# ORIGINAL ARTICLE

# Nonsurgical policy for treatment of bilioenteric anastomotic stricture after living donor liver transplantation

Atsuyoshi Mita,<sup>1</sup> Yasuhiko Hashikura,<sup>2</sup> Yuichi Masuda,<sup>1</sup> Yasunari Ohno,<sup>1</sup> Koichi Urata,<sup>1</sup> Yuichi Nakazawa,<sup>1</sup> Toshihiko Ikegami,<sup>1</sup> Masaru Terada,<sup>1</sup> Hironori Yamamoto<sup>3</sup> and Shin-ichi Miyagawa<sup>1</sup>

1 Division of Transplantation, Department of Surgery, Shinshu University School of Medicine, Matsumoto, Japan

2 Transplantation Center, Shinshu University School of Medicine, Matsumoto, Japan

3 Division of Gastroenterology, Department of Internal Medicine, Jichi Medical School, Tochigi, Japan

#### Keywords

balloon dilatation, biliary complication, biliary stricture, double balloon endoscopy, hepatojejunostomy, living donor liver transplantation.

## Correspondence

Atsuyoshi Mita MD, Division of Transplantation, Department of Surgery, Shinshu University School of Medicine, 3-1-1 Asahi, Matsumoto 390-8621, Japan. Tel.: +81 263 37 2930; fax: +81 263 37 2930; e-mail: mita@hsp.md.shinshu-u.ac.jp

Received: 6 August 2007 Revision requested: 1 September 2007 Accepted: 10 November 2007

doi:10.1111/j.1432-2277.2007.00609.x

#### Summary

Biliary complications remain a significant cause of morbidity following living donor liver transplantation. The purpose of this retrospective study was to assess the outcome of nonsurgical management for hepatojejunostomy stricture in our institution. We reviewed 22 patients with hepatojejunostomy stricture among the 231 patients who underwent living donor liver transplantation between June 1990 and December 2005. Hepatojejunostomy stricture was confirmed by percutaneous transhepatic or endoscopic retrograde cholangiography. Anastomotic strictures were treated by balloon dilatation. Percutaneous transhepatic cholangiography was performed on 15 of the 22 patients. Two of 15 patients, with complete obstruction of the anastomosis, were treated successfully by Yamanouchi magnet compression anastomosis. Although another two patients died of infectious disease that was unlikely to have been related to biliary complications, anastomotic patency was maintained in the other 13 patients. Endoscopic retrograde cholangiography was performed on seven of the 22 patients. None of the 22 patients required re-operation or died of biliary complications. The 5-year graft survival rate of 85.6% in the 22 patients with stricture was equivalent to that of the patients without stricture (82.9%, P = 0.98). Advances in intervention techniques have enabled wider application of nonsurgical approaches for this complication, and fair results have been obtained.

# Introduction

Biliary complications after liver transplantation are common and remain an important cause of morbidity, despite various advances in surgical techniques. The incidence of such complications is reportedly higher in living donor liver transplantation (LDLT), affecting approximately 20–30% of recipients, than in cadaveric liver transplantation [1]. In most cases, this complication involves stricture of the biliary anastomosis along with biliary leakage [2]. In the early days of LDLT, the treatment for most biliary complications was considered to be surgical repair, and patency rates at 1 year after open revision with hepatojejunostomy (HJ) ranged from 80% to 90% [2–4]. The disadvantages of surgery include the morbidity of an open procedure, and many patients with strictures might not be ideal surgical candidates because of associated complications or frequent episodes of cholangitis [5]. Currently, bilioenteric anastomotic strictures are conventionally managed by percutaneous transhepatic dilatation, ballooning and/or stenting through the use of interventional radiology techniques [2,6]. Thus, over the last decade, treatment modalities for bilioenteric anastomotic strictures have changed toward a primarily nonsurgical strategy, leaving surgical intervention for lesions that would otherwise not be curable [7].

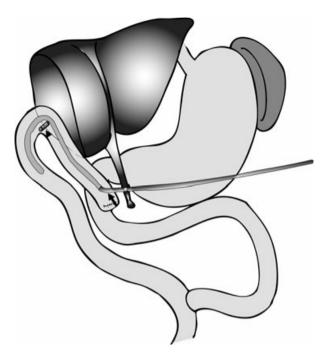
Endoscopic balloon dilatation is a useful treatment for stricture of choledochocholedochostomy, but not for stricture of HJ, because insertion of an endoscope through the afferent loop of a Roux-en-Y reconstruction is usually difficult [8]. However, a new enteroscopy insertion method, the double-balloon method, enables access to the entire small intestine including the afferent loop of a Roux-en-Y reconstruction [9,10].

In Japan, the law related to brain death was not enacted before 1997, and since then the number of cadaveric donors has been extremely limited. These circumstances have prompted us to make great efforts to avoid graft loss and re-transplantation. In principle, nonsurgical treatment is our main choice for HJ stricture after LDLT, and surgical treatment is chosen for HJ stricture only when it occurs in the early period after LDLT. Up to now, we have experienced no cases requiring surgical treatment. The purpose of the present study was to assess the outcomes of nonsurgical treatments for HJ stricture after LDLT.

# **Patients and methods**

We retrospectively reviewed the medical records of 22 patients (pediatric 6, adult 16) with bilioenteric anastomotic stricture from among the 231 patients (pediatric 110, adult 121) who underwent LDLT with HJ reconstruction at Shinshu University Hospital between June 1990 and December 2005. Considering the safety of the donors as well as the favorable results that had been obtained at our institution [11,12], we used the left lobe (n = 133) or left lateral segment (n = 93), except for four right-lobe grafts and one right lateral sector graft [13].

