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Editorial board member of *World Journal of Gastroenterology*, Marcos V Perini, MD, MSc, PhD, Senior Lecturer, Department of Surgery, University of Melbourne, Melbourne, Victoria 3084, Australia

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Rescue case of low birth weight infant with acute hepatic failure

Noriki Okada, Yukihiro Sanada, Taizen Urahashi, Yoshiyuki Ihara, Naoya Yamada, Yuta Hirata, Takumi Katano, Kentaro Ushijima, Shinya Otomo, Shujiro Fujita, Koichi Mizuta

Noriki Okada, Yukihiro Sanada, Taizen Urahashi, Yoshiyuki Ihara, Naoya Yamada, Yuta Hirata, Takumi Katano, Koichi Mizuta, Department of Transplant Surgery, Jichi Medical University, Shimotsuke 3290498, Japan

Kentaro Ushijima, Department of Clinical Pharmacology, Jichi Medical University, Shimotsuke 3290498, Japan

Shinya Otomo, Department of Pharmacy, Jichi Medical University, Shimotsuke 3290498, Japan

Shujiro Fujita, Department of Pediatrics, Yokohama City University School of Medicine, Yokohama 2360004, Japan

ORCID number: Noriki Okada (0000-0001-5655-625X); Yukihiro Sanada (0000-0003-2456-0400); Taizen Urahashi (0000-0002-6229-090X); Yoshiyuki Ihara (0000-0002-0028-0634); Naoya Yamada (0000-0003-0111-028X); Yuta Hirata (0000-0002-2728-2648); Takumi Katano (0000-0001-7649-7090); Kentaro Ushijima (0000-0003-2637-3916); Shinya Otomo (0000-0002-4857-0278); Shujiro Fujita (0000-0003-1790-3823); Koichi Mizuta (0000-0003-4270-4834).

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Correspondence to: Noriki Okada, MD, PhD, Department of Transplant Surgery, Jichi Medical University, 3311-1 Yakushiji, Shimotsuke 3290498, Japan. r0906no@jichi.ac.jp
Telephone: +81-285-587069
Fax: +81-285-587069

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Abstract

We report a case involving a rescued low birth weight infant (LBWI) with acute liver failure. Case: The patient was 1594 g and 32^{3/7} gestational wk at birth. At the age of 11 d, she developed acute liver failure due to gestational alloimmune liver disease. Exchange transfusion and high-dose gamma globulin therapy were initiated, and body weight increased with enteral nutrition. Exchange transfusion was performed a total of 33 times prior to living donor liver transplantation (LDLT). Her liver dysfunction could not be treated by medications alone. At 55 d old and a body weight of 2946 g, she underwent LDLT using an S2 monosegment graft from her mother. Three years have passed with no reports of intellectual disability or liver dysfunction. LBWIs with acute liver failure may be rescued by LDLT

after body weight has increased to over 2500 g.

Key words: Liver transplantation; Acute liver failure; Low birth weight infant; Transplantable body weight; Monosegment graft

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Core tip: We report a case involving a rescued low birth weight infant (LBWI) with acute liver failure. The patient was 1594 g at birth. At the age of 11 d, she developed acute liver failure due to gestational alloimmune liver disease. Medications were initiated, and body weight increased with enteral nutrition. Her liver dysfunction could not be treated by medications alone. At 55 d old with a body weight of 2946 g, she underwent living-donor liver transplantation (LDLT) using an S2 monosegment graft. Conclusion: LBWIs with acute liver failure may be rescued by LDLT after body weight has increased to over 2500 g.

