Pediatric Liver Transplant: Results of a Single Center

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Abstract

Objectives: **Liver transplant in the pediatric population has become an accepted treatment modality for children with end-stage liver disease. In this study, we analyze our experiences with pediatric liver transplant at our center.**

Materials and Methods: **Since September 2001, 8 deceased-donor and 96 living-donor liver transplants have been done in 101 children (mean age, 6.7 ± 5.5 years; range, 2 months to 17 years). The children's charts were reviewed retrospectively.**

Results: **Indications for liver transplant were cholestatic liver disease (n=17), biliary atresia (n=24), Wilson's disease (n=16), fulminant liver failure (n=18), hepatic tumor (n=13), and other (n=13). The median pediatric end-stage liver disease score was 23.1 ± 11.1 (range, –8 to 48). The median follow-up was 24.2 ± 19.4 months (range, 1-77 months). Three children underwent retransplant. The main complications were infections (25.9%) and surgical complications (39.5%) (including biliary complications and vascular problems). The incidence of acute cellular rejection was 42.3%. Sixteen children died during follow-up, and, at the time of this writing, the remaining 85 children (85%) were alive with good graft functioning, showing patient survival rates of 90%, 85%, and 83% at 6, 12, and 36 months, respectively.**

Conclusions: **In conclusion, the overall outcomes of pediatric liver transplantation at our center are quite promising.**

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Liver transplant (LT) is an established curative therapy for children with chronic end-stage liver disease or acute liver failure. Until 1980, less than 40% of children receiving LTs survived more than 1 year after transplant (1, 2). Outcomes following LT in children have significantly improved over the past 2 decades because of advances in surgical procedures, preservation technology, immunosuppressive management, and perioperative care (3, 4). The aim of this study was to analyze the results of 104 LTs in 101 children at our institution.

Materials and Methods

Between September 20, 2001, and February 29, 2008, 104 LTs were done in 101 children at the Baskent University Hospital in Ankara, Turkey. There were 62 boys (61.3%) and 39 girls (38.7%). The mean age of the children was 6.7 ± 5.5 years (range, 0.2-17 years), and their mean weight was 22.7 ± 15.6 kg (range, 5-64 kg). Forty-one children weighed less than 10 kg, and 38 were younger than 1 year old (Figure 1). Indications for liver transplant were cholestatic liver disease (n=17), biliary atresia (n=24), Wilson's disease (n=16), fulminant liver failure (n=18), hepatic tumor (n=13), and other (n=13). Eight grafts were obtained from deceased donors, 96 grafts were from living-related donors. Donor selection

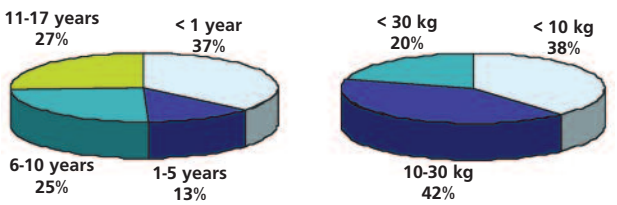

Figure 1. Distribution of the children's age and body weight

criteria are described elsewhere (5). Only donors with graft-to-recipient weight ratios > 0.8 and fatty liver < 30% were accepted. The residual liver volume as assessed by computed tomography always exceeded 35% of the total liver volume. The age of the donors ranged from 23 to 66 years. The relationships of the donors to the children were as follows: a mother in 40 children, a father in 37, a sibling in 4, a grandfather in 4, a cousin in 3, a paternal uncle in 5, a maternal uncle in 3, and a sister's husband in 1. The surgical technique used in the living donors has been described previously (6). In our subjects, segments 2 and 3 for the left lateral segment grafts; segments 2, 3, and 4 for the left lobe grafts; and segments 5 to 8 for the right lobe grafts were used for the transplants. Eight children received a right lobe graft, 26 received a left lobe graft, 62 received a left lateral segment graft, and the remaining 8 received a whole liver graft.

