apparently inserted during a prior surgery, was identified by CT in patient 3. Manifestations of portal hypertension were also detected in all the patients. This included splenomegaly, mild to moderate ascites, and varices (gastroesophageal, periportal, and periportal) (Fig. 1).

Liver transplantation

The donor was the child’s father (patients 1, 2) and the patient’s mother (patient 3). All donor grafts were taken from the left lobe (segments 2, 3, 4, and middle hepatic vein). Graft hepatic vein venoplasty was carried out in all three cases.

Among the three PC patients, the tortuous, closely packed veins and venules formed a spongelike lesion that extended into the intrahepatic portal lobar branches and measured approximately 3.5–8.5 cm in diameter. Hilar fibrosis and inflammation was noted, and this made hilar dissections around the hepatoduodenal ligament very laborious. Some technical difficulties were encountered, including the dissection of a PV that featured no obvious lumen, hemorrhage and the separation of the bile duct from the PV. Some of the dilated veins contained old, partially organized thrombi. The branches of the hepatic ducts showed segmental dilatation of the lumen and inspissated bile in patient 3. After dissection and ligation of the hepatic arteries, the PV and bile duct were clamped en masse.

Accurate knowledge of the patient’s anatomy is essential to plan appropriate steps for surgical anastomoses. If the cavernous segmental transformation is high enough that the PV can be encircled and clamped superior to the pancreas, one can carry out the usual venous anastomosis of the donor PV to a large recipient collateral vein of PV (patient 1) or to the confluence of the splenic and superior mesenteric veins (patient 2). In patient 2, a short bridging vein graft was inserted from the donor liver. There was a cordlike PV with no apparent lumen

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available to carry out venous anastomosis in patient 3, and accordingly, this patient required a jump bypass graft (Fig. 2). The great saphenous vein was harvested from the donor in a standard fashion at a length of 10 cm. The distal part (consistent with direction of flow) of the venous graft was anastomosed end-to-side to the superior mesenteric vein (SMV) with a running 6–0 prolene suture, then tunneled into the hepatic hilum through the avascular window anterior to the pancreas, beneath the pylorus. This proximal section of the venous graft was subsequently sutured to the donor’s PV using end-to-end anastomosis. The diameter of the PV anastomotic stoma in these three patients was 0.8 cm, 0.6 cm and 0.6 cm, respectively. This is our preferred approach for the extensive cavernous transformation. Extreme care was taken to prevent air and particulate embolisms in the cava. Our approach involved flushing the grafts with University of Wisconsin solution before completion of the hepatic vein and portal anastomoses.

The grafts were reperfused after completion of the PV anastomosis, followed by hepatic artery reconstruction using microsurgical techniques. In all three patients, the choledochus was identified with enough caliber to maintain bile flow. To enable early detection of possible vascular complications, intraoperative color Doppler ultrasound was carried out before and after abdominal closure to show vascular flow patterns, pulsatility and velocities after vessel reconstruction. The falciform ligament was reconstructed using left-side grafts. After hemostasis was confirmed, a closed suction drain was left in the retroperitoneal space and the wound was closed. Table 2 summarizes the three patients in terms of recipient and donor parameters.

**Histologic findings from the resected livers**

The results of examinations of the histology of the resected livers were very similar across all specimens. A
mild to severe mixed lymphatic cell infiltration was present in most of the portal tracts. This was associated with portal, periportal and marked periductal fibrosis in all the patients, with mild to moderate bile duct damage and mild proliferation of bile ductules in patient 3. In certain areas, the bile ducts were compressed by dilated and thrombosed veins with reactive biliary epithelia, bile lakes, focally acute cholangitis with periductal fibrosis. A complex network of tortuous, closely packed veins and venules, some with thrombosed lumen, formed a spongelike lesion. This lesion extended into the liver parenchyma in all patients, with an irregularly thickened PV wall (Fig. 3).