Okada N, Sanada Y, Urahashi T, Ihara Y, Yamada N, Hirata Y, Katano T, Ushijima K, Otomo S, Fujita S, Mizuta K. Rescue case of low birth weight infant with acute hepatic failure. *World J Gastroenterol* 2017; 23(40): 7337-7342 Available from: URL: <http://www.wjgnet.com/1007-9327/full/v23/i40/7337.htm> DOI: <http://dx.doi.org/10.3748/wjg.v23.i40.7337>

INTRODUCTION

Neonatal acute hepatic failure is a rare but serious disease^[1]. Reports have indicated that the cause of neonatal acute hepatic failure is most frequently gestational alloimmune liver disease (GALD), but in rare cases, it can be metabolic disorder, viral infection, or mitochondrial disorder, among other possibilities^[2]. The initial treatment for neonatal acute liver failure is apheresis and medication while the cause of hepatic failure is determined. If hepatic recovery has not been achieved with medication alone, liver transplantation is indicated^[1,3]. However, the living donor liver transplantation (LDLT) procedure for neonatal recipients is challenging due to the size mismatch between the liver graft and the body of the recipient. Moreover, management during the perioperative period is also challenging^[4]. LDLT is particularly difficult for low birth weight infants (LBWIs) for the reasons discussed above. In such cases, LDLT can result in recovery if the body weight of the infant can be increased by nutritional management^[5]. However, few reports have discussed LDLT for neonatal recipients with low body weights^[6]. Herein, we report a case involving a rescued LBWI with acute liver failure and discuss the limitation of body weight as an indicator for neonatal LDLT.

CASE REPORT

The patient was born at 32^{3/7} gestational wk because of fetal distress. Her birth weight was 1594 g (Figures 1 and 2). Immediately after birth, hypoglycemia and hypotension appeared. A daily administration of hydrocortisone and continuous administration of dopamine hydrochloride were started at the age of 1 day. At the age of 11 d, coagulation dysfunction and an elevated serum ferritin level due to acute liver failure were observed (ferritin 2865 ng/mL, total bilirubin 12.5 mg/dL, PT-INR 4.57). The patient's older sister was highly suspected of GALD by clinical course and pathological findings and underwent LDLT at 13 d of age. GALD was highly suspected in this case after excluding the possibility of metabolic disorder or infectious disease. The patient did not undergo magnetic resonance imaging or salivary gland biopsy for GALD diagnosis. We explained the high recurrence rate of GALD in siblings to her parents; however, the parents gave birth to a baby in another hospital without informing us. Starting at 11 d of age, exchange transfusion and medication therapy using high-dose gamma globulin and deferoxamine were initiated. The patient's body weight had been gradually increased with enteral nutrition using commercially available nutrients, administered 8 times per day *via* a gastric tube. She then recovered from hypotension, and the continuous administration of dopamine hydrochloride was finished. Exchange transfusion was performed a total of 33 times prior to LDLT. The patient was transported to our hospital at 44 d of age; at that time, her body weight was 2525 g. We speculated that her target body weight for LDLT would be over 2500 g based on the estimated graft volume. Computed tomography scan revealed a markedly atrophied liver (resected liver was 78 g). Laboratory data showed repeated coagulopathy and hyperbilirubinemia with daily exchange transfusion and a pediatric end-stage liver disease (PELD) score of 15.8 (T-bil 5.09 mg/dL, PT-INR 1.91, Albumin 3.4 g/dL). A liver biopsy revealed a marked loss of hepatocytes. The remaining hepatocytes were multinucleated, and there was widespread fibrosis around Glisson's sheath and in the parenchymal area (F3-4) (Figure 2F and G). Thus, her liver dysfunction could not be successfully treated *via* medications alone, and she was judged to be indicated for liver transplantation at that time. We tried to perform LDLT at 45 d of age, but before LDLT, she went into shock following hemothorax due to failure of catheter insertion with injury of the right subclavian artery. After the recovery period, she underwent LDLT at 55 d of age using an S2 monosegment graft from her mother (107 g, graft-recipient weight ratio (GRWR) 3.6%; Figure 2 A). The body weight of the patient at LDLT was 2946 g. At the time of LDLT, the PELD score of the patient was 21.9 (T-bil 13.74 mg/dL, PT-INR