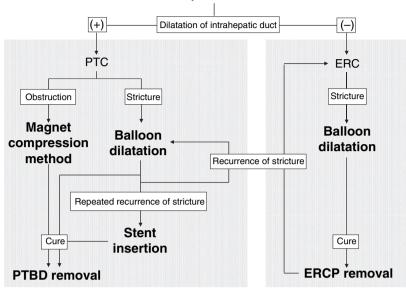
Hepatojejunostomy with Roux-en-Y anastomosis was performed using interrupted sutures of 4–0 absorbable braid (Vicryl, Ethicon, Somerville, NJ, USA) or 5–0 polydioxanone absorbable monofilament (PDS II, Ethicon) and stented internally with a short plastic tube (7-Fr. to 12-Fr. diameter and 2 cm to 3 cm in length). A tube jejunostomy (16-Fr PHYCON drain, Fuji Systems Corporation, Tokyo, Japan, or 16-Fr naso-gastric catheter, TERUMO, Tokyo, Japan) was placed on the afferent limb of the Roux-en-Y reconstruction for intra-bowel decompression (Fig. 1). The end of the afferent limb was sutured to attach upon the parietal wall. The tube was removed 3 months after transplantation after confirming that liver function had normalized.



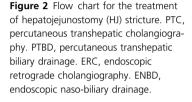
**Figure 1** Hepatojejunostomy with a Roux-en-Y anastomosis was performed using an internal stent (arrow head). A tube jejunostomy was placed on the afferent limb of the Roux-en-Y reconstruction to decrease intra-bowel pressure, passing through the 20 cm in length efferent limb with a longitudinal Witzel (arrow).

The presence of HJ stricture, as revealed by the results of liver function tests suggesting an obstructive pattern and/or intrahepatic bile duct dilatation on ultrasonography, performed frequently during hospitalization and routinely at the outpatient clinic, was confirmed by either percutaneous transhepatic (PTC) or endoscopic retrograde (ERC) cholangiography (Fig. 2). PTC was selected only when intrahepatic biliary dilatation was apparent. Otherwise, ERC was performed by narrow-diameter endoscopy through the tube jejunostomy or by double balloon endoscopy via the oral approach.

Our treatment strategy for biliary stricture is shown in Fig. 2. HJ strictures with intrahepatic biliary dilatation were treated initially by balloon dilatation using 8-mm-diameter balloons (Ultra-thin diamond catheter, Boston Scientific Japan, Tokyo, Japan) in the PTC group, followed by placement of a drainage catheter (8-Fr to 14-Fr PTC drainage tube; Sumitomo Bakelite Company Limited, Tokyo, Japan) through the stricture by means of a percutaneous transhepatic biliary drainage tube. After confirming resolution of the stricture on follow-up cholangiography, the percutaneous transhepatic biliary drainage catheter was withdrawn so that its tip was located just proximal to the initial stricture site, and then removed when normal liver function had been



Suspicion of HJ stricture fever, obstructive pattern of liver function test



maintained for 3 months. Balloon dilatations were repeated for recurrent narrowing of the anastomosis on follow-up cholangiography. When repeated balloon dilatation could not resolve the stricture, the 8-Fr stent tube (Hakko medical, Ueda, Japan) was placed across the stricture for 3 months. In such cases, a percutaneous transhepatic biliary drainage tube was placed in the intrahepatic duct for 3 months after removing the stent. When patients had complete obstruction of the anastomosis, Yamanouchi magnet compression anastomosis was performed [14,15].

As shown in Fig. 2, HJ strictures without biliary dilatation were treated initially by ERC using double balloon endoscopy (EN450-T5 or EC450-BI5; Fujinon, Saitama, Japan) and balloon dilatation with 6-mm-diameter balloons (Max Force; Boston Scientific Japan), followed by placement of a drainage catheter (PD-SS6.0F, CATHEX Co. Ltd., Kanagawa, Japan) through the stricture via the endoscopic naso-biliary drainage tract (Figs 2 and 3), as reported previously [16]. The catheter of the endoscopic naso-biliary drainage was removed after tests had confirmed improvement of liver function several days after balloon dilatation on account of difficulty with long-term placement. When laboratory data suggested re-stricture of the HJ anastomosis, ERC was performed for repeat balloon dilatation.

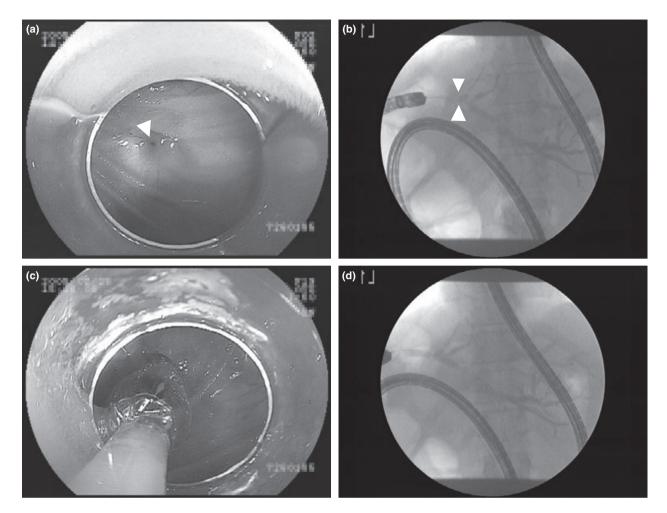
Particularly in respect of patients having HJ stricture without biliary dilatation in the early postoperative course (within 3 months), ERC was performed using narrowdiameter endoscope (P-10, Olympus Medical Systems Corp., Tokyo, Japan) through the tube jejunostomy gradually dilated up to 24-Fr using dilators (Sumitomo Bakelite Co. Ltd.) and drainage tubes (PHYCON drain; Fuji Systems Corporation). HJ strictures were treated by balloon dilatation using 8-mm-diameter balloons (Ultrathin diamond catheter, Boston Scientific Japan), followed by insertion of a tube jejunostomy near the anastomosis instead of a biliary drainage tube, which is difficult to place through the efferent limb of the jejunum. The tube jejunostomy was removed when normal liver function had been maintained for 3 months.

