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ORIGINAL ARTICLE

Comparative Analysis of Outcomes in Living and Deceased Donor Liver Transplants for Primary Sclerosing Cholangitis

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Abstract
Introduction Primary sclerosing cholangitits (PSC) is a progressive fibrosing cholangiopathy eventually leading to end-stage liver disease (ESLD). While literature for deceased donor liver transplantation (DDLT) for PSC abounds, only a few reports describe live donor liver transplant (LDLT) in the setting of PSC. We present a single-center experience on survival outcomes and disease recurrence for LDLT and DDLT for ESLD secondary to PSC.
Aim The aim of this study was to analyze survival outcomes and disease recurrence for LDLT and DDLT for ESLD secondary to PSC.
Patients and Methods A retrospective review of 58 primary liver transplants for PSC-associated ESLD, performed between May 1995 and January 2007, was done. Patients were divided into two groups based on donor status. Group 1 (n=14) patients received grafts from living donors, while group 2 (n=44) patients received grafts from deceased donors. An analysis of survival outcomes and disease recurrence was performed. Recurrence was confirmed based on radiological and histological criteria.
Results Recurrence of PSC was observed in four patients in LDLT group and seven in DDLT group. Retransplantation was required in one patient in LDLT group and nine patients in DDLT group. One patient (7%) among LDLT and six patients (14%) among DDLT died. The difference in patient and graft survival was not statistically significant between the two groups (patient survival, p=0.60; graft survival, p=0.24).
Conclusion This study demonstrates equivalent survival outcomes between LDLT and DDLT for PSC; however, the rate of recurrence may be higher in patients undergoing LDLT.

Keywords Liver transplant · Primary sclerosing cholangitis · Living donor · Deceased donor · Outcomes · Recurrence

Introduction

Primary sclerosing cholangitits (PSC) is a progressive fibrosing cholangiopathy characterized by inflammatory

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and fibrotic bile duct lesions forming multiple strictures and ectatic dilatations of the intra- and extrahepatic biliary system,[1–3] eventually leading to recurrent episodes of cholangitis and secondary fibrosis and cirrhosis. Mounting evidence now exists, which supports liver transplantation as the optimal treatment for decompensated liver disease with a 5-year graft survival in the range of 65.2% to 79%.[4–7] Recent studies based on deceased donor liver transplantation (DDLT) suggest that PSC can recur.[6–11] While literature for deceased donor liver transplantation for PSC abounds,[6–11] only a few reports describe live donor liver transplant (LDLT) in the setting of PSC-associated end-stage liver disease (ESLD).[12] Unlike with the DDLT population, the postoperative course in the LDLT group may be affected by the possible shared genetic background between the recipient and the donor, impacting long-term outcomes. It is unclear whether the outcome of LDLT is equivalent to or different from that of DDLT for PSC. We present a single-center experience on survival outcomes and disease recurrence for LDLT and DDLT for ESLD secondary to PSC.

Patients and Methods

A retrospective review was conducted of all primary liver transplants performed at our center from May 1995 to January 2007. Fifty-eight liver transplants were carried out for PSC-associated ESLD. Diagnosis of PSC was based on clinical signs and symptoms of jaundice, pruritus, and cholangitis, as well as the endoscopic retrograde cholangiopancreatography or transhepatic cholangiography findings of multiple strictures and dilatations of the intrahepatic and extrahepatic biliary ducts. The characteristic findings of PSC were further confirmed in the explant liver specimen with histologic sections showing overall bile duct loss, concentric and obliterative periductal fibrosis, and atrophy of ductal epithelium. In addition, other potential causes of progressive cholestatic liver disease including primary biliary cirrhosis (PBC), sarcoidosis, choledochal cysts, and chronic obstruction secondary to biliary stone disease were microscopically and grossly excluded upon pathologic examination. With a meticulous analysis of the radiographic and clinical data, the distinction between recurrent PSC and ischemic cholangiopathy was deliberated with all the available clinical, radiological, and biochemical evidence. All strictures were related to PSC recurrence and not to variant anatomy. Evidence of recurrence was further suggested by allograft biopsy showing a variety of bile duct alterations including epithelial damage, reduction in bile duct numbers, and in some circumstances background changes of an inflammatory infiltrate, portal edema, and cholangiolar proliferation.