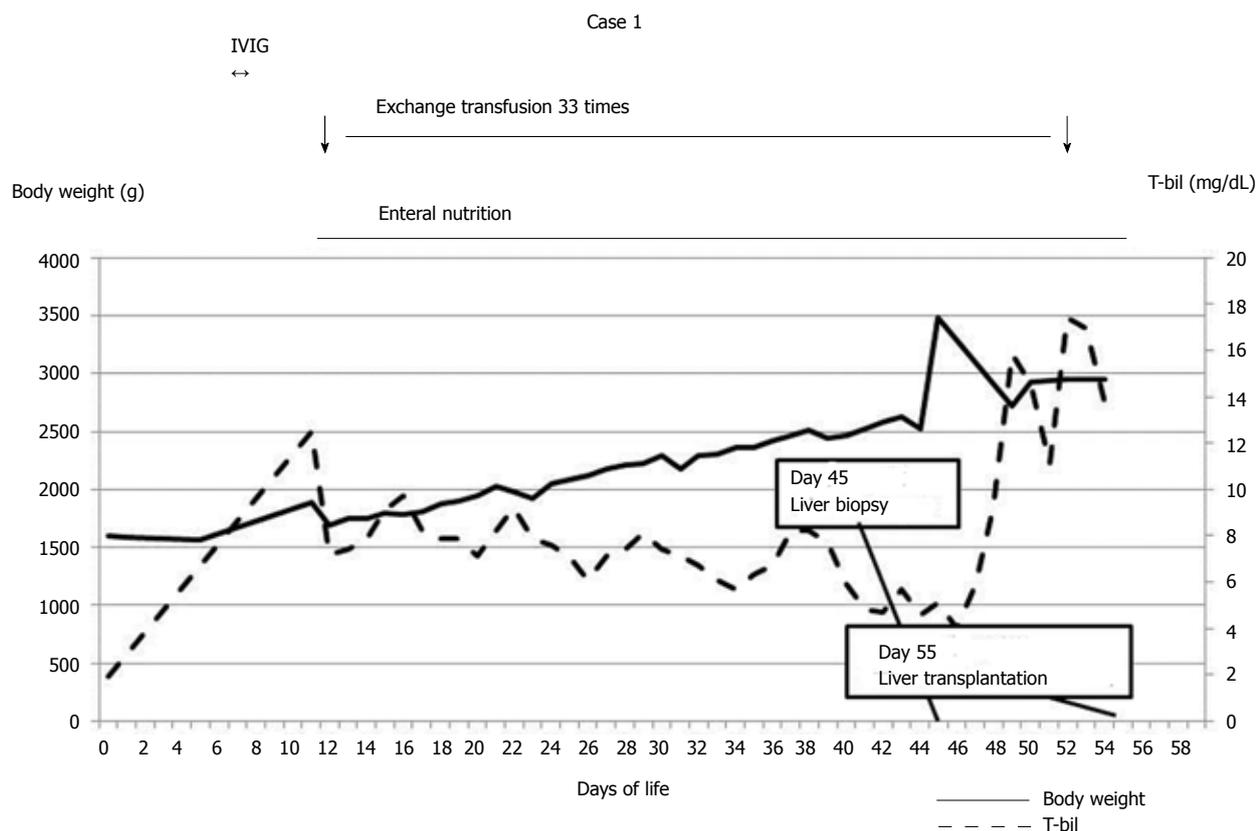


Figure 1 The preoperative treatment and changes in body weight and total bilirubin. The patient was able to gain weight due to the use of internal medication and enteral nutrition.

