



Vascular Complications After Living Donor Liver Transplantation: A Brazilian, Single-Center Experience

K. Steinbrück, M. Enne, R. Fernandes, J.M. Martinho, E. Balbi, L. Agoglia, J. Roma, and L.F. Pacheco-Moreira

ABSTRACT

Background. In living donor liver transplantation (LDLT), vascular complications are more frequently seen than in deceased donor transplantation. Early arterial, portal vein, or hepatic vein thromboses are complications that can lead to graft loss and patient death. The aim of this study was to assess the incidence, treatment, and outcome of vascular complications after LDLT in a single Brazilian center.

Methods. Between December 2001 and December 2010, we performed 130 LDLT. Sixty-four recipients were children (27 weighing <10 kg).

Results. Nine recipients had vascular complications. Hepatic artery thrombosis (HAT) occurred in 4 (3.1%), portal vein thrombosis (PVT) in 3 (2.3%), and hepatic vein thrombosis (HVT) and hepatic arterial stenosis (HAS) in 1 (0.8%) patient each. Complications were identified by Doppler and confirmed by angiography or angiotomography. Patients with HAT were listed for retransplantation. One died before retransplant. Two children were submitted to retransplantation; one is still alive, with neurologic sequelae. One adult with HAT was retransplanted with a deceased donor graft and is doing well 58 months after surgery. Two patients with PVT died as a consequence of graft malfunction. In the other case, portal vein arterialization was performed, but patient died 11 months posttransplant. HVT was detected after cardiac reanimation and was treated with an endovascular stent. This patient died 3 months after LDLT. HAS was diagnosed after liver abscess development and was successfully treated by endovascular angioplasty. No recurrence was observed after 22 months. Follow-up ranged from 9 to 117 months.

Conclusion. Pediatric patients are more prone to develop vascular complications after LDLT. Long-term survival was statistically lower for recipients with vascular complications (33.3% vs 77.7%; $P = .008$).

VASCULAR PROBLEMS such as thrombosis and stenosis of the hepatic artery, portal vein, and hepatic vein are among the most serious complications reported after liver transplantation and are more frequently seen among recipients of living donor transplantations (LDLT).¹⁻⁴ These complications can lead to increased morbidity, graft loss, and patient death. The aim of this study was to assess the incidence, treatment, and outcome of vascular complications after LDLT in a single Brazilian center.

PATIENTS AND METHODS

Between December 2001 and December 2010, we performed 130 LDLT. There were 64 pediatric and 66 adult recipients. Patients' charts were analyzed to identify vascular complications related to

the hepatic artery, portal vein, and hepatic vein. Details of the preoperative donor evaluation, as well as donor operative technique have been described previously.^{5,6}

From the Liver Transplantation Unit (K.S., M.E., R.F., E.B., L.A., J.R., L.P.-M.), Bonsucesso Federal Hospital/MS, Rio de Janeiro; and the Medical Sciences Post-Graduation Program, Fluminense Federal University (K.S., R.F., J.M.M., L.A., J.R.), Niterói, Brazil.

Address reprint requests to Klaus Steinbrück, Hospital Federal de Bonsucesso, Serviço de Transplante Hepático e Cirurgia Hepato-Biliar, Av. Londres, 616, prédio 3/2º andar, Rio de Janeiro/RJ, Brazil. E-mail: drsteinbruck@yahoo.com.br

Pediatric Group

Sixty-two patients underwent 64 transplantations, including 2 retransplants. The male/female rate was 50%/50%. Recipients ranged from 7 months to 15 years old (50% were <2 years old), and their weight ranged from 5 to 50 kg (42% weighed <10 kg). Graft to recipient weight ratio (GRWR) varied from 0.6% to 6.8% (mean, 2.83%). Biliary atresia was the main indication for transplantation (36 cases; 56.2%). The others were acute liver failure in 11 (17.1%) cases, hepatoblastoma in 5 (7.8%), biliary hypoplasia in 3 (4.6%), hepatic artery thrombosis after LDLT in 2 (3.1%), and Caroli disease, hepatocellular carcinoma, secondary biliary cirrhosis, α 1-antitrypsin deficiency, autoimmune cirrhosis, Alagile syndrome, and congenital hepatic fibrosis in 1 each (1.6%), each. For the hepatic vein, in most cases, a piggy-back anastomosis was executed with continuous 5/0 polypropylene suture. The portal vein anastomosis was then performed end-to-end, using a 6/0 or 7/0 polydioxanone continuous suture for the posterior wall and interrupted for the anterior wall. Interrupted 8/0 polypropylene sutures were done in the hepatic artery. Grafts transplanted were 3 left livers (LL; 4.7%), 46 left lateral sections (LLS; 71.9%), and 15 reduced left lateral sections (segment III) (RLLS; 23.4%).