All biliary and vascular anastomoses were done with loupe magnification (2.5×) by the same surgeon. The technical details of the hepatic vein and portal vein anastomoses have been previously described (7). If there was a size discrepancy between the graft portal vein and the recipient portal vein, the smaller-sized portal vein was spatulated from both the anterior and the posterior walls to create a wide anastomosis site. An inferior hepatic vein larger than 5 mm was encountered in the 2 right lobe grafts. These 2 inferior hepatic veins were anastomosed end to side to the inferior vena cava. In the first 51 LTs, we made a hepatic arterial anastomosis by means of a modified parachute technique (8). We changed our hepatic arterial reconstruction technique in the last 53 LTs (9). Twenty-eight of the 104 liver grafts had 2 hepatic arteries. In 8 of those 28 grafts, 2 separate anastomoses were made between the graft hepatic arteries and the native hepatic artery branches. In the remaining 20 grafts, the adjacent edges of the neighboring hepatic arteries were spatulated and sutured together at the back table to create a single hepatic artery opening, and the native common hepatic artery was anastomosed to that orifice. One of the 104 liver grafts had 3 hepatic arteries. In that individual, the adjacent edges of the neighboring hepatic arteries were spatulated and sutured together to create a single hepatic artery.

Biliary reconstruction was completed with a duct-to-duct anastomosis in 64 LTs and with a Roux-en-Y hepaticojejunostomy in 40 LTs. Nine of the 104 liver grafts had 2 bile ducts. In these 9 grafts, the adjacent edges of the neighboring bile ducts were spatulated and sutured together to create a single bile duct opening. Two of the 104 liver grafts had 3 bile ducts. In 1 of these grafts, the adjacent edges of the neighboring bile ducts were spatulated and sutured together to create a single bile duct opening. In the second graft with 3 bile ducts, 2 neighboring ducts were sutured together and anastomosed end to end to the common bile duct, and the third duct was anastomosed separately end to side to the common bile duct. One of the 104 liver grafts had 4 bile ducts. In this graft, 2 neighboring ducts were sutured together, and 3 separate anastomoses were made over a jejunum limb. To allow external bile drainage, we used a T tube in 5 children, a straight feeding tube in 20 children, and a transhepatic biliary catheter in 40 children. In the remaining 39 children, no tubes or stents were used for external bile drainage.

Ultrasonographic examination of hepatic perfusion was done twice daily during the first week after surgery. In addition, routine ultrasonographic examinations were scheduled 1 month after orthotopic liver transplant and at 3-month intervals thereafter. A heparin drip infusion was begun on the day of transplant and was adjusted to maintain active coagulation time whole-blood trough levels between 150 and 200 seconds. The heparin infusion was continued for 1 week. After that, anticoagulation therapy consisted of aspirin (40 mg daily) and dipyridamole (4 mg/kg tid). All children also received tacrolimus-based immunosuppression. Tacrolimus blood levels were maintained between 10 and 15 ng/mL during the first month and then between 5 and 10 ng/mL, thereafter. Methylprednisolone (10 mg/kg) was administered intraoperatively. It was continued postoperatively from 10 mg/kg, tapered to 0.1 mL/kg at the end of the first month, and stopped at the end of the third month. Children received antifungal, antiviral, and antipneumocystis prophylaxis for 6 months after surgery.

Survival rates were estimated by the Kaplan-Meier method and compared with a log-rank test. Factors that affected mortality were analyzed with a Cox proportional hazards regression model. The level of significance was set at .05. Statistical analyses were calculated with SPSS statistical software (Statistical Product and Services Solutions, version 11.0, SPSS Inc, Chicago, IL, USA).