2.12, Albumin 3.7 g/dL). The operation duration was 13 h and 37 min and bleeding was 700 mL [238 mL/recipient body weight (kg)]. A transverse incision was created, and the liver was resected with temporary bypass of the portal vein. The resected liver was 78 g (Figure 2E). The recipient's right hepatic artery (2.0 mm) was anastomosed to the graft's left hepatic artery (2.5 mm) *via* a dorsal position of the portal vein anastomosis using a microsurgical technique. The graft-to-recipient distance ratio (GRDR) was 2.4 (58/24). Biliary reconstruction was performed using a Roux-en-Y hepaticojejunostomy. The abdominal wound could not be closed because respiratory failure occurred due to abdominal compartment syndrome (Figure 2B and C). Intraoperative water balance was +1645 mL [558 mL/recipient body weight (kg)]. After LDLT, continuous hemodiafiltration (CHDF) had been performed for systemic edema, removing water as long as blood pressure and portal vein flow remained steady. Respiratory failure due to abdominal compartment syndrome and lung edema gradually improved. Thus, on postoperative day (POD) 5, the abdominal skin of the patient was closed without closing the abdominal fascia (Figure 2D). Tacrolimus and methylprednisolone were used as the standard postoperative immunosuppression therapy regimen. Acute rejection occurred on POD 17, so steroid pulse therapy was initiated. After steroid pulse therapy, liver

enzyme, PT-INR and T-bil were nearly normalized. Respiratory failure due to large-for-size graft syndrome had been prolonged; however, it gradually improved, and she was extubated on POD 81. Cytomegalovirus infection and catheter infection occurred several times and were treated using antiviral or antibiotic drugs. The patient showed difficulty eating sufficient meals and required habilitation to eating. She was discharged on POD 225. Vessel complications did not occur. Three years have passed since LDLT, with no reports of intellectual disability or liver dysfunction.

DISCUSSION

Liver transplantation for a small recipient

We rescued a case involving an LBWI with acute liver failure; however, few reports to date describe such a case (Table 1). To our knowledge, the lightest LDLT recipient had a body weight of 2.4 kg at the time of LDLT^[7], and the youngest LDLT recipient in Japan was 9 d of age^[8-10]. For deceased donor liver transplantation, the smallest reported recipient was 2 kg, and the youngest was 7 d of age^[11]. No consensus has been reached regarding the safe lower limit of body weight for small recipients. A recent UNOS database analysis of infants weighting less than 5 kg revealed one-year patient and graft survival rates of 77.7% and 66.1%, respectively^[12]. A recent Japanese

