Single Case

Renoportal Anastomosis in Left Lateral Lobe Living Donor Liver Transplantation: A Pediatric Case

Hiroyuki Ogasawara[a]   Chikashi Nakanishi[a]   Shigehito Miyagi[a]
Kazuaki Tokodai[a]   Yasuyuki Hara[a]   Wataru Nakanishi[a]   Koji Miyazawa[a]
Kenji Shimizu[a]   Hiroyuki Kumata[a]   Hitoshi Goto[a]   Masafumi Goto[b]
Michiaki Unno[a]   Takashi Kamei[a]

[a]Department of Surgery, Graduate School of Medicine, Tohoku University, Sendai, Japan;
[b]Division of Transplantation and Regenerative Medicine, Tohoku University, Sendai, Japan

Keywords

Abstract

In adult liver transplantation, renoportal anastomosis (RPA) has been introduced as a useful technique for patients with grade 4 portal vein thrombosis and a splenorenal shunt. Here, we report a pediatric case in which RPA allowed a left lateral lobe living donor liver transplantation (LDLT) despite portal vein thrombosis and a large splenorenal shunt. At 36 days old, the patient underwent a Kasai operation for biliary atresia. At 17 months old, she underwent LDLT because of repetitive cholangitis. Pretransplant examinations revealed a large splenorenal shunt and portal vein thrombosis. Simple end-to-end portal reconstruction and clamping of the collateral route after removing the thrombosis were unsuccessful. Thus, RPA was performed using a donor superficial femoral vein as an interpositional graft. The portal vein pressure was 20 mm Hg after arterial reperfusion. Ligation of the splenic artery reduced

Ogasawara et al.: Renoportal Anastomosis in Left Lateral Lobe Living Donor Liver Transplantation: A Pediatric Case

the portal vein pressure. Although she developed severe acute cellular rejection and chylous ascites, there were no signs of portal vein complications. She was discharged 73 days after transplantation without any signs of renal dysfunction. The patient's condition was good at her last follow-up, 22 months after transplantation. To our knowledge, this is the youngest case of RPA in pediatric left lateral lobe LDLT. Additionally, this is the first case of RPA with splenic artery ligation and using the donor's superficial femoral vein as the venous graft for RPA. Although long-term follow-up is necessary, RPA could be a salvage option in LDLT in infants if other methods are unsuccessful.

Introduction

Liver transplantation (LT) is considered the most effective therapy for end-stage liver disease. Portal vein thrombosis (PVT) is a well-recognized complication in patients with liver cirrhosis, with a reported incidence of 5–15% [1]. Initially, PVT was recognized as an absolute contraindication for LT because of the associated technical problems and high incidence of PVT recurrence after LT. In patients with biliary atresia, a leading indication for pediatric LT, portal vein (PV) phlebosclerosis is frequently seen in response to repeated cholangitis. Therefore, PV reconstruction is technically more challenging in pediatric patients with biliary atresia. Surgical techniques such as PV venoplasty, interpositional graft, and mesenteric jump graft using a donor vein graft allow surgeons to perform LT in patients with phlebosclerotic and thrombosed PV [2].

In the technique of renoportal anastomosis (RPA), the PV of the liver graft is anastomosed to the recipient left renal vein using an interpositional graft. In 1997, Sheil et al. [3] reported the first successful LT involving the left renal vein for PV reconstruction. Their patient had a thrombosed PV and a functional, surgically created proximal left renal vein-to-side splenic vein shunt (renal-lieno shunt). Since then, RPA has been increasingly performed in patients with PVT and splenorenal shunt (SRS). Recently, in adult liver transplant patients with PVT and a large spontaneous SRS, RPA has been introduced in order to establish good portal inflow [1]. However, there are very few reports of RPA in pediatric patients. Here, we report a pediatric case in which RPA allowed left lateral lobe living donor liver transplantation (LDLT) despite PVT and a large SRS.

