

CASE REPORT

Auxiliary partial orthotopic living donor liver transplantation in a patient with alcoholic liver cirrhosis to overcome donor steatosis

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Summary

The efficacy of auxiliary partial orthotopic liver transplantation (APOLT) to overcome the problems associated with a markedly steatotic graft in a living donor has not been fully explored. We have recently performed APOLT in a patient with alcoholic liver disease, where the only potential candidate donor was affected by 50% macrovesicular steatosis and 30% microvesicular steatosis. The recipient's left liver was resected and the donor's left liver, corresponding to a 0.46% graft-to-recipient weight ratio, was orthotopically transplanted. The postoperative course of this patient was uneventful, except for a transient large amount of ascites. Native liver volume in the recipient serially decreased, and the volume of the graft serially increased after transplantation. Four months after transplantation, the donor and recipient are doing well with a normal liver function. In conclusion, APOLT may be a feasible solution for a markedly steatotic living donor graft in patients with alcoholic liver disease.

Introduction

Auxiliary partial orthotopic liver transplantation (APOLT) is an attractive alternative to total transplantation because it can help overcome some of the limitations of living donor liver transplantation (LDLT). APOLT was initially developed to treat patients with fulminant and subfulminant hepatic failure [1,2], in whom preserved native liver has the potential to regenerate. Recently, this technique has also been used in patients with non-cirrhotic metabolic liver diseases [3], small-for-size grafts [4], and ABO incompatible grafts [5].

Grafts with severe steatosis are frequently associated with primary graft nonfunction, delayed graft function, and postoperative morbidity [6,7]. Moreover, major liver resec-

tion in donors with steatosis is known to be associated with a significant increase in postoperative morbidity [8]. Thus, the majority of centers do not consider donors with marked steatosis or a high body mass index for LDLT.

We report a case of successful APOLT in a patient with alcoholic liver cirrhosis, from a living donor with a marked degree of liver steatosis. This is the first description of APOLT in a patient with alcoholic liver disease with a special emphasis on overcoming steatotic liver in a living donor.

Case report

A 52-year-old Korean man was referred to our hospital for LDLT because of end stage alcoholic liver cirrhosis.

On admission to our hospital, encephalopathy with mild confusion and irritability was present, and his abdomen was tense because of severe ascites. His preoperative liver condition was grade C on the Child–Pugh scale (score 12) and his Model for End-Stage Liver Disease score was 20. There was no evidence of malignancy or of another space-occupying lesion mimicking a malignant tumor, or of focal thrombus in the portal vein. However, a hepatofugal portal flow pattern was observed on the preoperative Doppler scan.

The only donor candidate was his 24-year-old son. The donor's height and body weight were 180 cm and 96.3 kg, and his body mass index was 29.7 kg/m². The results of his liver function tests were completely normal. His liver-spleen attenuation ratio was 1.11 on non-contrast computed tomography (CT) images, and the graft-to-recipient weight ratio (GRWR) predicted by CT volu-