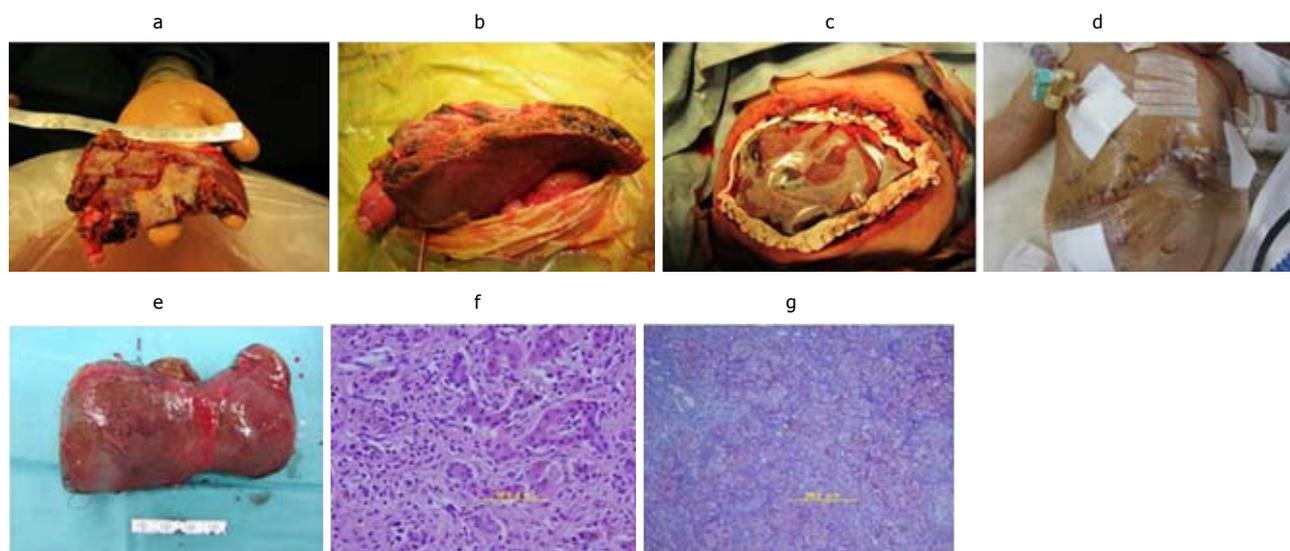


Figure 2 Images. A: The S2 monosegment graft (107 g) on the back table; B: An image obtained after reperfusion, the graft was too large to close the abdominal fascia; C: The abdominal fascia could not be closed at the time of living donor liver transplantation (LDLT), excess water was removed by continuous hemodiafiltration after LDLT; D: Secondary skin closure was performed on postoperative day 5; E: The resected liver was 78 g; F: Hematoxylin and eosin staining revealed a marked lack of hepatocytes and the presence of multinucleated hepatocytes; G: Azan staining revealed widespread fibrosis around Glisson's sheath and the parenchymal area (F3-4).

Table 1 Problems and management for low birth weight infant with acute liver failure		
	Problems	Management
Pre-LT	Low body weight Liver failure Donor	Enteral nutrition targeting to over 2500 g Apheresis (exchange transfusion, plasmapheresis) Informed consent
LT	Large-for-size graft syndrome Hepatic artery reconstruction Abdominal compartment syndrome	Monosegment graft Brunch patch, dorsal approach Skin closure
Post-LT	Fluid overload Respiratory failure	Open management→secondary skin closure Aggressive water removal using CHDF

CHDF: Continuous hemodiafiltration.

study reported an improved survival rate of 90.1% in an infant recipient within 3 mo^[7]. Recipients who were within 3 mo of age also showed a higher rate of biliary complications^[7]. However, our case did not have biliary complications. The problems we experienced in the described case were related to the following: (1) large-for-size graft syndrome^[4]; (2) the transplantable body weight of the recipient; and (3) vessel reconstruction difficulties^[13]. Challenges in managing transplantation for LBWIs with acute liver failure are nearly always due to the low body weight and fragility of the recipient.

The lower limit of the recipient's body weight for living donor liver transplantation

In liver transplantation, the transplantable body weight of a neonatal recipient is limited by graft size. If the GRWR is greater than 4.0%, the risk of abdominal compartment syndrome and insufficient blood supply in relation to a large-for-size graft are increased; the recipient abdomen cannot be closed, or massive

hepatocyte necrosis may occur in the transplanted graft^[8,10,14,15]. In pediatric LDLT, particularly in cases for which the recipient's body weight is less than 6 kg, monosegment grafts or hyper-reduced left lateral grafts have been transplanted^[4]. A total of 268 pediatric patients underwent LDLT 275 times between May 2001 and December 2015 at Jichi Medical University Hospital. These transplantation procedures involved 196 left lateral segment grafts, with a median graft weight of 230 g (range, 138-382 g). Monosegment grafts were transplanted in 13 cases (including 9, 3, and 1 cases involving S2, reduced S2, and S3 monosegment grafts, respectively); in these cases, the median graft weight was 124 g (range, 93-180 g). A hyper-reduced left lateral segment graft was transplanted in 1 case, and the graft volume was 172 g. The smallest graft that we have transplanted was 93 g, and the median GRWR was 3.6% (2.4%-4.2%). A previous study reported hyper-reduced left lateral segment graft weights of 72-189 g,

with most graft weights being 100-150 g^[7]. The lower limit of the smallest graft volume from a living donor is approximately 100 g^[10]. Given this graft weight, transplantable recipient body weights of 2500 g or more will result in a GRWR of 4.0% or less. Based on this estimation, 2500 g is a reasonable body weight for such recipients.

The specific peritransplant management of living donor liver transplantation for low birth weight infant

In our experience, the body weights of the described patient were increased by enteral nutrition and became greater than 2500 g at 44 d of age. Another case of increased body weight by enteral nutrition was reported, and the body weight increased to greater than 2500 g at 41 d of age^[5]. Despite the existence of acute liver failure, the body weights of the patients increased to over 2500 g *via* enteral nutrition while exchange transfusions were performed. Thus, in cases of acute liver failure, body weight may be increased with enteral nutrition if appropriate medication therapy that includes apheresis, such as exchange transfusion or plasmapheresis, is performed.