Results

Between September 2001 and February 2008, 8 deceased-donor (7.6 %), and 96 living-donor (92.4%) LTs were done in 101 children. When these 101 children were classified according to Child-Pugh scores, 10 were classified as A, 38 were classified as B, and 53 were classified as C. The mean Child-Pugh score was 9.7 ± 1.6 (range, 5-14), and the mean pediatric end-stage liver disease score was 23.1 ± 11.1 (range, –8 to 47). Our patients remained in the intensive care unit for a mean duration of 2.3 ± 2.2 days (range, 1-12 days), and the mean length of stay in the hospital was 20.5 ± 13.3 days (range, 6-52 days). Twenty-five children had undergone a previous procedure including a Kasai procedure (n=19), splenectomy (n=1), and exploratory laparotomy (n=5). In those 25 LT recipients, the mean length of surgery was 11 ± 1.5 hours (range, 9-13 hours), which was longer than that in the remaining 79 LTs (length of surgery, 7.8 ± 1.1 hours; range, 6-9 hours). Preoperatively, 8 children had gastrointestinal bleeding, 58 had ascites, 37 had encephalopathy, 74 had jaundice, and 72 had coagulopathy. The median bilirubin level was 369.45 ± 290.77 μmol/L (range, 5.13 ± 1022.82 μmol/L), and the median prothrombin time was 26.6 ± 17.1 seconds (range, 12.6-141 seconds). Prior to LT, 48 children underwent plasmapheresis to treat hyperbilirubinemia.

The mean graft hepatic artery diameter was 2.4 ± 0.5 mm (range, 1.2-3.5 mm). All children except 11 received a blood transfusion (1.8 ± 2.1 U; range, 1-17 U) of erythrocyte suspensions. The median graft-to-recipient weight ratio was 2.5% ± 1.3% (range, 0.8%-6.1%). The mean cold ischemia time for living-donor grafts was 67 ± 17.1 minutes (range, 47-110 minutes), and for deceased-donor grafts, it was 7.5 ± 2.3 hours (range, 6-12 hours).

Biliary and vascular complications were among the most common complications in our series. Twelve children (11.5%) developed a hepatic arterial thrombosis (HAT), which was diagnosed with routine daily Doppler ultrasound examination. Among the 11 children with an HAT, 9 diagnoses were made before any elevation in the children's liver functioning tests. One of these 12 children had fever at the time of the HAT. Among these 12 children, 2 required reoperation for HAT. The remaining children were treated with interventional radiologic techniques such as intraluminal stent placement, balloon angioplasty, and continuous thrombolysis. Nine HATs (17.6%) occurred among the first 51 LTs, which had used a modified parachute technique for arterial reconstruction, and 3 HATs (5.6%) occurred among the last 53 LTs, which used the new hepatic arterial reconstruction technique. A clear mechanical cause for the HAT was identified in 6 children, all of whom were among the first 51 children. Stenoses were observed at the site of the anastomosis in 5 children, and from the beginning of the main hepatic artery in 1 child. In the remaining 6 children, no specific causative factor for the HAT could be identified. A thrombus, identified by angiography, was located at the site of the anastomosis in 9 children, at the multiple intrahepatic arterial branches in 2 children, and at the beginning of the main hepatic artery in 1 child. The clinical features of the HATs are shown in the Table. Moreover, 3 of the first 51 children developed a hepatic arterial stenosis and were treated with balloon angioplasty and intraluminal stent

Table. Characteristics of early hepatic arterial thromboses

Patient No.	Recipients (age in years; sex)	Type of graft	Time of HAT (days)	Site of HAT	Mechanical factor for HAT	Treatment of HAT	Follow-up	Patency of hepatic artery	Outcome
1	0.6, M	Whole	0	Anastomosis	No	Umbilical vein graft	48 mo	Yes	Alive
2	1.6, M	Left	4	Anastomosis	Stenosis	PTFE graft	2 mo	Yes	Dead
3	16, M	Right	12	Anastomosis	No	Thrombolysis+stent	42 mo	No	Alive
4	12, F	Right	1	Anastomosis	Stenosis	Thrombolysis+PTA	35 mo	Yes	Alive
5	14, F	Right	1	Anastomosis	No	Thrombolysis+stent	33 mo	Yes	Alive
6	3, F	Left lat	7	multiple	Stenosis	Continuous thrombolysis	22 mo	Yes	Alive
7	16, F	Left	5	Anastomosis	Stenosis	Thrombolysis+PTA	3 mo	Yes	Dead
8	0.6, F	Left lat	5	Anastomosis	No	Thrombolysis+stent	12 d	Yes	Dead
9	0.9, F	Left lat	5	Beginning of the main HA	Stenosis	Thrombolysis+stent	17 mo	Yes	Alive
10	13, F	Left	2	multiple	No	Continuous thrombolysis	8 d	No	Dead
11	0.6, F	Left lat	8	Anastomosis	No	Thrombolysis+stent	9 mo	Yes	Alive
12	2.5, M	Left lat	4	Anastomosis	No	Thrombolysis+stent	1 mo	Yes	Alive