Case Report

A 15-month-old female was referred to our department for LDLT. At 36 days old, she underwent a Kasai operation for biliary atresia. Six months later, she developed cholangitis. Antibiotics and percutaneous biliary drainage failed to relieve the symptoms of cholangitis and she had persistent intermittent high-grade fever (over 40°C) for more than 6 months. The patient's laboratory findings before transplantation are shown in Table 1. Her Child–Pugh score was 6 (grade A), and her pediatric end-stage liver disease (PELD) score was 1. The PELD score is used to estimate relative disease severity and the likely survival of patients awaiting LT. Computed tomography (CT) showed multiple bile lakes, complete PVT

(grade II according to the Yerdel classification [4]), and a large spontaneous SRS (Fig. 1). Doppler ultrasonography showed the absence of intrahepatic portal flow. At 17 months old, she underwent LDLT because of repetitive cholangitis.

The PV trunk had become phlebosclerotic and fragile owing to inflammation. The bowel did not become congested after clamping the PV, indicating sufficient communication between the portomesentric system and the SRS.

A left lateral liver graft from her father was used. He was 32 years old and ABO identical. The graft weight was 220 g, and the graft/recipient weight ratio was 2.6%. Because the graft's left hepatic vein was very short, an anterior wall patch of the graft's hepatic vein was made using a donor superficial femoral vein (SFV) in bench surgery. An SFV of 10 cm long was obtained (Fig. 2a), and the remainder of the anterior wall patch was reserved for use as an interpositional graft if the recipient's PV was insufficient for anastomosis. Although the native PV from the bifurcation of the splenic vein to the cut end was phlebosclerotic, some blood flowed from the cut end after removing the thrombus. Therefore, the graft's left PV was anastomosed to the recipient's PV. However, Doppler ultrasonography showed the absence of intrahepatic portal flow after portal reperfusion.

The duodenum was mobilized using the Kocher maneuver. The infrahepatic vena cava and the left renal vein were exposed. After the left renal vein was encircled and mobilized, it was clamped (Fig. 2b). Because intrahepatic and extrahepatic portal flow was still absent after clamping the left renal vein, we considered that PV venoplasty or a mesenteric jump graft with ligation of the SRS was more likely to be unsuccessful. Therefore, RPA was performed. The left renal vein was divided very close to the vena cava. The donor SFV was anastomosed to the proximal renal vein as an interpositional graft (Fig. 2c). Then, the first PV anastomosis was divided, and the donor's left PV was anastomosed to the interpositional graft (Fig. 2d, e). After these procedures, the PV flow was sufficient upon reperfusion, which was determined by Doppler ultrasonography. The PV pressure was 20 mm Hg after arterial reperfusion. Ligation of the splenic artery reduced the PV pressure to 17 mm Hg. The surgical time, cold ischemia time, and warm ischemia time were 891, 124, and 51 min, respectively. The estimated blood loss during surgery was 1,158 mL.

The patient's posttransplant course is shown in Figure 3. CT at postoperative day (POD) 14 confirmed the RPA was patent, without stenosis or thrombosis. She developed severe acute cellular rejection. Her transaminase levels were elevated to 323 IU/L for aspartate aminotransferase and 255 IU/L for alanine transaminase on POD 16. A liver biopsy revealed severe acute rejection, with a rejection activity index score of 8 (P3, B2, V3). It was successfully treated by 2 courses of methylprednisolone pulse therapy followed by intravenous methylprednisolone therapy and mycophenolate mofetil. The patient's ascites increased and became chylous after resuming milk intake; the maximum output was 700 mL on POD 6. The ascites decreased after the administration of medium-chain triglyceride milk, and disappeared after the treatment of acute cellular rejection. A third liver biopsy revealed late-onset hyperbilirubinemia (maximum total bilirubin of 6.8 mg/dL), which was due to acute cellular rejection. Her liver function gradually improved and she was discharged at POD 73. There were no signs of renal dysfunction. The patient's condition was good at her last follow-up, 22 months after transplantation.

Ogasawara et al.: Renoportal Anastomosis in Left Lateral Lobe Living Donor Liver Transplantation: A Pediatric Case

Discussion

It is thought that RPA in adult patients with grade 4 PVT or a very small phlebosclerotic PV decreases the complexity of PV reconstruction and provides adequate portal perfusion [1]. It has also been reported that RPA is useful in adult-to-adult LDLT [5, 6]. However, only 3 cases of RPA have been reported in pediatric LDLT. Two recipients were much older than our patient, and either an extended left lobe graft or a left lobe graft was used in those patients [2, 7]. One recipient received a left lateral lobe graft, but he was much older than our patient [8]. To our knowledge, our patient was the youngest case of RPA in pediatric LDLT with a left lateral lobe graft.