Successful treatment of the HJ stricture was defined as resolution of the clinical symptoms, liver function test abnormalities, and intrahepatic biliary dilatation for 3 months.

Data were expressed as mean  $\pm$  standard deviation, and statistical analyses were performed using StatView software (Version 5.0, SAS Institute Inc., Cary, NC, USA). The log rank test was used to compare the Kaplan–Meier curves for graft survival. Differences at P < 0.05 were considered statistically significant. Written informed consent for the procedures was obtained from each of all of the patients.

# Results

The characteristics of the patients, indications for transplantation and graft types are presented in Table 1. Follow-up duration from treatment of HJ stricture to the last follow-up was  $38.0 \pm 35.8$  months. The time interval between liver transplantation and the onset of HJ stricture was  $6.0 \pm 8.9$  months.



**Figure 3** (Case 176) An 18-year-old man developed hepatojejunostomy stricture at 4.6 months after living donor liver transplantation. (a, b) Endoscopic retrograde cholangiography by double balloon endoscopy revealed narrowing of the anastomosis (arrow heads). (c, d) Balloon dilatations were performed twice followed by patency of the anastomosis for 4 months. The period required for treatment was 8.2 months.

PTC was performed on 15 of 22 patients with HJ stricture (Fig. 2). Of these patients, two died; one on account of Pneumocystis carinii pneumonia and the other because of bacterial pneumonia before completion of the treatment for biliary stricture. In another two patients, PTC showed complete obstruction at the HJ anastomosis. The number of repeated balloon dilatations was  $4.6 \pm 3.1$  (Table 1). Anastomotic patency was maintained exclusively by balloon dilatation in five patients. Another six patients additionally required insertion of a stent tube across the anastomosis, and the duration of stent placement was  $5.4 \pm 3.7$  months. In the latter six patients, anastomotic patency was maintained after removing the stent tube. However, one of them, who received an ABO-incompatible graft, died on account of pulmonary edema (Case 20). The total period required for treatment in the PTC group was  $13.5 \pm 11.7$  months.

Endoscopic retrograde was performed on seven of the 22 patients; narrow-diameter endoscopy was used in two patients and double balloon endoscopy in five (Table 1, Figs 2 and 3). Balloon dilatation of the stricture was repeated  $2.0 \pm 1.0$  times and anastomotic patency was maintained for  $13.3 \pm 10.5$  months after the latest dilatation. The duration of treatment was  $2.5 \pm 3.3$  months in the ERC group.

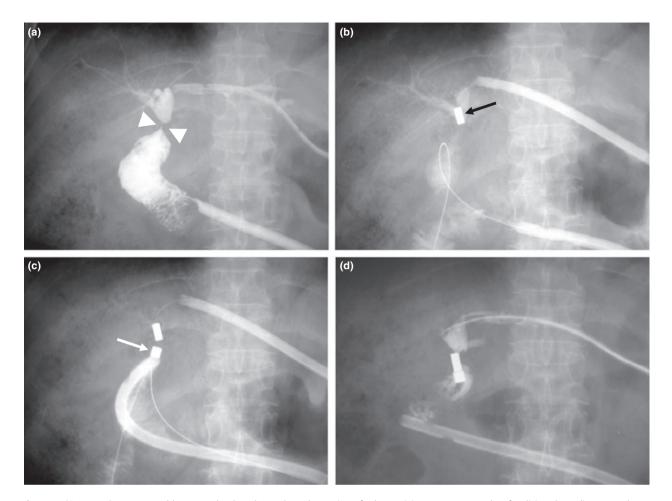
Stones in the intrahepatic biliary duct were detected in three patients (Table 1). The interval between transplantaappearance of tion until the stones was 17.7  $\pm$  26.1 months. Two of these patients had HJ stricture and the other (Case 123) developed intrahepatic stones 3.5 years after magnet compression anastomosis. Two (Cases 123, 180) of the three patients with intrahepatic stones were treated successfully by extracorporeal shock-wave lithotripsy with endoscopic naso-biliary drainage insertion (Table 1). Extracorporeal shock-wave