At times, it is impossible to close the abdominal fascia of the recipient during LDLT due to abdominal compartment syndrome. In this case, the abdominal fascia could not be closed during LDLT; instead, the case required secondary skin closure. In cases involving pediatric LDLT patients, apheresis and dialysis are reportedly effective^[16]. Aggressive water removal using CHDF was required in this case. Thus, even if LDLT for an LBWI with acute liver failure is successfully performed after an increase in body weight, circulation and respiratory management are challenging due to volume overload. Under these circumstances, aggressive water removal using CHDF and secondary abdominal closure approaches are useful and important tools for successful postoperative management.

Hepatic arterial reconstruction during living donor liver transplantation

Vessel reconstruction in cases similar to the described case is difficult due to the high GRDR between the hepatic vein and the portal vein bifurcation and to diameter mismatch for the hepatic artery between the graft and the recipient^[13]. Hepatic artery reconstruction by the dorsal approach of portal vein anastomosis has been reported to help reduce the risk of hepatic artery complications among small recipients if the GRDR is over 2.4^[13]. At times, the hepatic arteries of such recipients are thin. The hepatic artery of this case was not thin; however, the GRDR was 2.4, and the hepatic artery was anastomosed by a dorsal approach. In our experience of 13 cases of LDLT using monosegment grafts, the thinnest artery among the recipients was 1.2 mm, with 3 cases involving hepatic arteries of less than 1.5 mm. The graft-to-recipient diameter ratio was 0.67: 1.67. In addition, the dorsal position was

selected for hepatic artery anastomotic approaches in 7 cases (53.8%), and the branch patch technique was selected in 5 cases (38.5%). Hepatic artery complications after LDLT occurred in 3 cases, although interventional radiology was sufficient for achieving recovery in all 3 cases.

We rescued a case of LBWI with acute liver failure by performing LDLT. In cases involving LBWIs with acute liver failure, infants may be rescued by LDLT after their body weights have been increased to over 2500 g *via* repeated exchange transfusions and enteral nutrition.

COMMENTS

Case characteristics

The patient was 1594 g, 32^{3/7} gestational wk at birth and immediately after birth, hypoglycemia and hypotension appeared.

Clinical diagnosis

At the age of 11 d, coagulation dysfunction and an elevated serum ferritin level due to acute liver failure were observed.

Differential diagnosis

The case was highly suspected of gestational alloimmune liver disease and differential diagnosis was metabolic disorder, infectious disease and Neimann-Pick disease type C.

Laboratory diagnosis

Laboratory data showed repeated coagulopathy and hyperbilirubinemia with daily exchange transfusion and a pediatric end-stage liver disease score of 15.8.

Imaging diagnosis

Computed tomography scan revealed a markedly atrophied liver.

Pathological diagnosis

A liver biopsy revealed a marked loss of hepatocytes and the remaining hepatocytes were multinucleated, and there was widespread fibrosis around Glisson's sheath and in the parenchymal area (F3-4).

Treatment

The case underwent living donor liver transplantation at 55 d of age using an S2 monosegment graft from her mother (107 g).

Related reports

Kasahara M *et al* and Mizuta K *et al* reported the living donor liver transplantation for small recipient (*Exp Clin Transplant* 2014; 12 Suppl 1:1-4, *Am J Transplant* 2010; 10: 2547-2552).

Term explanation

Low birth weight infant (LBWI) is defined as a birth weight of an infant of 2499 g or less regarding gestational age.

Experiences and lessons

In cases involving LBWI with acute liver failure, infants may be rescued by living donor liver transplantation after their body weights have been increased to over 2500 g.

Peer-review

The authors presented an interesting case with LBWIs (1594g, 323/7 wk) with acute liver failure.

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