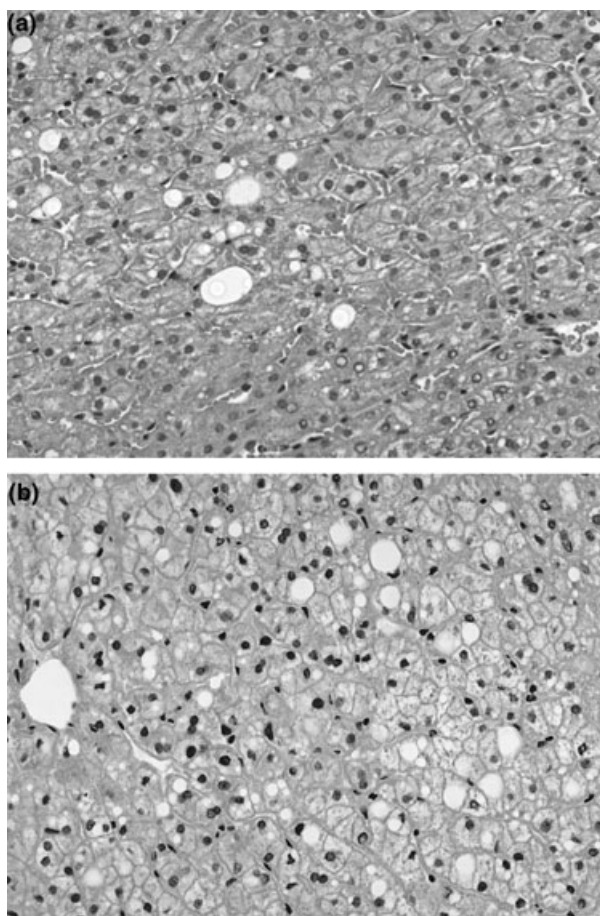


Figure 1 Histological findings of the donor liver by Hematoxylin–Eosin staining. (a) The preoperative specimen by needle biopsy showed 10% macrovesicular steatosis (MaS) and 15% microvesicular steatosis (MiS). (b) The intraoperative specimen by wedge biopsy showed 50% MaS and 30% MiS.

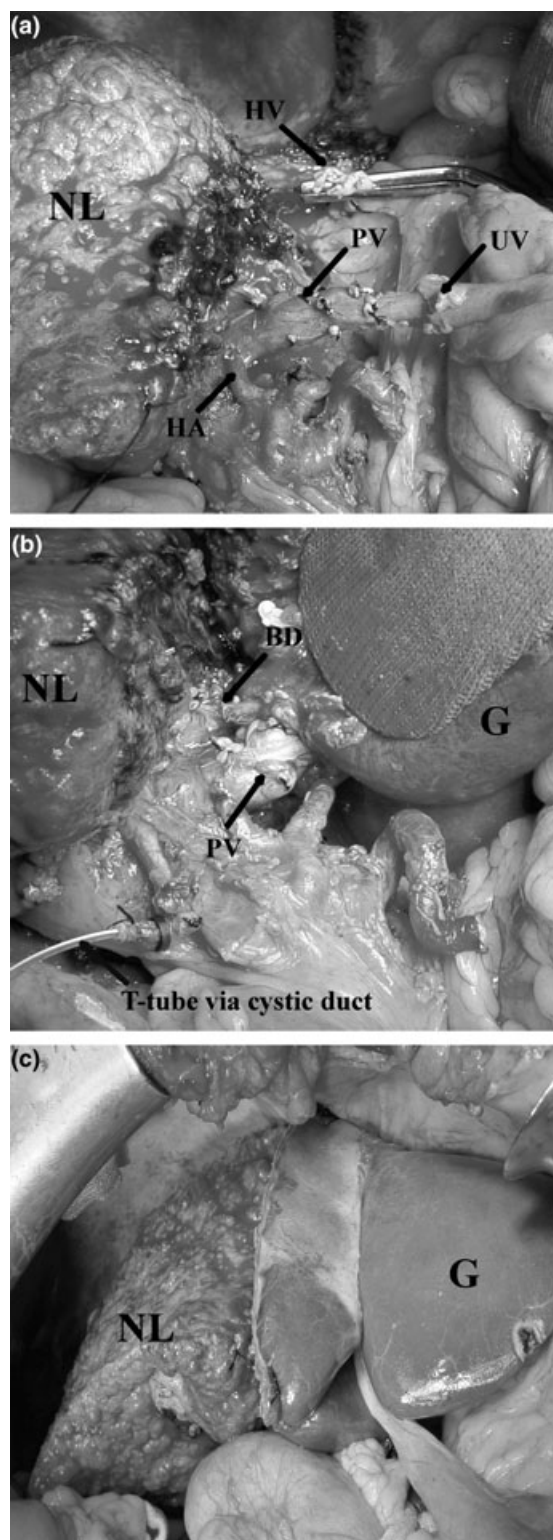


Figure 2 Intraoperative view of the native liver and of the orthotopically transplanted liver graft. UV, umbilical vein; HV, hepatic vein; HA, hepatic artery; BD, bile duct; PV, portal vein; NL, native liver; G, graft.