						since the heainning	Unset after	stricture					Duration	Patency after	
$ \begin{array}{cccccccccccccccccccccccccccccccccccc$		Age at LDLT	Gender	Reason for LDLT	Graft	of treatment (months)	LDLT (months)		age		pproach	Treatment (times)	treatment (months)	treatment (months)	Outcome
	20	m	Σ	BA	LS	18.6	3.7		+		TC	BD(4), St, PLT	18.2	0.5	Died of lung edema
$ \begin{array}{cccccccccccccccccccccccccccccccccccc$			1		:		•			i			1		18.6 months later
523         M         PEC         L         1038         15.7         +         PTC         B0(d), St         5.7         981           18         M         RAP         L         1013         4.9         +         PTC         B0(d), St         5.7         981           10         F         BA         L         1013         4.9         +         PTC         B0(d), St         5.7         981           10         F         BA         L         1013         4.9         +         PTC         B0(d), St         5.7         981           10         F         BA         L         10         3.8         0.7         +         PTC         B0(d), St         6.4         73.4           0.5         F         L(C(typel)         L         10         7.2         1.1         2.2         MA           53.1         M         HCC, L(typel)         L         17.3         Unclear         F         PTC         B0(2), MCA, ESW(19)         4.7         6.2         2.2           53.1         M         HCC, L(typel)         L         17.3         Unclear         F         PTC         B0(2), MCA, ESW(19)         4.7         62.2	23	60.0	ц.	PBC		98.8	44.4			<u>م</u>	2	BD(8), St	18.5	80.4	Cured
45.         M         FAP         LI         1013         4.9         PTC         BO(2), S1         0.2         101.1           1.8         M         BA         L         1013         4.9         PTC         BO(2), S1         0.2         36.4           1.8         M         BA         L         9.88         0.7         +         PTC         BO(3)         19.2         73.4           1.0         F         Citulinemia         L         5.8         2.0         +         PTC         BO(3)         19.2         73.4           0.5         F         HF         L         7.9         7.9         +         PTC         BO(3)         19.2         73.4           0.5         F         HF         L         1.0         7.9         7.9         1.1         73.4           0.5         F         LC         LC         L         1.1         2.2         NA           53.1         M         HCC, LC(typec)         L         17.3         Unclear         +         PTC         BD(3), S1         6.4         73.4           53.1         M         HCC, LC(typec)         L         17.3         Unclear         +	55	52.3	Σ	PBC	╘	103.8	15.7	+		٩	12	BD(4), St	5.7	98.1	Cured
$ \begin{array}{cccccccccccccccccccccccccccccccccccc$	65	45.5	Σ	FAP	Ц	101.3	4.9			'n_	TC	BD(2), St	0.2	101.1	Cured
$ \begin{array}{cccccccccccccccccccccccccccccccccccc$	73	1.8	Σ	BA	LS	36.8	12.0	+		ш	RC(DBE)	BD(1)	0.5	36.4	Cured
$ \begin{array}{cccccccccccccccccccccccccccccccccccc$	83	24.9	щ	Citrullinemia	H	98.8	0.7			Ē	TC	BD(3)	19.2	79.6	Cured
0.5         F         HH         IS         79.7         I.5         79.7         I.5         79.7         I.5         73.4           45.8         F         LC(typeB)         L1         708         7.9         7.9         7.9         73.4         73.4           59.4         F         LC         LC         L1         7.08         7.9         7.9         73.4           53.1         M         HCC, LC(typeB)         L1         2.2         1.1         2.2         NA           63.4         F         BA         L2         L1         2.2         NA         64.0           63.1         M         HCC, LC(typeB)         L1         1.2         0.7         +         +         PTC         -         2.2         NA           63.8         M         HCC, LC(typeB)         L1         1.8         6.8         +         PTC         -         2.2         NA           63.8         M         HCC, LC(typeB)         L1         1.8         PTC         -         2.9         42.3           63.9         F         BA         HCC         HC(typeB)         LC(typeB)         1.1         1.8         7.3	97	1.0	щ	BA	LS	86.6	2.7	+		Ē	TC	BD(2)	2.1	84.5	Cured
45.8       F       LC(typeB)       L1       70.8       7.9       +       PTC       B0(4), St       6.8       64.0         59.4       F       LC       LL       1.1       2.2       1.1       7.2       NA         59.4       F       LC(typeC)       L1       2.2       1.1       2.2       NA         53.1       M       HCC, LC(typeC)       L1       17.3       Unclear       -       2.2       NA         53.1       M       HCC, LC(typeC)       L1       17.3       Unclear       -       2.2       NA         63.3       M       HCC, LC(typeB)       L1       1.3       Unclear       +       +       PTC       B0(5)       2.9       42.3         63.3       F       BA       L5       4.3       6.8       +       +       PTC       2.9       42.3         63.4       F       BA       LC       1.1       1.8       6.8       4.2       42.3         65.4       M       HCC, LC(typeB)       L1       1.8       6.8       4.4       9.0         18.8       M       Allagille syndrome       L1       1.5       4.3       5.6       4.4	104	0.5	щ	FHF	LS	79.7	1.5			Ē	TC	MCA	6.4	73.4	Cured
59.8       F       LC       LL       66.8       5.0       +       PTC       BD(2), MCA, ESWL(9)       4.7       62.2         59.4       F       LC(typeC)       LL       2.2       1.1       -       2.2       NA         53.1       M       HCC, LC(typeC)       LL       17.3       Unclear       -       2.2       NA         0.8       F       BA       LS       45.2       0.7       +       +       PTC       BD(2)       8.3       9.0         0.8       F       BA       LS       45.2       0.7       +       +       PTC       BD(5)       2.9       47.3       62.3         0.8       F       BA       LS       45.8       +       +       PTC       BD(5)       2.9       42.3         0.8       M       HCC, LC(typeB)       LL       1.18       6.8       +       PTC       BD(1)       5.9       42.3         5.4       F       BA       LS       4.87       4.8       +       PTC       BD(1)       5.9       7.0         5.3       F       PBC       LL       11.5       5.2       5.2       42.3       5.4         5	108	45.8	щ	LC(typeB)	H	70.8	7.9			Ē	TC	BD(4), St	6.8	64.0	Cured