Immunosuppression and follow-up
All patients had their portal system parameters recorded both preoperatively and postoperatively through CDU, as indicated in Tables 3 and 4. Every month thereafter, the patients underwent a clinical examination and basic laboratory tests. These included CTA at 1, 6 and 12 months (Fig. 4) after LDLT, and every 3 months thereafter. One recipient (patient 3) died in our facility as a result of early PV rethrombosis, which was detected by routine color Doppler ultrasound, accompanied by massive gastrointestinal bleeding 7 days after transplantation. The other two recipients survived and were studied until February 2008, with a mean follow-up of 26 months (range, 24–28). There were no steroid-resistant rejection cases or chronic rejection cases. Similarly, there were no obvious postoperative complications in the two surviving patients. Persistent arterial hypertension was not seen in any recipient. All the laboratory tests including liver function tests, uric acid, fasting blood glucose and routine blood tests were within normal limits from 2 weeks, and up to 2 years, postoperatively. Data concerning growth showed that at 2 years postoperative, the height and weight of the two children had greatly improved. The two survivors attended regular elementary schools.

Immunosuppression was conferred by treatment with tacrolimus and low-dose steroids. Tacrolimus was the primary immunosuppressive agent and it was given starting one day before transplantation at a dose of 0.1 mg/kg/day, divided into two doses. The target post-transplantation whole blood concentration of tacrolimus was 10–12 ng/mL during the first 2 weeks and around 10 ng/mL thereafter. Steroids were given starting

Table 2 Summary of recipient and donor operative variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>Patient 1</th>
<th>Patient 2</th>
<th>Patient 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total operative time</td>
<td>565 min</td>
<td>710 min</td>
<td>1126 min</td>
</tr>
<tr>
<td>Cold ischemia</td>
<td>30 min</td>
<td>21 min</td>
<td>55 min</td>
</tr>
<tr>
<td>Warm ischemia</td>
<td>28 min</td>
<td>33 min</td>
<td>48 min</td>
</tr>
<tr>
<td>Blood type combinations(D–R)</td>
<td>O-A</td>
<td>O-A</td>
<td>O-B</td>
</tr>
<tr>
<td>Graft weight</td>
<td>400 g</td>
<td>420 g</td>
<td>280 g</td>
</tr>
<tr>
<td>Graft/recipient ratio</td>
<td>2.1%</td>
<td>2.15%</td>
<td>1.24%</td>
</tr>
<tr>
<td>Venous graft (length)</td>
<td>No</td>
<td>GSV (3 cm)</td>
<td>GSV (10 cm)</td>
</tr>
<tr>
<td>Blood loss</td>
<td>560 mL</td>
<td>680 mL</td>
<td>1100 mL</td>
</tr>
<tr>
<td>Packed red cell transfusion</td>
<td>300 mL</td>
<td>400 mL</td>
<td>800 mL</td>
</tr>
</tbody>
</table>

D–R, donor–recipient; GSV, great saphenous vein.
at the time of graft reperfusion at a dose of 10 mg/kg, and subsequent dosages were gradually reduced from 1 mg/kg/day to 0.3 mg/kg/day over the first month.

DISCUSSION

MOST PHYSICIANS IN China might neglect portal venous obstruction in clinical practice. In fact, portal venous obstruction is an important cause of non-cirrhotic portal hypertension, which results in the rapid development of tortuous collateral veins around the thrombosed portal vein forming a “cavernoma”, termed as portal cavernoma. The portal cavernoma networks could be seen around the structures adjacent to the obstructed portal vein including the bile ducts, gallbladder, pancreas, gastric antrum, duodenum, etc. Paracholedochal varices (portal cavernoma) usually first develop within the hepatoduodenal and hepatocolic ligaments, and then might extend into the midsized intrahepatic portal veins (intrahepatic portal cavernoma) or lead to the formation of gallbladder varices.

The most common etiology of PC in pediatric patients is intra-abdominal infection, which occurs in 50% of all cases. This is markedly different from the etiology in adults. Congenital anomalies of the portal venous system have been detected in 20% of children who have PV obstruction and thrombosis. Such anomalies are often associated with cardiovascular problems and biliary tract abnormalities. Therefore, these anomalies should be excluded in neonates or in children with PC in the absence of other etiological factors. In our patients, preoperative examinations were carried out and no obvious cardiovascular anomalies were detected. Biliary morphological abnormalities are commonly identified in patients who have PC. In adults, the observed expression of such abnormalities ranges from 80% to 93% of all PC patients. However, as observed in the patients in the present study, the biliary morphological abnormalities include stenoses and angulations related to compression of the biliary tree by the lumen of PC veins were present only in patient 3. The reduced incidence of biliary complications in children might be explained in terms of an etiology of thrombosis, in contrast with the scenario in adults. Liver cirrhosis or
neoplasia might play a leading role in adult patients with PC, which is always associated with biliary damage and is termed portal biliopathy.12