PV sclerosis and PVT are frequently seen in patients with biliary atresia because of repeated cholangitis [9]. Surgical techniques, such as PV venoplasty, interpositional graft, or mesenteric jump graft using a donor vein graft, allowed surgeons to perform pediatric LDLT in a patient with phlebosclerotic and thrombosed PV [2]. Huge collateral vessels, such as the left gastric vein, may be used instead [10].

The presence of large collateral vessels, such as a large SRS, may cause steal phenomenon, which can lead to graft atrophy and graft failure after transplantation. The SRS or the distal left renal vein could be ligated in order to disconnect the SRS [11]. In our patient, simple end-to-end PV anastomosis after removing the PVT was unsuccessful. Clamping the distal left renal vein failed to restore portal flow. Clamping the left renal vein had virtually the same effect as clamping the SRS because she had no collateral shunt vessels but had one large SRS. We considered that venoplasty or an interpositional graft would have been unsuccessful because the entire native PV was phlebosclerotic. A mesenteric jump graft with ligation of the SRS or distal left renal vein is another option to provide adequate portal perfusion. However, it is extremely difficult to use a mesenteric jump graft, especially in pediatric patients who are susceptible to PVT or PV stenosis [12]. We thought that it would have been too risky to perform a time-consuming and difficult technique in an emergency situation, such as failure of an initial PV reconstruction. Therefore, RPA was performed instead because it is much easier to perform. In our patient, it took 28 min to perform RPA without using a microscope or loupe. Additionally, there were no PV complications, such as obstruction, stenosis, or thrombosis.

Despite the advantage of RPA, it is thought that it may not fully relieve portal hypertension. Quintini et al. [1] reported that some patients with RPA needed proximal splenic artery embolization to modulate liver inflow and to overcome ascites caused by unrelieved portal hypertension. Matsumoto et al. [6] described RPA in a patient with transient posttransplant ascites and hyperbilirubinemia, consistent with graft overperfusion syndrome, also known as small-for-size syndrome. In adult LDLT, it is important to perform portal pressure control, mainly via splenectomy or splenic artery ligation, to prevent small-for-size syndrome. Some groups have reported that the acceptable range of portal pressure is 15–20 mm Hg [13]. Small-for-size syndrome is rare in pediatric LDLT because of the small body size, and the liver graft weight of 220 g and graft/recipient weight ratio of 2.6% seemed to be sufficient in our patient. However, the portal pressure after arterial reperfusion was 20 mm Hg. This is probably because the addition of left renal venous return into the graft via the RPA caused excessive inflow. Therefore, the splenic artery was ligated, which decreased the portal pressure to 17 mm Hg. Adjustment of the portal pressure prevented graft overperfusion syn-

drome. Our patient's ascites was not due to graft overperfusion, because her ascites was chylous and decreased after the administration of medium-chain triglyceride milk. Splenectomy would have disconnected the SRS, and RPA with splenectomy is partly contraindicated. Therefore, we considered that ligation of the splenic artery was a good strategy in LDLT with RPA. Moreover, splenectomy would have increased the risk of infection in pediatric patients [14]. To our knowledge, our patient was the first reported case of RPA with splenic artery ligation. Another concern of RPA is that it may affect renal function by causing left kidney congestion [6]. In our patient, there were no signs of renal dysfunction. Modulation of portal pressure might prevent renal dysfunction arising from left kidney congestion.

In prior reports, the recipient's internal jugular vein or external iliac vein, or an external stented polytetrafluoroethylene graft were used as venous grafts for RPA in LDLT [5, 6]. We used recipient SFV grafts for middle hepatic vein reconstruction or an interpositional graft [15]. Severe complications following dissection of the SFV, such as prolonged edema or foot numbness, have never been observed. Therefore, we used the donor's SFV as the venous graft for RPA. The diameter and length of the SFV were ideal for the interposition of a venous conduit between the recipient's left renal vein and the donor's PV.