69.4       F       LC(typeC)       LL       2.2       1.1       2.2       NA         53.1       M       HCC, LC(typeC)       LL       17.3       Unclear       2.2       9.0         53.1       M       HCC, LC(typeC)       LL       17.3       Unclear       2.2       9.0         0.8       F       BA       LS       45.2       0.7       +       +       7.2       8.3       9.0         0.8       F       BA       LS       45.2       0.7       +       +       7.3       9.0         0.8       F       BA       LS       45.2       0.7       +       +       7.3       9.0       42.3         63.8       M       HCC, LC(typeB)       LL       1.8       6.8       +       +       7.7       2.9       42.3         63.9       F       PRC       LL       1.1       1.8       6.7       5.2       42.3         63.9       F       PRC       LC       B.0(1)       1.1       8.7       5.2       10.1         18.8       M       Allagille syndrome       LL       11.5       5.2       10.1         66.4       M       LC(type	123	59.8	щ	LC	⊣	66.8	5.0		+		TC	BD(2), MCA, ESWL(9)	4.7	62.2	Cured
53.1       M       HCC, LC(typeC)       Ll       17.3       Unclear       8.3       9.0         0.8       F       BA       LS       45.2       0.7       +       +       PTC       BD(S)       2.9       42.3         0.8       F       BA       LS       45.2       0.7       +       +       PTC       BD(S)       2.9       42.3         63.8       M       HCC, LC(typeB)       LL       1.8       6.8       +       +       PTC       BD(S)       2.9       42.3         63.8       M       HCC, LC(typeB)       LL       1.8       6.8       +       +       PTC       BD(13), St       40.1       8.7         5.4       F       PBC       LL       11.5       5.2       FRC(DBE)       BD(13), St       40.1       8.7         63.9       F       PBC       LL       11.5       5.2       FRC(DBE)       BD(13), St       40.1       8.7         18.8       M       Allagille syndrome       LL       15.9       5.6       +       FRC(DBE)       BD(1), ESWL(3)       0.5       15.4         59.9       M       LC(typeC)       LL       15.9       7.0       24.4	127	69.4	ш	LC(typeC)	Ľ	2.2	1.1			Ē	TC	I	2.2	NA	Died of pneumonia
53.1       M       HCC, LC(typeC)       Ll       17.3       Unclear       ERC(DBE)       BD(2)       8.3       9.0         0.8       F       BA       LS       45.2       0.7       +       +       PTC       BD(5)       2.9       42.3         0.8       F       BA       LS       45.2       0.7       +       +       PTC       BD(5)       2.9       42.3         63.8       M       HCC, LC(typeB)       Ll       1.8       6.8       +       PTC       BD(1)       2.9       42.3         5.4       F       BA       LS       48.7       4.8       +       PTC       ERC(DBE)       BD(1)       5.9       42.3         63.9       F       PBC       LL       11.5       5.2       ERC(DBE)       BD(2)       1.5       10.1         18.8       M       Allagille syndrome       LL       15.1       4.6       +       FRC(DBE)       BD(1), ESVL(3)       0.5       15.4         59.9       M       LC(typeC)       LL       15.9       5.6       +       PTC       BD(4)       5.0       7.0       24.4         58.5       M       HCC, LC(typeB)       LL															68 days later
0.8       F       BA       LS       45.2       0.7       +       +       PTC       BD(5)       2.9       42.3         63.8       M       HCC, LC(typeB)       Ll       1.8       6.8       +       +       PTC       2.9       42.3         5.4       F       BA       LS       48.7       4.8       +       PTC       -       1.8       NA         5.4       F       BA       LS       48.7       4.8       +       PTC       BD(13), St       40.1       8.7         63.9       F       PBC       LL       11.5       5.2       ERC(DBE)       BD(2)       1.5       10.1         18.8       M       Allagille syndrome       LL       15.9       5.6       +       +       ERC(DBE)       BD(1), ESWL(3)       0.5       15.4         59.9       M       LC(typeC)       LL       15.9       5.6       +       PTC       BD(4)       7.0       24.4         58.5       M       HC, LC(typeB)       LL       15.9       7.0       24.4         39.0       F       FAP       LL       15.6       7.0       24.4         59.0       F       AP	141	53.1	Σ	HCC, LC(typeC)	Ľ	17.3	Unclear			ш	RC(DBE)	BD(2)		9.0	Cured
63.8       M       HCC, LC(typeB)       Ll       1.8       6.8       +       PTC       -       1.8       NA         5.4       F       BA       LS       48.7       4.8       F       PTC       BD(13), St       40.1       8.7         63.9       F       PBC       LL       11.5       5.2       ERC(DBE)       BD(2)       1.5       10.1         18.8       M       Allagille syndrome       LL       15.1       4.6       +       ERC(DBE)       BD(2)       1.5       7.0         66.4       M       LC(typeC)       LL       31.3       3.5       +       FRC(DBE)       BD(1), ESVL(3)       0.5       15.4         58.5       M       HCC, LC(typeB)       LL       15.9       5.6       +       FRC(DBE)       BD(1), ESVL(3)       0.5       15.4         58.5       M       HCC, LC(typeB)       LL       15.9       7.0       24.4         58.5       M       HCC, LC(typeB)       LL       12.6       4.4       7.0       24.4         59.0       F       FAP       LL       12.6       4.4       7.0       5.9       7.0	143	0.8	ш	BA	LS	45.2	0.7	+		Ē	TC	BD(5)		42.3	Cured