metry was 1.04%, for a right lobe graft. Currently, in our center, a preoperative liver biopsy was performed on donors who were suspected of having a steatosis by a preoperative imaging study [10]. Thus, a liver biopsy was performed on the present donor. Liver specimens taken by needle biopsy contained 10% macrovesicular steatosis (MaS) and 15% microvesicular steatosis (MiS). Therefore, we planned to perform urgent LDLT because of the recipient's poor condition and ongoing encephalopathy using the son's right liver.

However, at the time of the donor operation, the donor's liver was suspected to contain marked steatosis because of the presence of rounded edges, a yellow discoloration, and a greasy firm texture. The donor underwent an intraoperative wedge liver biopsy to re-confirm the degree of hepatic steatosis. And, the reported degree of steatosis was 50% MaS and 30% MiS (Fig. 1). Thus the graft was considered insufficient to support the recipient's metabolic demand and right hepatectomy in the donor was viewed as being too risky. Therefore, we decided to perform APOLT using the donor's left liver. The recipi-

ent's left liver (segments 2–4 according to Couinaud's segmentation; 263 g) was resected, and the donor's left liver (segments 2–4; 350.4 g) corresponding to a GRWR of only 0.46% was transplanted orthotopically.

A special type of dissection was performed for the left hepatectomy in the recipient, which was previously described [11]. A transection plane was drawn after clamping the left hepatic artery and the left portal vein. The liver parenchymal transection was carried out using a meticulous surgical technique with an ultrasonic aspirator and a bipolar coagulator. The distal part of the middle hepatic vein was exposed. The bile duct remained intact until the parenchymal transection was complete. After a further parenchymal transection along the inferior margin of the left medial segment of the liver, the left bile duct was fully exposed. The stump of the left bile duct was incised at the level of the umbilical portion as long as possible. The remaining perihepatic dissection was resumed. During the recipient hepatectomy, the recanalized umbilical vein, which was directly connected to the portal vein, was preserved without ligation till portal vein anastomosis