Adult Group

Sixty-five patients underwent 66 LDLT, including 1 retransplant. Thirty-eight were male (58%) and 27 were female (42%). Recipient mean age was 50.5 years (range, 19–69 y) and mean GRWR was 1.1% (range, 0.77%–1.92%). Cirrhosis due to hepatitis C virus (HCV) was the first indication for transplantation (26 cases; 39.4%). Others indication were hepatocellular carcinoma in 14 (21.2%) cases, alcoholic cirrhosis in 7 (10.6%), amyloidosis in 4 (6.1%), cryptogenic cirrhosis in 3 (4.5%), autoimmune cirrhosis, cirrhosis associated with hepatitis B virus (HBV), secondary biliary cirrhosis, and primary sclerosing cholangitis in 2 (3%) each, and Budd–Chiari syndrome, hemochromatosis, Caroli disease, and congenital hepatic fibrosis in 1 (1.5%) each. Hepatic vein anastomosis was done end-to-side with continuous 4/0 or 5/0 polypropylene suture. Portal vein anastomosis was performed end-to-end with continuous and interrupted 5/0 or 6/0 polypropylene suture. Hepatic artery anastomoses were done in the same fashion as in the pediatric group. Grafts for the adult group were 65 right livers (RL; 98%) and 1 LL (2%).

All recipients were operated with the use of 3 \times loupe magnification for hepatic and portal vein anastomoses and 6.5 \times for hepatic artery. Doppler examination was carried out just after anastomoses to ensure vascular patency and repeated within the first week posttransplantation. Graft function was monitored by detailed biochemical tests. Hematocrit was kept under 30%. Except for a few reported cases of allergy, every patient received low-molecular-weight heparin and aspirin after transplantation.

The Fisher exact test was used to identify risk factors for vascular complications. $P < .05$ was considered significant.

RESULTS

Vascular complications were found in 9 (7%) recipients, 6 children and 3 adults. Hepatic artery thrombosis (HAT) was identified in 4 (3.1%) patients, 3 children and 1 adult. Portal vein thrombosis (PVT) occurred in 3 (2.3%) children. Hepatic vein thrombosis (HVT) and hepatic artery stenosis (HAS) occurred in 1 (0.8%) adult patient, each. Among the children, mean age was 3.4 years (range,

0.9–9.2), mean weight was 13.3 kg (range, 5–30), and mean GRWR was 2.55% (range, 1.3%–3.9%). The male/female ratio was 50%/50%. Among the adults, complications occurred in 2 men and 1 woman. Recipients' age and GWRW were 49, 51, and 35 years and 0.93%, 1.4%, and 1.3%, respectively. HAT was diagnosed 12–22 days posttransplantation and was associated to increase of liver function tests, biloma formation, and graft failure. All pediatric recipients with HAT received LLS grafts and were transplanted owing to acute liver failure, biliary atresia, or Alagile syndrome. The adult patient with HAT was transplanted because of HCV cirrhosis and received a RL graft. Diagnosis of HAT was first made by Doppler ultrasonography and then confirmed by angiotomography. These 4 patients were listed for retransplantation. One child died before retransplantation, from HAT complications, on the postoperative day (POD) 34. The other 2 underwent LDLT again, although, 1 died 7 months after retransplantation due to HHV-6 hepatitis. The other is still alive with neurologic sequelae. The adult with HAT was retransplanted with a deceased donor graft and is doing well 58 months after surgery. PVT was identified during transplantation in 1 case and POD 19 and 30, in the other 2 cases. Three patients had biliary atresia. Grafts transplanted were 2 RLL (recipients weighed <10 kg) and 1 LLS. PVT was diagnosed by Doppler ultrasonography and confirmed by angiotomography in 2 cases. The child with PVT identified during transplantation underwent 2 unsuccessful attempts at portal vein anastomosis after thrombectomy. Arterialization of the portal vein to splenic artery with an iliac artery graft was done and surgery finished as routine. This patient died 11 months posttransplantation, after several infectious episodes. The other 2 children died on POD 23 and 46, owing to thrombosis and malfunction of the graft. HVT occurred in 1 patient just after transplantation, while she was in the intensive care unit (ICU) and developed cardiac arrest. After cardiopulmonary resuscitation (CRP), HVT was identified by Doppler ultrasonography. Cavography confirmed thrombosis and an endovascular stent was satisfactory placed to treat the problem. This patient died 3 months after LDLT because of septicemia. HAS was suspected 4 months after LDLT, when a patient developed intrahepatic abscess. Arteriography confirmed HAS. Percutaneous transarterial angioplasty was performed and the patient was maintained under anticoagulation therapy. No recurrence of stenosis was detected after 22 months. Follow-up of patients submitted to LDLT ranged from 9 to 117 months.