5.4       F       BA       LS       48.7       4.8       PTC       BD(13), St       40.1       8.7       6         63.9       F       PBC       LL       11.5       5.2       ERC(DBE)       BD(2)       1.5       10.1       0         18.8       M       Allagille syndrome       LL       11.5       5.2       ERC(DBE)       BD(2)       1.5       10.1       0         66.4       M       LC(typeC)       LL       15.9       5.6       +       ERC(DBE)       BD(1), ESWL(3)       0.5       7.2       0         59.9       M       LC(typeC)       LL       15.9       5.6       +       ERC(DBE)       BD(1), ESWL(3)       0.5       7.12       0       7.0       24.4       0         58.5       M       HCC, LC(typeB)       LL       15.9       1.0       +       ERC(NDE)       BD(4)       7.0       24.4       0         39.0       F       FAP       LL       12.6       4.4       PTC       BD(3)       6.7       5.9       0       7.0       0       7.0       0       7.0       0       7.0       0       7.0       0       7.0       0       7.0       0       <	146	63.8	Σ	HCC, LC(typeB)	Ц	1.8	6.8	+		Ē	TC	I	1.8	NA	Died of Pneumocystis
5.4     F     BA     LS     48.7     4.8       63.9     F     PBC     LL     11.5     5.2     ERC(DBE)     BD(13), St     40.1     8.7     0       63.9     F     PBC     LL     11.5     5.2     ERC(DBE)     BD(2)     1.5     10.1     0       18.8     M     Allagille syndrome     LL     15.1     4.6     ERC(DBE)     BD(2)     1.5     10.1     0       66.4     M     LC(typeC)     LL     15.9     5.6     +     ERC(DBE)     BD(1), ESWL(3)     0.5     15.4     0       59.9     M     LC(typeC)     LL     15.9     5.6     +     ERC(NDE)     BD(1), ESWL(3)     0.5     15.4     0       58.5     M     HCC, LC(typeB)     LL     15.9     1.0     +     ERC(NDE)     BD(4)     7.0     24.4     0       39.0     F     FAP     LL     12.6     4.4     +     PTC     BD(3)     6.7     5.9     0.0															carinii pneumonia
5.4         F         BA         LS         48.7         4.8         PTC         BD(13), St         40.1         8.7           63.9         F         PBC         LL         11.5         5.2         ERC(DBE)         BD(2)         1.5         10.1         8.7           18.8         M         Allagille syndrome         LL         11.5         5.2         ERC(DBE)         BD(2)         1.5         10.1           18.8         M         LC(typeC)         LL         15.9         5.6         +         ERC(DBE)         BD(1), ESWL(3)         0.5         7.2           66.4         M         LC(typeC)         LL         15.9         5.6         +         ERC(DBE)         BD(1), ESWL(3)         0.5         15.4           59.9         M         LC(typeC)         LL         13.3         3.5         PTC         BD(4)         7.0         24.4           58.5         M         HCC, LC(typeB)         LL         12.6         4.4         PTC         BD(4)         7.0         24.4           39.0         F         FAP         LL         12.6         4.4         PTC         BD(3)         6.7         5.9															56 days later
63.9         F         PBC         LL         11.5         5.2         ERC(DBE)         BD(2)         1.5         10.1           18.8         M         Allagille syndrome         LL         15.1         4.6         ERC(DBE)         BD(2)         1.5         10.1           66.4         M         LC(typeC)         LL         15.9         5.6         +         ERC(DBE)         BD(1), ESWL(3)         0.5         7.2           59.9         M         LC(typeC)         LL         31.3         3.5         +         ERC(DBE)         BD(1), ESWL(3)         0.5         7.4           58.5         M         HCC, LC(typeB)         LL         15.9         1.0         +         ERC(NDE)         BD(4)         7.0         24.4           58.5         M         HCC, LC(typeB)         LL         15.9         1.0         +         ERC(NDE)         BD(4)         7.0         24.4           39.0         F         FAP         LL         12.6         4.4         PTC         BD(3)         6.7         5.9	160	5.4	ш	BA	LS	48.7	4.8			Ē	TC	BD(13), St	40.1	8.7	Cured
18.8         M         Allagille syndrome         LL         15.1         4.6         ERC(DBE)         BD(2)         8.0         7.2           66.4         M         LC(typeC)         LL         15.9         5.6         +         ERC(DBE)         BD(1), ESWL(3)         0.5         15.4           59.9         M         LC(typeC)         LL         31.3         3.5         PTC         BD(4)         7.0         24.4           58.5         M         HCC, LC(typeB)         LL         15.9         1.0         +         ERC(NDE)         BD(4)         7.0         24.4           39.0         F         FAP         LL         12.6         4.4         PTC         BD(4)         5.0         7.0	173	63.9	щ	PBC	H	11.5	5.2			ш	RC(DBE)	BD(2)	1.5	10.1	Cured
66.4         M         LC(typeC)         LL         15.9         5.6         +         ERC(DBE)         BD(1), ESWL(3)         0.5         15.4           59.9         M         LC(typeC)         LL         31.3         3.5         +         PTC         BD(4)         7.0         24.4           58.5         M         HCC, LC(typeB)         LL         15.9         1.0         +         ERC(NDE)         BD(4)         7.0         24.4           39.0         F         FAP         LL         12.6         4.4         PTC         BD(3)         6.7         5.9		18.8	Σ	Allagille syndrome	H	15.1	4.6			ш	RC(DBE)	BD(2)	8.0	7.2	Cured
59.9         M         LC(typeC)         LL         31.3         3.5         PTC         BD(4)         7.0         24.4           58.5         M         HCC, LC(typeB)         LL         15.9         1.0         +         ERC(NDE)         BD(4)         9.0         7.0         24.4           39.0         F         FAP         LL         12.6         4.4         PTC         BD(3)         6.7         5.9		66.4	Σ	LC(typeC)	H	15.9	5.6		+		RC(DBE)	BD(1), ESWL(3)	0.5	15.4	Cured
58.5 M HCC, LC(typeB) LL 15.9 1.0 + ERC(NDE) BD(4) 9.0 7.0 39.0 F FAP LL 12.6 4.4 PTC BD(3) 6.7 5.9		59.9	Σ	LC(typeC)	H	31.3	3.5			Ē	TC	BD(4)	7.0	24.4	Cured
39.0 F FAP LL 12.6 4.4 PTC BD(3) 6.7 5.9		58.5	Σ	HCC, LC(typeB)	H	15.9	1.0	+		ū	RC(NDE)	BD(4)	9.0	7.0	Cured
		39.0	ш	FAP	Ľ	12.6	4.4			Ē	TC	BD(3)	6.7	5.9	Cured
65.6 F LC(typeC) LL 14.7 0.6 ERC(NDE) BD(2) 6.4	220	65.6	ш	LC(typeC)	H	14.7	0.6			ш	RC(NDE)	BD(2)	6.4	8.3	Cured