Depending on the clinical manifestation, a variety of procedures have been proposed, including PV bypass, gastroesophageal devascularization, portosystemic shunt, and stent insertion14–16 in selected children in order to recover PV integrity. Unfortunately, because of the poor quality of the splanchnic veins and fibrous adhesion, these surgical procedures are not often feasible and are associated with significant recurrence rates as a result of hemorrhage and biliary infection. During the past few years, increasingly sophisticated techniques have become available for liver transplantation surgery. As a consequence, the indications have broadened for orthotopic liver transplantation.17,18 Other researchers have argued that portal cavernoma should be recognized as one of the indications for liver transplantation.17 Cavernous transformations become more technically challenging at the time of orthotopic liver transplantation. Because this procedure is associated with perioperative morbidity and mortality as a result of rethrombosis, pancreatitis, and increased blood loss,19,20 many transplantation techniques to deal with cavernous transformation have been suggested.21 These include PV grafting, extra-anatomic venous reconstruction, and many others. Today, almost all patients with portal system thrombosis, even of a very extensive nature, can safely undergo orthotopic liver transplantation. There exist several reports on adult patients who undergo liver transplantation after diagnosis of PC. However, we were unable to find any reports that study liver transplantation in cases of pediatric PC. To our knowledge, the present report is the first to describe the successful use of liver transplantation in treating pediatric PC patients. This remains true even though two of our three patients had essentially normal liver function preoperatively. This might not be an important factor when planning liver transplantations, as most patients generally do bleed to a severe extent, and this might require surgical intervention and could avoid or decrease the difficult and fatal complications involved in liver transplantation if carried out at an early stage.

Preoperative knowledge of a PV abnormality might minimize the threat of the anhepatic phase of the LDLT procedure. Preoperative CTA can accurately determine the detailed PV vascular anatomy status in candidates for LDLT.22 We believe that the presence of mesenteric variceal abnormalities on a preoperative CTA scan is a good indicator of the feasibility of a liver transplantation. Abdominal Doppler ultrasonography is the standard diagnostic investigation, because it can provide reliable information regarding the directionality of blood flow and visualization of the formation of new vessels around the thrombus (cavernoma).23 In patient 3, preoperative CTA and CDU imaging showed the presence of a very extensive cavernoma that presented in the superior mesenteric vein. We subsequently shifted our practice to promote the use of a jump bypass graft as long as 10 cm. CDU and CTA offered a noninvasive and complementary examination method that was appropriate for our needs.

Table 3 Preoperative and postoperative caliber change of the portal system

<table>
<thead>
<tr>
<th>Patient 1</th>
<th>Preoperative 0.8 cm</th>
<th>Preoperative 0.8 cm</th>
<th>Preoperative 0.6 cm</th>
<th>Preoperative 0.8 cm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient 2</td>
<td>Preoperative 1.2 cm</td>
<td>Preoperative 1.0 cm</td>
<td>Preoperative 0.6 cm</td>
<td>Preoperative 0.6 cm</td>
</tr>
<tr>
<td>Patient 3</td>
<td>Preoperative 1.0 cm</td>
<td>Preoperative 1.3 cm</td>
<td>Preoperative 0.6 cm</td>
<td>Preoperative 0.39 cm</td>
</tr>
</tbody>
</table>

PV, portal vein; SMV, superior mesenteric vein; SV, splenic vein.