Patients were divided into two groups based on donor status. Group 1 (n=14) comprised nine men and five women with a mean age of 44±12 years, who received grafts from living donors. Group 2 (n=44) consisted of 34 men and ten men with a mean age of 43±11 years, who received grafts from deceased donors. All living related donors underwent a pretransplant liver biopsy which was found to be microscopically normal and in particular negative for latent PSC. The mean Model for End-Stage Liver Disease (MELD) score was 12±5 in group 1 and 16±9 in group 2. The mean overall follow-up was 41.5±

Table 1 Patient Characteristics in LDLT vs. DDLT

	LDLT (n=14)	DDLT (n=44)	p value
Age	44±12 (median43)	43±11 (median42)	0.62
LOS	12±3 (median13)	25±27 (median11)	0.79
Males	9	34	
Females	5	10	
Race			
Caucasian	13	38	
African American	0	6	
Hispanic	1	0	
Blood group			
A	8	11	
B	0	3	
AB	0	2	
O	6	27	
Missing	0	1	
MELD score	12±5 (median10)	16±9 (median14)	0.25
Follow-up days	57.2±35.9	41.5±24.8	0.13

MELD Model for End-stage Liver Disease, *LOS* length of stay, *LDLT* live donor liver transplant, *DDLT* deceased donor liver transplant

24.8 months in group 1 and 57.2±35.9 months in group 2 (Table 1). In group 1, the mean duration to transplant after diagnosis was 57.8±42.2 months, and none of the patients had a colectomy pretransplant. The demographics of patients in group 1 are summarized in Table 2.

All hepatic resections in living donors were performed by a single surgeon with cavitron ultrasound surgical aspirator (Valley Lab, Boulder, CO, USA), unipolar electrocautery, liga clips, prolene sutures, and silk ties.

Statistical Analysis

Means of continuous variables were compared by *t* tests and correlations by Pearson's test. Categorical variables were compared by chi-square testing. Odds ratios were calculated using logistic regression. Statistical analysis was performed with SPSS Windows-based version 15.0 (SPSS, Chicago, IL, USA).

Results

Recurrence

Recurrence of PSC was observed in four patients in LDLT group and seven patients in DDLT group. Among recipients of living donor grafts, four patients experienced PSC recurrence as determined by radiological and histological criteria (Table 3). One patient had received the graft from spouse, and the remaining five patients had biologically related donors. The mean time to recurrence was 219 days in this patient who required retransplant (Table 2). This patient's cholangiogram showed diffuse beading and irregularity of the ducts (Fig. 1). Eventually, the graft was lost, with histologic confirmation of the diagnosis of PSC recurrence in the explant; the patient retransplanted and is now doing well.

Among deceased donor allograft recipients, seven patients developed recurrence (Table 3). The suspicion of recurrence was based initially on elevated liver function tests (LFTs) with a cholestatic picture and confirmed with cholangiography that demonstrated multiple intrahepatic biliary strictures. Evidence of recurrence was further confirmed by allograft biopsy. Three patients required retransplantation, two for recurrent disease, pathologically confirmed on explant examination, and one for a non-PSC type of biliary stricture. Of these three, one patient died 8 months after retransplant due to sepsis and multisystem organ failure. Of the other four, three were managed with percutaneous biliary drainage, and one did not require radiological intervention over a mean follow-up period of 77.3±19.0 months. One of the three patients requiring percutaneous drainage had only stenosis of hepatic duct at confluence on percutaneous transhepatic cholangiogram (PTC); however, the biopsy was suggestive of recurrent PSC. The remaining two patients had multiple intrahepatic bile duct strictures.

Out of the remaining 47 patients with no recurrence, 29 patients required a PTC for elevated liver function tests. Of these, 18 had a normal cholangiogram and 11 patients had biliary anastamotic strictures on cholangiogram.

Retransplant

Graft loss was defined as graft failure requiring retransplantation or as a result of death. Retransplantation was required in one patient in LDLT group and nine patients in DDLT group (Table 3). The living donor recipient who required retransplantation had graft failure related to recurrent PSC ($n=1$). Among deceased donors, retransplantation was required in nine patients for the following indications: hepatic artery thrombosis (HAT; $n=3$), recurrent PSC ($n=2$), primary nonfunction ($n=2$), hepatitis C viral infection ($n=1$), and non-PSC-related biliary stricture ($n=1$). Three patients with HAT required a retransplant 0.3, 0.8, and 1.5 months after primary transplant, respectively. Three of the nine patients who required retransplant died 60.9, 2.0, and 24.6 months after primary transplant due to sepsis and multisystem organ failure.