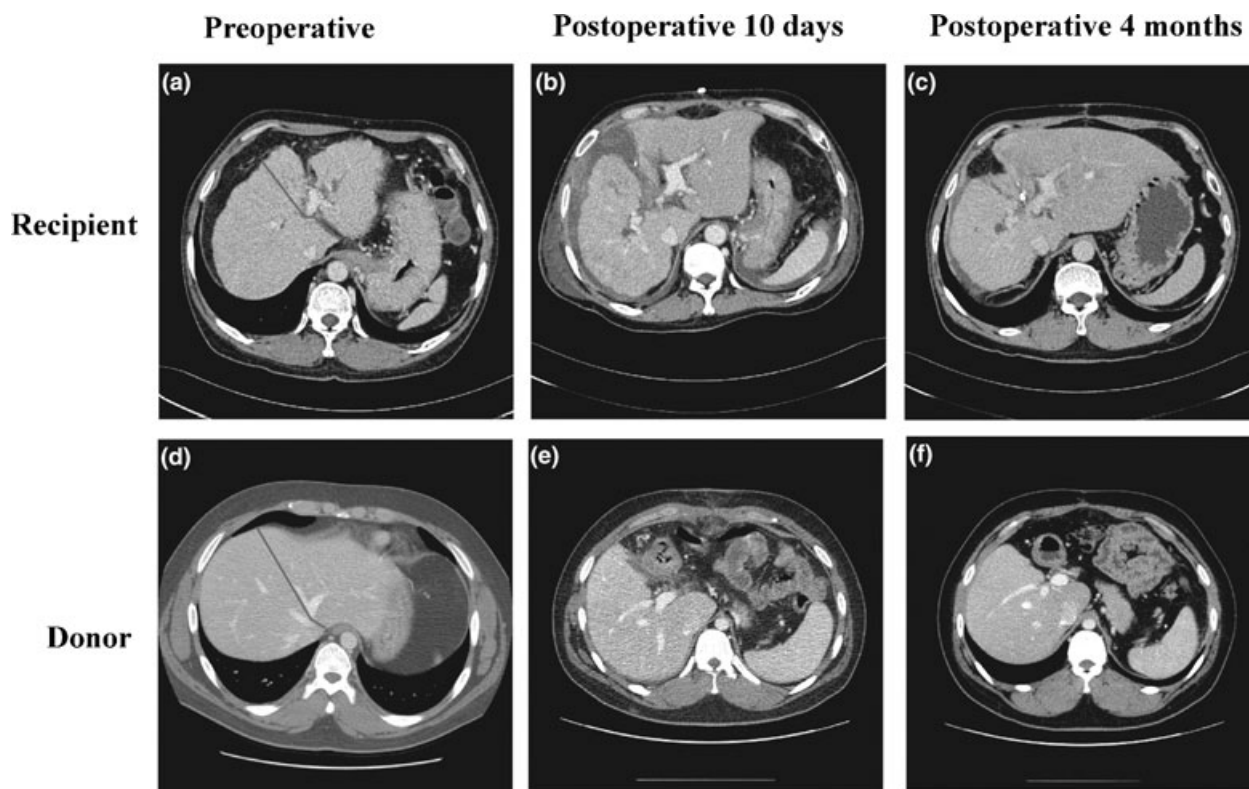


Figure 3 Computed tomographic (CT) findings of the recipient (Panels a, b, and c) and donor (Panels d, e, and f). Panels a and d showed preoperative CT scans of the recipient and the donor, respectively. The continuous line indicates the transection line. The volume of the native right liver in the recipient serially decreased and the volume of the graft increased after transplantation (Panels b and c). The remnant liver in the donor regenerated well (Panels e and f).

(Fig. 2). This procedure leaves the recipient's preoperative hepatic circulation unperturbed and facilitates hemodynamic stability. The donor's left hepatic vein and segment 4 tributary vein of the middle hepatic vein were reconstructed to a common cloaca on the back table and anastomosed to the recipient left hepatic vein in an end-to-end fashion. The donor's portal vein was anastomosed to the corresponding recipient left portal vein, and an end-to-end anastomosis between the left hepatic artery of the graft and that of the recipient was carried out under an operating microscope. An intraoperative Doppler scan demonstrated a hepatofugal portal flow pattern and dominant hepatic artery flow in the native right liver, and peak velocity in the graft left portal vein of 81.5 cm/s and in the left hepatic artery of 74.4 cm/s. This finding suggested that the majority of portal flow supplied the graft and that the native liver was supplied by the originally hypertrophied hepatic arterial blood flow. A bile duct reconstruction was then performed between the left hepatic duct of the graft and that of the recipient by duct-to-duct anastomosis with a tube stent via the recipient cystic duct. Total operative time was 470 min and two pints of red blood cells and two pints of fresh frozen plasma were infused. A histological examination of the resected left liver revealed severe micronodular cirrhosis and the absence of malignancy.

The donor recovered well, and was discharged 14 days after hepatectomy without any morbidity. CT scan performed on the 10th postoperative day showed that the remnant right liver had regenerated to 83.4% of the original whole liver volume. According to a CT scan performed at 4 months after the donation, the remnant liver had regenerated to 99.3% of its preoperative state (Fig. 3).