Table 4 Time cause of postoperative portal flow

<table>
<thead>
<tr>
<th>Postoperative time</th>
<th>1 day</th>
<th>2 days</th>
<th>3 days</th>
<th>7 days</th>
<th>14 days</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient 1</td>
<td>Portal flow 0.25 m/s</td>
<td>0.27 m/s</td>
<td>0.23 m/s</td>
<td>0.26 m/s</td>
<td>0.24 m/s</td>
</tr>
<tr>
<td>Patient 2</td>
<td>Portal flow 0.28 m/s</td>
<td>0.31 m/s</td>
<td>0.32 m/s</td>
<td>0.27 m/s</td>
<td>0.30 m/s</td>
</tr>
<tr>
<td>Patient 3</td>
<td>Portal flow 0.37 m/s</td>
<td>0.35 m/s</td>
<td>0.32 m/s</td>
<td>0.29 m/s</td>
<td>0.36 m/s</td>
</tr>
</tbody>
</table>

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essment of candidates using a combination of CDU and CTA in pediatric patients before LDLT to facilitate definition of a suitable surgical strategy.

During intraoperative dissection of the PV by ever-sion of the vessel wall, the identified proximal abnormality extended into the intrahepatic PV in all of the patients, but patient 1 did exhibit a distally limited confluence of the splenic and superior mesentenic veins. Given the large size and high quality of the PV, it was amenable to simple thrombectomy, with direct anastomosis of the donor PV to the recipient PV. In patient 2, there was an extension of the cavernoma or thrombus to the superior mesenteric-splenic venous confluence or beyond. However, a high quality, easily mobilized PV allowed for a simple PV-to-PV anastomosis, with a single prosthetic short graft. These straightforward procedures might be considered in any patient who shows the appropriate anatomy. We have shown that patients with a minimally thrombosed PV can undergo successful transplantation with similar results to non-PV thrombosis patients. However, if the PV is proximally occluded or narrowed, distally open to the SMV, the extra-anatomic jump graft from donor PV to the recipient SMV should pass through the transverse mesocolon anterior to the pancreas and posterior to the pylorus. By this means, we avoided the need for an extensive peripancreatic dissection. Good flow was established in all cases soon after transplantation and there were no recurrent bleeding episodes on short-term follow-up. CDU and CTA angiography 1 month after surgery confirmed integrity in two of the three patients.

Although thrombus might be one of the indications for liver transplantation, altered portal blood flow and anticoagulation factors after liver transplantation might also be associated with the development of PV rethrombosis, as shown in the literature, which suggests incidence rates ranging from 6.2% to 28.6%. The mortality associated with rethrombosis was 100%, as reported in one study by J. Lendoire. Patients with interposition grafts are at high risk for PV. In the present study, patient 3 exhibited rapidly progressive thrombus, despite anticoagulation therapy with coumadin. The etiology of rethrombosis, not yet fully understood, might be related to several factors, including an increased resistance to portal flow and endothelial injury as a result of an elevated portal pressure. There exists no generally accepted criteria for the use of prophylactic anticoagulation. Some advocate preventive regimens with low molecular weight heparin, dextran, coumadin derivatives, or aspirin, although the role of these prophylactic measures remains unclear.

In conclusion, we have discussed three pediatric transplant-eligible patients who were diagnosed with PC and who subsequently underwent successful living-relative liver transplantation. The transplantation procedure was technically feasible, and there was no procedure-related mortality in any of our patients. The ideal treatment for portal thrombosis in liver transplantation scenarios depends on how extensive the PC is and on the experience of the surgeon. We confirm that LDLT can resolve most cases of PC, which is not a contraindication to liver transplantation at the present time, even though it might be associated with an increase in operative time and a lower survival rate consistent with the degree of PV extension. Complete splanchnic vein thromboses might require more complex surgical techniques. However, such complications were not present in any of the patients in the present study. Approaches

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to such cases would require portal arterIALIZATION or cavoportal haemitransposition, and perhaps even combined liver–intestine transplantation if necessary.28

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There are no conflicts of interest for all the authors.

REFERENCES

sis among 404 adult living donor liver transplantations. 
26 Kim YJ, Ko GY, Yoon HK, Shin JH, Ko HK, Sung KB. 
Intraoperative stent placement in the portal vein during or 
27 Condat B, Pessione F, Helene Denninger M, Hillaire S, 
Valla D. Recent portal or mesenteric venous thrombosis: 
increased recognition and frequent recanalization on anti-
28 Vianna R, Giovanardi RO, Fridell JA, Tector AJ. Multi-
visceral transplantation for diffuse portomesenteric 
thrombosis in a patient with life-threatening esophago 
gastroduodenal bleeding. *Transplantation* 2005; 80: 
534–5.