Survival

One patient (7%) among live donor recipients and six patients (14%) among deceased donor recipients died (Table 3). Amongst the former, the patient who died had developed refractory ascites after transplant and required the placement of a Denver shunt. The shunt later became infected, leading to removal of the stent followed by serial paracentesis and drain placement, resulting eventually in the death of the patient from liver failure, 36.7 months after primary transplant. Amongst the deceased donor recipient group, the most common cause of death was sepsis with multisystem organ failure ($n=4$). Out of these four patients, one developed PSC recurrence, for which the patient was retransplanted 16.4 months later but died of sepsis and multisystem organ failure 24.6 months after primary transplant.

One patient, who was found to have a co-existing cholangiocarcinoma at explant biopsy, developed abdominal wall metastases and died of metastatic cholangiocarcinoma 13.1 months after transplant. In another patient who passed away at home, the cause of death could not be ascertained.

Actuarial overall patient and graft survival at 1, 2, 3, and 5 years was 96%, 94%, 90%, 88%, and 89%, 87%, 83%, 81%, respectively (Fig. 2a). Actuarial patient survival at 1, 2, 3, and 5 years was 100%, 100%, 87%, and 87% for

Table 2 Demographics and Outcomes in LDLT

Case	Age	Sex	donor	Graft type	ABO	MELD score	LOS	Follow-up (months)	Pretransplant treatment	Warm Ischemia Time	Explant biopsy	Colectomy	Duration to Txp (months)	Recurrence (days)	Retransplant	Survival (days) graft	Survival (days) patient	Current status
1	40	M	Sister	Right lobe	A	6	11	7.1	No	0:53	Active cirrhosis	No	30.0	No	No	217	217	Alive
2	37	M	Brother	Right lobe	O	23	11	35.2	Yes	0:44	Active cirrhosis, adenocarcinoma (well differentiated) of gall bladder	No	60.0	Yes (400)	No	1,071	1,071	Alive
3	40	F	Mother	Right lobe	A	17	14	35.6	Yes	1:01	Active cirrhosis	No	168.0	No	No	1,084	1,084	Alive
4	57	F	Spouse	Right lobe	A	Pre-MELD	7	36.7	No	0:58	Active cirrhosis	No	84.0	Yes (219)	Yes	1,117	1,117	Alive
5	53	M	Spouse	Right lobe	A	16	16	55.9	No	0.36	Active cirrhosis	No	48.0	No	No	1,701	1,701	Died
6	62	M	Nephew	Right lobe	A	Pre-MELD	8	70.8	Yes	0:36	Active cirrhosis, dysplasia	No	36.0	No	No	2,156	2,156	Alive
7	57	F	Son	Right lobe	O	Pre-MELD	9	77.9	No	0:51	Active cirrhosis	No	60.0	No	No	2,373	2,373	Alive
8	59	M	Sister	Right lobe	O	7	15	12.6	No	0:41	Active cirrhosis	No	40.0	No	No	385	385	Alive
9	40	F	Son	Right lobe	A	Pre-MELD	16	65.7	No	0:33	Active cirrhosis severe dysplasia	No	36.0	Yes (540)	No	2,000	2,000	Alive
10	38	F	Brother	Right lobe	O	6	10	36.8	Yes	0:30	Fibrosis, chronic active hepatitis	No	72.0	Yes (1900)	No	1,120	1,120	Alive
11	19	M	Aunt	Right lobe	O	10	14	37.2	Yes	0:35	Active cirrhosis	No	36.0	No	No	1,134	1,134	Alive
12	28	M	Brother	Right lobe	A	10	14	0.9	Yes	0.45	Active cirrhosis	No	38.0	No	No	28	28	Alive
13	47	M	Spouse	Right lobe	O	12	17	32.6	Yes	0:54	Active cirrhosis	No	12.0	No	No	994	994	Alive
14	50	M	Son-in law	Right lobe	A	Pre-MELD	12	75.6	Yes	1:07	Active cirrhosis	No	24.0	No	No	2,268	2,268	Alive

MELD Model for End-Stage Liver Disease, *LOS* length of stay, *Txp* transplant, *M* male, *F* female

Table 3 Recurrence, Retransplant, and Death

	LDLT (n=14)	DDLT (n=44)	P value
Recurrence	4 (28%)	7 (16%)	0.29
Retransplant	1 (7%)	9 (20%)	0.25
Death	1 (7%)	6 (14%)	0.5
Retransplant			
Cause			
HAT	0 (0%)	3 (7%)	
Primary nonfunction	0 (%)	2 (5%)	
Recurrent PSC	1 (7%)	2 (5%)	
Biliary stricture	0 (0%)	1 (2%)	
Hepatitis C	0 (0%)	1 (2%)	
Total	1 (7%)	9 (20%)	
Death			
Sepsis	0 (0%)	4 (9%)	
Metastatic cholangiocarcinoma	0 (0%)	1 (2%)	
Unknown	0 (0%)	1 (2%)	
Hepatic failure	1 (7%)	0 (0%)	
Total	1 (7%)	6 (14%)	