Abbreviations: d, day; F, female; HA, hepatic artery; HAT, hepatic arterial thrombosis; left lat, left lateral segment; M, male; mo, month; PTA, percutaneous transluminal angioplasty; PTFE, polytetrafluoroethylene

placement. Five children with a HAT died during follow-up. Only 1 child died of a HAT 8 days after the HAT. The remaining 4 children died 57, 82, 13, and 32 days after the HAT. At the time of death, these 4 children had patent hepatic arteries and normal results on their liver function tests. A statistically significant relationship between HAT and mortality was not found ($P = .897$).

Hepatic vein stenoses developed in 2 children (1.9%) 1 and 14 months after the LTs. At the time of diagnosis, these children had ascites and elevated liver enzyme levels. Hepatic vein stenoses were treated with repeated balloon dilatation and intraluminal stent placement. Portal vein stenoses developed in 4 children (3.8 %) 8, 10, 11, and 14 months after LT. While 1 of these 4 children had massive ascites, an enlarged spleen, anemia, and thrombocytopenia, the remaining 3 children had no clinical signs or symptoms. These 3 portal vein stenoses were diagnosed during a follow-up examination. Children with portal vein stenoses all were successfully treated by percutaneous transhepatic balloon dilatation. There were no complications related to the treatment procedures. The children who had venous complications have not died during follow-up ($P = .387$).

Eleven children (10.5%) developed a biliary leak, and 9 children (8.6%) developed a biliary stenosis at the anastomotic site. All bile leaks except 1 were treated with a nonoperative procedure (percutaneous drainage) with excellent results. In 1 child, to treat the bile leak, a duct-to-duct anastomosis was converted to a hepaticojejunostomy 21 days after LT. All bile duct stenoses were also treated with repeated cholangioplasty with excellent results. Moreover, we observed minor catheter-related biliary leaks in 5 children (4.8%) and multiple intrahepatic bile duct stenoses with a transhepatic catheter in 4 (3.8%). All minor leaks were treated conservatively; multiple intrahepatic bile duct stenoses were treated with balloon cholangioplasty. In our series, there was no graft loss or short-term morbidity because of bile complications. Three children who had biliary complications died, but this result was not statistically significant ($P = .362$). Fifteen early re-operations were done in 14 children. Eight children were operated on for intra-abdominal bleeding, 2 for HATs, 1 for peptic ulcer perforation, 1 for a small bowel perforation, 1 for large bowel perforation, 1 for incisional dehiscence, and 1 for chylous ascites.

Twenty-seven (25.9%) infection complications occurred in our patients. Cytomegalovirus sepsis occurred in 7 children, Epstein-Barr virus sepsis occurred in 2, multidrug-resistant *Pseudomonas* sepsis occurred in 2, *Enterococcus* sepsis occurred in 5, *candida albicans* sepsis occurred in 1, and methicillin-resistant *staphylococcus aureus* occurred in 1. Moreover, pneumonia occurred in 3 children, urinary tract infection occurred in 2, tuberculosis occurred in 1, acute otitis media occurred in 1, *Varicella zoster* infection occurred in 1, and *Aspergillosis* occurred in 1. Of these 27 children, 9 (33.3%) died because of uncontrolled sepsis (1 with cytomegalovirus, 1 with Epstein-Barr virus, 4 with *Enterococcus*, 1 with *pseudomonas*, 1 with methicillin-resistant *staphylococcus aureus*, and 1 with candida).