After transplantation, the postoperative course of the recipient was uneventful. A CT scan performed at 10 days after transplantation showed a well-regenerated graft (from 438 ml preoperatively to 615 ml at 10 days after APOLT) and an atrophied native liver (from 562 ml preoperatively to 493 ml at 10 days after APOLT). The patient was discharged at 39 days after transplantation without any major morbidity, with liver function tests within the normal range. Bile juice secretion scintigraphy by [^{99m}Tc]-DISIDA at 3 months after transplantation revealed that the grafted left liver secreted most of the bile juice. On the other hand, the native right liver showed diminished bile juice secretion (Fig. 4). A CT scan performed at 4 months after transplantation demonstrated that the graft had regenerated to 695 ml (159% of the original volume), and that the native liver volume had reduced further to 345 ml (61.4% of the original volume). Four months after transplantation, both donor and recipient are doing well with normal liver functions (Fig. 5).

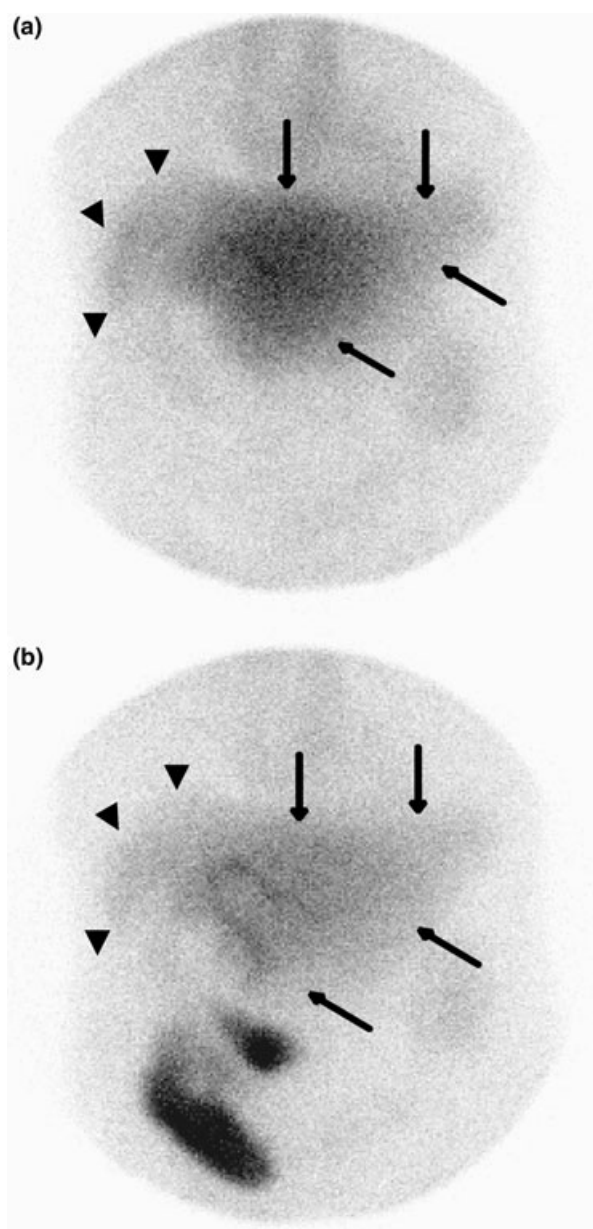


Figure 4 Bile juice secretion [^{99m}Tc]-DISIDA scintigraphic findings (Panel a, scan taken 10 min after injection; Panel b, scan taken 60 min after injection) performed 3 months after transplantation. Bile juice secretion scintigraphy revealed that the left lobe of the liver (arrowhead), i.e. the graft liver, secreted most of the bile juice. Conversely, the right lobe (arrow), i.e. the native liver, showed diminished function.