In conclusion, this is the youngest case of RPA in pediatric left lateral lobe LDLT. Additionally, this is the first case of RPA with splenic artery ligation and using the donor's SFV as the venous graft for RPA. Although long-term follow-up is necessary, RPA for infants with a phlebosclerotic small PV and a large SRS could be a salvage option if other methods are unsuccessful.

Statement of Ethics

The authors have no ethical conflicts to disclose.

Disclosure Statement

The authors have no conflicts of interest to disclose.

References

1 Quintini C, Spaggiari M, Hashimoto K, et al: Safety and effectiveness of renoportal bypass in patients with complete portal vein thrombosis: an analysis of 10 patients. Liver Transpl 2015;21:344–352.
2 Matsuura T, Yanagi Y, Saeki I, et al: Outcome of modified portal vein anastomosis for recipients with portal vein thrombosis or stenosis before living donor liver transplantation. J Pediatr Surg 2011;46:2291–2295.
3 Sheil AG, Stephen MS, Chui AK, et al: A liver transplantation technique in a patient with a thrombosed portal vein and a functioning renal-lieno shunt. Clin Transplant 1997;11:71–73.
4 Yerdel MA, Gunson B, Mirza D, et al: Portal vein thrombosis in adults undergoing liver transplantation: risk factors, screening, management, and outcome. Transplantation 2000;69:1873–1881.
5 Moon DB, Lee SG, Ahn CS, et al: Technical modification of reno-portal anastomosis in living donor liver transplantation for patients with obliterated portal vein and large spontaneous splenorenal shunts. Hepatogastroenterology 2008;55:2193–2199.

Ogasawara et al.: Renoportal Anastomosis in Left Lateral Lobe Living Donor Liver Transplantation: A Pediatric Case