Table 1. Characteristics and outcomes of patients with hepatojejunostomy stricture.

© 2007 The Authors

Journal compilation © 2007 European Society for Organ Transplantation **21** (2008) 320–327



**Figure 4** (Case 123) A 59-year-old woman developed complete obstruction of a hepatojejunostomy 5 months after living donor liver transplantation. She was treated successfully by Yamanouchi magnet compression anastomosis. (a) Contrast medium injected through the biliary drainage tube and through the tube jejunostomy showed obstruction of the hepatojejunostomy (arrow head). (b) The daughter magnet (black arrows) was inserted into the intrahepatic duct through the percutaneous transhepatic biliary drain. (c) The parent magnet (white arrow) was placed near the anastomosis in the afferent loop of the jejunum through the tube jejunostomy under fluoroscopic guidance. (d) The two magnets were allowed to attract each other transmurally to create a fistula between the intrahepatic duct and the afferent limb of the jejunum. A bilioenteric fistula was established 25 days after this procedure. The patient developed a stone in the intrahepatic biliary duct 3.5 years after the procedure, and was treated successfully by extracorporeal shock-wave lithotripsy.

lithotripsy was required nine and three times, and the duration of the treatment was 2 months and 16 days, respectively. The other patient (Case 20) was treated successfully by percutaneous transhepatic lithotomy.

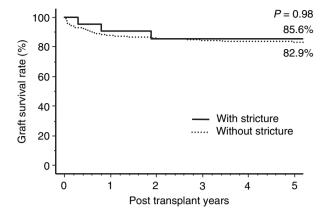
Two patients with complete obstruction of the anastomosis (PTC group) were treated successfully with Yamanouchi magnet compression anastomosis (Figs 2 and 4). Bilioenteric fistulas were established 12 and 25 days after the procedure. One patient maintained patency of the bilioenteric fistula and normal liver function for 6 years, the other had a stone in the intrahepatic biliary duct as described previously.

Among the 22 patients with HJ stricture, none required re-operation or died of biliary complications. The 5-year graft survival rate of 85.6% in these patients was equivalent to that in patients without biliary complications (82.9%, P = 0.98) (Fig. 5).

### Discussion

We used nonsurgical techniques for management of bilioenteric anastomotic strictures in post LDLT patients post LDLT patients and obtained fair results. No patient died of secondary biliary cirrhosis or sepsis on account of cholangitis or required reconstruction or re-transplantation for HJ stricture.

In patients with HJ strictures, PTC with balloon dilatation is generally recommended as a minimally invasive



**Figure 5** The graft survival rate showed no significant differences between patients with and without hepatojejunostomy stricture.

therapeutic intervention [7]. In our series, balloon dilatation alone achieved patency in only 45.5% (5/11) of the PTC group. The other patients required placement of a stent tube for several months. After removal of the stent tube, anastomotic patency was maintained in all cases. Prolonged biliary stenting after operative bile duct repair has been reported to decrease the incidence of recurrent stenosis [17]. PTC could be performed only when the intrahepatic duct was dilated. In patients with HJ reconstruction, if a percutaneous approach fails, surgical repair becomes necessary [6]. In the case of endoscopic intervention, an approach through the Roux-en-Y limb is problematic because the success rate of ERC using a conventional endoscope is reportedly low [8]. Recently, however, endoscopic balloon dilatation using double balloon endoscopy has been regarded as an alternative form of percutaneous transhepatic management [16]. A balloon-attached endoscope is passed through a balloon-attached overtube (sliding tube), allowing both observation of the entire small intestine including the afferent loop of the Roux-en-Y reconstruction and endoscopic therapy, which could not be achieved by conventional endoscopy [18]. The discomfort caused by an indwelling naso-biliary drainage tube prompted us to shorten the duration of treatment in ERC compared to that in PTC. Repeated ERC with balloon dilatation was performed as necessary.

We have placed a tube-jejunostomy in order to decrease intra-intestinal pressure, and we usually remove it at around 3 months after transplantation. Two patients who developed biliary stricture within 3 months after transplantation underwent balloon dilatation with a narrow-diameter endoscope through this jejunostomy. A narrow-diameter endoscope could be passed through the site of the tube jejunostomy after dilation up to 24-Fr in diameter. With this approach, it was easier to reach the site of HJ than with double balloon endoscopy through the Roux-en-Y limb. We have applied the long efferent limb of the Roux-en-Y anastomosis, as shown in Fig. 1, and fortunately we have not experienced any complications attributable to the length of the limb. Hutson *et al.* reported that this technique allows repeated dilatation of bilioenteric anstomotic stricture easily and safely [19]. Management with this procedure was effective in the cases complicated by stricture in the early postoperative course.

Yamanouchi *et al.* have reported the use of magnetic compression anastomosis, in which transmural compression with two magnets causes gradual ischemic necrosis to create an anastomosis between the bile duct and the small intestine [14]. In our series, two patients with complete obstruction were treated successfully with this procedure without any complications.

In the present study, the incidence of bilioenteric anastomotic stricture in post LDLT patients was lower than in other reports [20–23]. One possible explanation may be that our series included many pediatric patients (47.6%), and the incidence of bilioenteric anastomotic stricture in pediatric patients (5.5%) was lower than in adult patients (13.2%).