LDLT live donor liver transplant, *DDLT* deceased donor liver transplant, *HAT* hepatic artery thrombosis, *PSC* primary sclerosing cholangitis

LDLT and 95%, 93%, 87%, and 87% for DDLT, respectively (Fig. 2b). Actuarial graft survival at 1, 2, 3, and 5 years was 100%, 100%, 87%, and 87% for LDLT and 86%, 84%, 78%, and 78% for DDLT, respectively (Fig. 2c). Difference in patient and graft survival was not statistically significant between the two groups (patient survival, p=0.60; graft survival, p=0.24).

Discussion

Liver transplant is the definitive treatment of complications from primary sclerosing cholangitis, namely recurrent cholangitis and liver failure. It has been shown that PSC is fraught with not only the risk of recurrence (as with PBC) but at an increased rate and at an earlier point than with other autoimmune processes. Its progressively fibrosing nature remains unamenable to any other form of therapy. DDLT for PSC is widely reported, while the literature on LDLT for PSC remains sparse.[13–17] The incidence of PSC recurrence in DDLT is approximately 20% (6–37%), diagnosed around 4 years after transplantation.[5,7–11,18–22] To date, few other studies have reported the outcome of LDLT for PSC from biologically related donors.[23–32] Yamigawa et al. reviewed 66 patients with PSC who underwent LDLT in Japan. The 5-year survival rate was 72%, and the rate of recurrence diagnosed on histological and cholangiographic findings was 25%.[12] Another report evaluated recurrence with a longer follow-up and a recurrence rate of 50%, when restricted to cases of biologically related live donors.[30] This series, though it presents with the longest follow-up period after LDLT for PSC described in the literature to date, being limited to nine patients, led the authors to conclude that the results obtained from their study have a large confidence interval, are prone to type 2 error, and would require confirmation by a larger series.

In our series, the overall rate of recurrence was 28%, and in biologically related live donors, it was 37%. However, none of the patients required retransplant. The patient who received graft from spouse was diagnosed with recurrent PSC and presented with typical radiologic images of non-anastomotic biliary strictures of the intrahepatic biliary tree with beading and irregularity (Fig. 1), which occurred 219 days post-LDLT which is consistent with recurrent PSC.

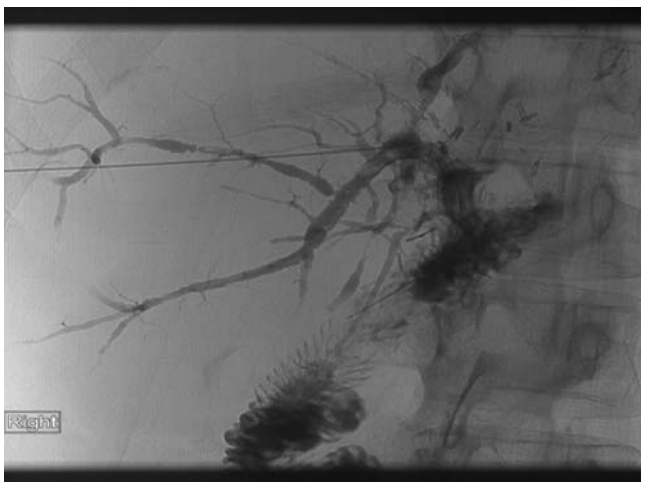

Figure 1 Cholangiogram in a LDLT recipient with PSC recurrence showing diffuse beading and irregularity of ducts.