DISCUSSION

The risk of vascular complications is relatively high in LDLT when compared with whole graft transplantation, especially in pediatric recipients.^{1–4} Reported rates can be as high as 25%, 16%, and 11% for HAT, PVT, and HAS, respectively.^{7–9} HVT is an unusual complication. In our experience, HAT occurred in 3.1% of patients, PVT in 2.3%, and HVT and HAS in 0.8%, which are regarded low incidences.

In the HAT group, we observed a greater propensity for this complication in children than in adults ($P > .3$). Additional risk factors for HAT in children—namely, age <2 years and body mass <10 kg¹⁰—were present in only 1 child (1.6 years old) and could not be associated as a risk for this group. The type of graft was the same (LLS) for the 3 children, but it was not considered a risk factor ($P > .5$). As seen in our patients, early HAT usually presents with acute hepatic failure, a sudden and significant elevation of liver enzymes, unexplained sepsis, or liver infarction. Patients with HAT are at significant risk for graft loss and increased morbidity and mortality, and our experience confirms these observations.^{11–14} Retransplantation was indicated for all 4, but only the adult was successfully treated with a deceased donor graft and is still alive without complications. From our 3 pediatric cases, 2 managed to be retransplanted, but only 1 is alive. This child developed severe neurologic sequelae, caused by brain damage, associated with liver failure. Different from previous reports,¹⁰ our adult patient with HAT had a better outcome when compared with children with HAT.

PVT was observed only in children ($P > .1$); all had biliary atresia. Portal vein hypoplasia, which is common in patients with this malformation, is among the main risk factors for PVT,^{15,16} and it was present in all children who developed this complication in our series ($P > .2$). In 2 cases, recipient body mass was <10 kg and, to avoid graft-to-recipient size mismatch, they received a RLLS graft. Large-for-size grafts can increase the incidence of vascular complications because of vessel kinking, low portal flow speed, disproportion of vessels diameter, and increased intra-abdominal pressure.^{17,18} The use of RLLS, although it reduces the chance of size mismatch, adds technical difficulty because of very small vessel diameter. In our series, we performed 15 LDLT with RLLS grafts and, despite the 2 TVP cases, it was not considered a risk for vascular complication ($P > .1$). Complications related to the portal vein are less common than those of the hepatic artery, but are associated with a high graft loss incidence, as well.¹⁹

Hepatic vein complications are less frequent, with overall incidences around 1%.²⁰ We had 1 case of HVT that was diagnosed after an episode of cardiac arrest. We believe that the low flow and the trauma caused by cardiac reanimation maneuvers were responsible for this complication. It was confirmed and managed by interventional radiology. HAS should be suspected in patients with unexplained elevation of liver function tests and biliary complications. In our case, it was considered when recipient presented with liver abscess, 4 months after LDLT. Ultrasound-guided drainage of the collection revealed infected bile. Angiography confirmed stenosis in the site of anastomosis and it was successfully treated by balloon angioplasty.

The survival rates for 1, 6, and 12 months for recipients with and without vascular complications were 88.9%, 66.7%, and 33.3%, and 87.5%, 79.5%, and 77.7%, respectively. The 1-year survival rate was statistically inferior for patients with vascular complications ($P = .008$).

In conclusion, vascular complications after LDLT still represent a significant cause of graft and patient loss. In our experience, pediatric patients had a higher tendency to develop vascular problems after LDLT compared with adults. The use of RLLS did not increase the rate of vascular problems. The 1-year survival rate was statistically poorer for recipients with vascular complications.

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