A retransplant was done in 3 children. One who required retransplant 7 months after his first living-donor LT (LDLT) because of chronic rejection died of sepsis 10 days after his second transplant. The other child, who required retransplant 12 days after his first LDLT because of primary nonfunction of the graft, died of intracranial bleeding 76 days after his second transplant. The last one, who required retransplant 8 months after her first LDLT owing to chronic rejection, died of cardiac arrest 30 days after her second transplant. Ten children had concomitant hepatocellular carcinoma, and 2 children had hepatoblastoma. Hepatocellular carcinoma was detected incidentally in 3 of 9 children. During a mean follow-up of 22.2 ± 17.4 months (range, 1-71 months), 1 of the 9 children with hepatocellular carcinoma experienced a tumor recurrence in the omentum. This recurrence was treated surgically, and the child is alive at the time of this writing with good graft functioning. One child with hepatoblastoma developed a lymphoproliferative disorder 22 months after his LT, and died 2 months later owing to chemotherapy-related sepsis. Moreover, 3 children developed lymphoproliferative disorders 6, 27, and 17 months after their LTs, and were treated with chemotherapy; their tacrolimus treatment was changed to sirolimus.

Forty-five episodes of acute allograft rejection (42.3%) occurred in 35 children during the first 12 months after LT. Thirty-eight episodes were treated with steroid pulse therapy. The remaining 7 steroid-resistant episodes of acute rejection were treated with plasmapheresis, and antilymphocyte globulin. Chronic rejection developed in 2 children, and retransplant was required in these 2 children.

During the study, 16 children (15%) died of acute respiratory distress syndrome (n=2), brain death (n=1), primary nonfunction (n=1), sepsis with multiorgan failure (n=10), intracranial bleeding (n=1), and cardiac arrest (n=1). Fourteen of 16 deaths occurred during the 6 months after the LT, the remaining 2 deaths occurred subsequent to that. We did not find any statistically significant factors related with mortality. At the time of this writing, the remaining 85 children (85%) are alive and are experiencing good graft function. Overall 6-, 12-, and 36-month patient survival rates were 90%, 85%, and 83%.

Discussion

Technical advances in LT, including reduced-size transplant, split LT, use of living donors for partial LTs, and improved immunosuppressive therapies have allowed LT to become an effective therapeutic option for children with end-stage liver disease and acute liver failure (10, 11). Biliary atresia is the most common indication, and congenital metabolic disorders are the second most common indication for LT (12, 13). In our series, 24 of 101 children (23.7%) underwent LT for biliary atresia, and 29 children (28.7%) (16 with Wilson's disease, 5 with fulminant Wilson's disease, 4 with tyrosinemia, 1 with α-1 antitrypsin deficiency, and 3 with Crigler-Najjar syndrome type 1) underwent LT for metabolic disorders.

Biliary and hepatic arterial complications were the most common complications in our series. The use of reduced-size grafts with more-complicated surgery from living-related donors may increase the incidence of bile duct complications such as biliary stenosis and biliary leak (14). The presence of a stent in the biliary duct may lead to specific complications, which account for 30% to 60% of all biliary complications (15, 16). These complications include bile leakage around the stent, cholangitis after cholangiography, displacement of the stent, and biliary peritonitis after stent removal. Since September 2001, we have done 104 LTs in children from living or deceased donors. In 65 LTs, we used different drainage techniques such as a T tube, a straight feeding tube, or a transhepatic catheter. In these 65 children, our biliary complication rate was 26.9%. Thirty-five percent of these complications were directly related to the drainage catheters. Because of this high rate of catheter-related complications, after December 2006, we stopped using a catheter for bile duct reconstruction in LT. After December 2006, we did 39 LTs. Biliary leak occurred in 2 children, and a biliary stenosis occurred in 2.