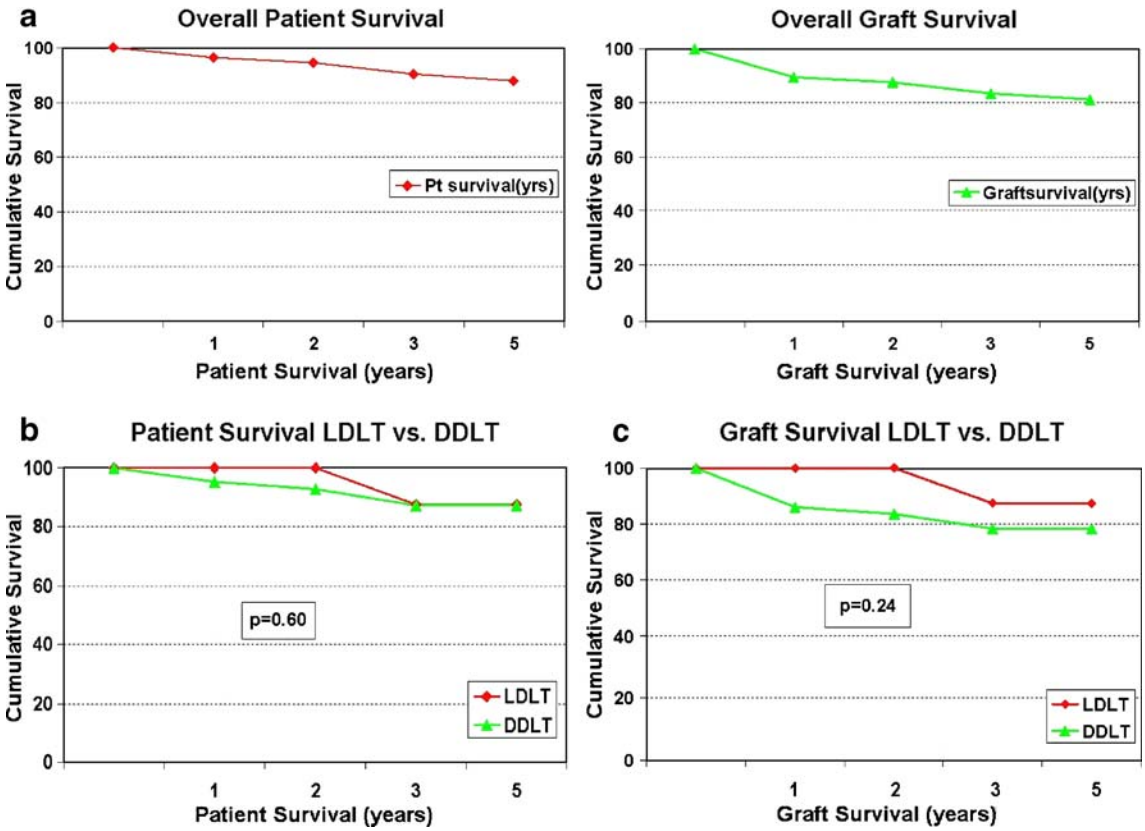

Figure 2 **a** Actuarial overall patient survival and actuarial overall graft survival. **b** Actuarial patient survival in LDLT vs. DDLT. **c** Actuarial graft survival in LDLT vs. DDLT.

While the precise etiology and pathogenesis of PSC remain unknown, the involvement of both immunologic as well as genetic factors has a strong but difficult to estimate influence.[33] An association between susceptibility to the development of PSC and human leukocyte antigen (HLA) gene complex was investigated by Tamura et al.[30] reporting the HLA-B8DR3 haplotype to be more common among PSC patients than among control patients, but this difference was not statistically significant with regard to recurrent PSC. In their series of nine cases of recurrent PSC among 49 PSC patients after DDLT, HLA-B8DR3 disparity did not seem to affect the outcome. Whether it is the associated HLA genes per se or some other closely linked genes that are responsible for the recurrence is yet to be determined; however, LDLT for PSC might offer a unique opportunity to examine the genetic aspects involved in disease recurrence. Current literature remains, at best, speculative with regards to a faster rate of recurrence with LDLT. This is being blamed on the hereditary commonality of donor and recipient as the association HLA B8 and PSC is recognized as is that of HLA DR2 and HLA DR3 haplotypes with PSC. Futugawa et al. have recently reported lower graft survival rates in PSC patients undergoing LDLT. Our study, being retrospective in nature, cannot account for a number of confounding factors which may influence the outcomes reported.

In conclusion, our study, though limited by its small sample size, demonstrates equivalent survival outcomes between patients who underwent DDLT or LDLT for PSC; however, the rate of recurrence may be higher in patients undergoing LDLT. In a majority of patients, this did not lead to graft loss or affect patient survival in our long follow-up period. The superior graft quality, as well as the favorable elective timing of LDLT, conferred marginally better patient and graft survival over DDLT in our analysis. Based on our results and those of others, we suggest doing a pooled analysis of data from different centers to develop a better understanding of the genetic aspects involved in disease recurrence.

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Conflicts of Interest None.

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