Although this treatment policy did not result in graft loss, a long period of hospitalization was required, and thus the cost was higher. However, a surgical approach would have involved a higher risk of graft loss for patients complicated by cholangitis and liver dysfunction than a nonsurgical one. In Japan, the marked paucity of cadaveric donors is problematic. Therefore, avoidance of graft loss and re-transplantation is of primary importance when performing LDLT under these circumstances. For this reason, nonsurgical repair has been the first choice for treatment of HJ stricture post LDLT, and the results have been reported here.

We have assessed the outcome of nonsurgical techniques (balloon dilatation, stent placement, and magnet compression anastomosis) for the management of HJ stricture post LDLT. Although the duration of treatment is rather long, advances in interventional techniques (e.g. narrow-diameter endoscopy, double balloon endoscopy, and magnet compression) have enabled wider application of a nonsurgical approach for this complication, and yielded fair results.

## Authorship

AM designed and performed the research and drafted the article, YH designed the research and revised the article, YM, YO, KU, YN, TI and MT all collected data, HY collected data and revised the article, and S-IM revised the article.

## References

- 1. Todo S, Furukawa H, Kamiyama T. How to prevent and manage biliary complications in living donor liver transplantation? *J Hepatol* 2005; **43**: 22.
- 2. Greif F, Bronsther OL, Van Thiel DH, *et al.* The incidence, timing, and management of biliary tract complications after orthotopic liver transplantation. *Ann Surg* 1994; **219**: 40.
- 3. Davidson BR, Rai R, Nandy A, Doctor N, Burroughs A, Rolles K. Results of choledochojejunostomy in the treatment of biliary complications after liver transplantation in the era of nonsurgical therapies. *Liver Transpl* 2000; **6**: 201.
- 4. Culp WC, McCowan TC, Lieberman RP, Goertzen TC, LeVeen RF, Heffron TG. Biliary strictures in liver transplant recipients: treatment with metal stents. *Radiology* 1996; **199**: 339.
- 5. Sung RS, Campbell DA, Jr. Rudich SM, *et al.* Long-term follow-up of percutaneous transhepatic balloon cholangioplasty in the management of biliary strictures after liver transplantation. *Transplantation* 2004; **77**: 110.
- Egawa H, Inomata Y, Uemoto S, *et al.* Biliary anastomotic complications in 400 living related liver transplantations. *World J Surg* 2001; 25: 1300.
- 7. Pascher A, Neuhaus P. Bile duct complications after liver transplantation. *Transpl Int* 2005; **18**: 627.
- Hintze RE, Adler A, Veltzke W, Abou-Rebyeh H. Endoscopic access to the papilla of Vater for endoscopic retrograde cholangiopancreatography in patients with billroth II or Roux-en-Y gastrojejunostomy. *Endoscopy* 1997; 29: 69.
- 9. Yamamoto H, Sekine Y, Sato Y, *et al.* Total enteroscopy with a nonsurgical steerable double-balloon method. *Gastrointest Endosc* 2001; **53**: 216.
- Kuno A, Yamamoto H, Kita H, *et al.* Double-balloon enteroscopy through a Roux-en-Y anastomosis for EMR of an early carcinoma in the afferent duodenal limb. *Gastrointest Endosc* 2004; **60**: 1032.
- Hashikura Y, Kawasaki S, Terada M, *et al.* Long-term results of living-related donor liver graft transplantation: a single-center analysis of 110 transplants. *Transplantation* 2001; **72**: 95.

- Kawasaki S, Makuuchi M, Matsunami H, *et al.* Living related liver transplantation in adults. *Ann Surg* 1998; 227: 269.
- Hashikura Y, Ikegami T, Nakazawa Y, et al. Domino liver transplantation in living donors. *Transplant Proc* 2005; 37: 1076.
- Muraoka N, Uematsu H, Yamanouchi E, *et al.* Yamanouchi magnetic compression anastomosis for bilioenteric anastomotic stricture after living-donor liver transplantation. *J Vasc Interv Radiol* 2005; 16: 1263.
- Takao S, Matsuo Y, Shinchi H, *et al.* Magnetic compression anastomosis for benign obstruction of the common bile duct. *Endoscopy* 2001; 33: 988.
- Haruta H, Yamamoto H, Mizuta K, *et al.* A case of successful enteroscopic balloon dilation for late anastomotic stricture of choledochojejunostomy after living donor liver transplantation. *Liver Transpl* 2005; 11: 1608.
- Pitt HA, Miyamoto T, Parapatis SK, Tompkins RK, Longmire WP, Jr. Factors influencing outcome in patients with postoperative biliary strictures. *Am J Surg* 1982; 144: 14.
- Yamamoto H, Kita H, Sunada K, *et al.* Clinical outcomes of double-balloon endoscopy for the diagnosis and treatment of small-intestinal diseases. *Clin Gastroenterol Hepatol* 2004; 2: 1010.
- Hutson DG, Russell E, Yrizarry J, *et al.* Percutaneous dilatation of biliary strictures through the afferent limb of a modified Roux-en-Y choledochojejunostomy or hepaticojejunostomy. *Am J Surg* 1998; 175: 108.
- 20. Chen CL, Fan ST, Lee SG, Makuuchi M, Tanaka K. Living-donor liver transplantation: 12 years of experience in Asia. *Transplantation* 2003; **75**: S6.
- Soejima Y, Taketomi A, Yoshizumi T, *et al.* Biliary strictures in living donor liver transplantation: incidence, management, and technical evolution. *Liver Transpl* 2006; 12: 979.
- 22. Ramacciato G, Varotti G, Quintini C, *et al.* Impact of biliary complications in right lobe living donor liver transplantation. *Transpl Int* 2006; **19**: 122.
- Giacomoni A, Lauterio A, Slim AO, *et al.* Biliary complications after living donor adult liver transplantation. *Transpl Int* 2006; 19: 466.