HAT is a severe complication following LT. During the early postoperative period, it has a mortality rate of 50%. The incidence of early HAT has been reported to be 2.6% to 20% in adults, 9% to 14.9% in pediatric patients, and as high as 30% in children aged younger than 1 year (17). Previous studies have demonstrated that the risk of an HAT is increased in patients who weigh less than 10 kg, are younger than 3 years old, are female, have a graft with multiple arteries, and have a hepatic artery with a diameter of less than 2 mm (18, 19). Other risk factors for an HAT include multiple transplants, a recipient-to-donor weight ratio higher than 1.25, ABO incompatibility, technical complications during reconstruction, acute rejection episodes, and long cold ischemia times (17). In our children with HATs, 6 recipients were under the age of 3 years and weighed less than 10 kg. In 5 recipients, after thrombolysis, a stenosis at the site of the anastomosis was found. Owing to the high rate of these anastomotic stenoses in the first 51 LTs, we changed our hepatic arterial reconstruction technique to a wide anastomosis. In the last 54 LTs, we have seen only 3 instances of an early HAT.

The incidence of hepatic vein complications is relatively low, and Bull and associates reported that it was 4% in reduced-size or split, and 2% in living-related, grafts (20). Similarly, in our series, 2 children had a hepatic vein stenosis. The reported incidence of portal vein complications varies from 1.2% to 16.5% in pediatric patients undergoing LT (21, 22). There are 2 major problems in portal vein reconstruction in children. The vascular structure of a child with biliary atresia may often be impaired by previous surgery, and the child may have recurrent cholangitis. Another problem is the difference in the diameter of the portal vein between adults and infants. At our center, if there is a size discrepancy, the smaller-sized portal vein is spatulated from the anterior and posterior walls to make a wide anastomosis. In our series, we saw 4 (3.8%) portal vein stenoses. Children with venous complications were all successfully treated by interventional radiological techniques.

The size of the liver graft is an important consideration especially in small babies (each of whom weighed less than 10 kg or was under the age of 1 year). In most such cases, the left lateral segment is usually used. However, if the graft-to-recipient weight ratio is higher than 4%, infants who weigh less than 10 kg may require further reduction of the left lateral segment to a monosegment to overcome the weight discrepancy from donor to recipient. Some authors propose solutions that include the hyperreduction of liver grafts (monosegmental liver grafts) or delayed abdominal closure (23). At our center, for small babies, we use Doppler ultrasound to detect portal flow after abdominal closure. In our 22 children, the graft-to-recipient weight ratio was equal to or higher than 4%. Despite these rather large graft sizes, we were able to close our abdominal incisions primarily in all of these 22 small babies except 2. We did not encounter any abdominal compartment syndromes. Abdominal closure resulted in insufficient portal flow caused by pressure on the graft in 2 children. In these 2 children, the abdominal cavity was closed 3 and 19 days after LT. While only skin closure was used in first child, a Gore-Tex graft was used in the second one. The intraoperative course was smooth in these infants, and because the more complicated surgery required for the transplant of monosegment grafts may increase the complication rate (24), we did not reduce the graft volume.

Posttransplant lymphoproliferative disorder has a 5% to 7% prevalence rate on long-term follow-up following LT (25). Four patients (3.8 %) in our series had posttransplant lymphoproliferative disorder. Sixteen of the 101 children (15%) died during the study. The most common cause of death was uncontrolled sepsis (62.5%).

In conclusion, we realized that improved survival following LT may be related to developments in surgical techniques and immunosuppressive therapy. The overall outcomes of pediatric LT at our center are very promising. With improved care of younger children and the combined efforts of the parents and medical team, the number of the children receiving transplants will increase in the future. Unfortunately, in our study, only 8 children (7.6%) received a liver from a deceased donor. There is still a considerable shortage of deceased-donor grafts and at this point, living-donor LTs may overcome organ scarcity. Significant efforts continue to be made to educate the public about organ transplantation and donation.

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