Discussion

Functional competition, the struggle between the graft and the native liver for portal blood flow, is an inherent problem in APOLT. In APOLT for a small-for-size graft, it is important to make a pre-emptive transection of the

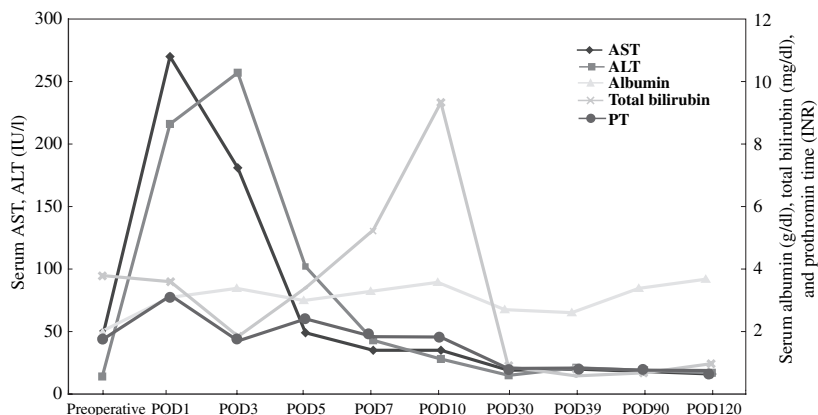


Figure 5 Postoperative changes in the aspartate aminotransferase, alanine aminotransferase, albumin, and total bilirubin levels, as well as prothrombin time in the recipient.

portal branch to the native liver at the time of transplantation [12]. However, a portal flow diversion technique was not mandatory in the present case because the recipient suffered from chronic alcoholic liver disease, and such a liver may be dependent on arterial supply; moreover, the flow pattern of the portal vein was hepatofugal. We attempted APOLT with the presumption that the graft liver would be supplied by the entire portal flow and that the reduced native liver would be sustained by the original hypertrophied arterial supply.

If a native liver has a higher possibility of malignancy *in situ*, its removal after graft growth may also be considered in some cases, such as in primary sclerosing cholangitis [9]. Following regeneration of the native liver, the auxiliary graft can be either removed or left to atrophy gradually by tapering off immunosuppression and provoking controlled rejection. However, the practical problem is how to distinguish between recipients with and without sufficient hepatic function to support the graft. Unfortunately, experience with APOLT is still limited and this question remains to be resolved.

Auxiliary partial orthotopic liver transplantation was initially introduced as a temporary or permanent support for patients with fulminant hepatic failure [1]. Since then, its indications have been extended to liver-based metabolic disorders [3] and size mismatch cases [4]. The rationale behind APOLT for fulminant hepatic failure focuses on the full native liver regeneration and the discontinuation of immunosuppressive therapy. Therefore, the auxiliary graft should support the remnant native liver during regeneration [2]. In noncirrhotic metabolic disorders, the underlying principle behind APOLT is that the graft can compensate for the enzyme deficiencies without the complete removal of the native liver, which may help the recipient in cases of potential graft failure. Weight alone is not a suitable guide because a graft with an MaS <30% can be used without increasing the risk of primary graft nonfunction or morbidity in LDLT [10], but MaS, particularly when >30%, increases the risk of graft dys-

function and primary nonfunction [13]. Therefore, the rationale for APOLT in the present case involving the use of a markedly steatotic graft from a living donor differed completely from that for other indications. As in cases with a small-for-size graft, the native liver left partially in place is expected to support the function of the newly implanted insufficient graft liver during the immediate early postoperative period. After transplantation, the grafted liver expands its function in proportion to its volume growth, while the native liver cannot grow or maintain its function. The other important rationale for APOLT using a markedly steatotic graft as in the present case, and one which should not be ignored, is that marked steatosis ($\geq 30\%$) is an independent predictor of complications following hepatic resection [14], and that the operative risk of right hepatectomy is too hazardous for the donor.

To date, the only available solution to the problems encountered using a markedly steatotic liver in LDLT is adult-to-adult LDLT using dual grafts. However, the procedure has limited appeal because of its high requirement for economic and medical resources and its technical demands, and therefore, liver transplantation using dual grafts is not widely performed [15]. The present case thus suggests a feasible means of overcoming donor steatosis in LDLT, and urges that preconceptions concerning steatotic livers be challenged.

In conclusion, APOLT may be a feasible treatment modality in patients with alcoholic liver disease, especially in those with a potential donor with a markedly steatotic liver.

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