6   Matsumoto Y, Ikegami T, Morita K, et al: Renoportal anastomosis in right lobe living donor liver transplantation: report of a case. Surg Today 2013;43:1316–1320.
7   Uchida H, Sakamoto S, Shigeta T, et al: Living donor liver transplantation with renoportal anastomosis for a patient with congenital absence of the portal vein. Case Rep Surg 2012;2012:670289.
8   Neto JS, Fonseca EA, Candido HL, et al: Alternatives for vascular reconstruction in pediatric living donor liver transplantation. Pediatr Transpl 2016;20:717–722.
9   Saad S, Tanaka K, Inomata Y, et al: Portal vein reconstruction in pediatric liver transplantation from living donors. Ann Surg 1998;227:275–281.
10 Kitajima T, Sakamoto S, Uchida H, et al: Living donor liver transplantation with alternative porto-left gastric vein anastomosis in patients with post-Kasai extrahepatic portal vein obstruction. Pediatr Transpl 2013;17:E100–E103.
11 Castillo-Suescun F, Oniscu GC, Hidalgo E: Hemodynamic consequences of spontaneous splenorenal shunts in deceased donor liver transplantation. Liver Transpl 2011;17:891–895.
12 Gurevich M, Guy-Viterbo V, Janssen M, et al: Living donor liver transplantation in children: surgical and immunological results in 250 recipients at Université Catholique de Louvain. Ann Surg 2015;262:1141–1149.
13 Hori T, Ogura Y, Ogawa K, et al: How transplant surgeons can overcome the inevitable insufficiency of allograft size during adult living-donor liver transplantation: strategy for donor safety with a smaller-size graft and excellent recipient results. Clin Transplant 2012;26:E324–E334.
14 Haller JA Jr, Jones EL: Effect of splenectomy on immunity and resistance to major infections in early childhood: clinical and experimental study. Ann Surg 1966;163:902–908.
15 Sato K, Sekiguchi S, Watanabe T, et al: The use of recipient superficial femoral vein as a venous graft for portal vein reconstruction in right lobe living donor liver transplantation. Transplant Proc 2009;41:195–197.
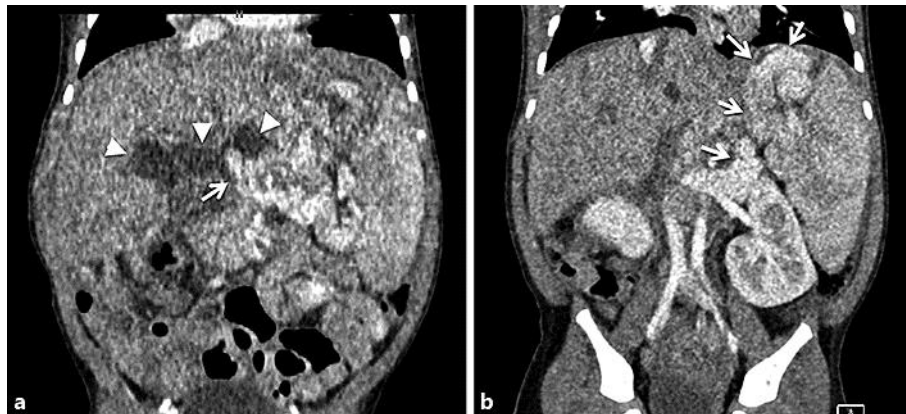
Fig. 1. Pretransplant computed tomography images. **a** Image shows multiple bile lakes (arrowheads) and stenosis of the portal vein (arrow), which disappears into the liver. **b** Image shows a large, spontaneous splenorenal shunt (arrows).
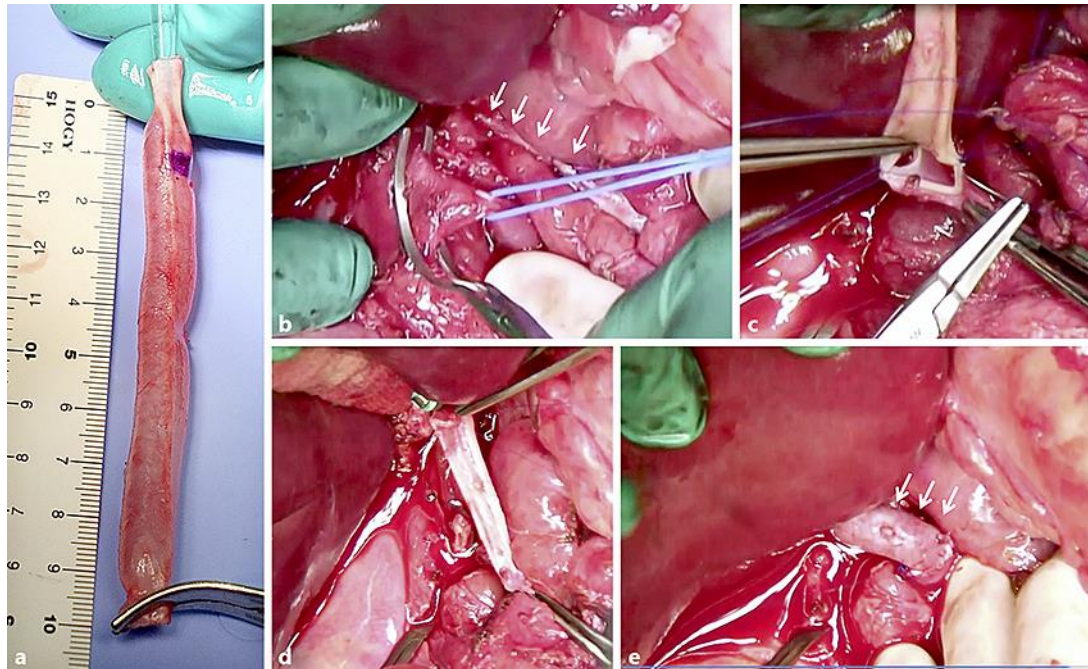
Case Reports in
Gastroenterology

Case Rep Gastroenterol 2017;11:584–592

590

| DOI: 10.1159/000481160 | © 2017 The Author(s). Published by S. Karger AG, Basel www.karger.com/crg |

Ogasawara et al.: Renoportal Anastomosis in Left Lateral Lobe Living Donor Liver Transplantation: A Pediatric Case

Fig. 2. Intraoperative findings. **a** The donor's superficial femoral vein (SFV) was used as a venous graft. It was about 10 cm long. **b** The left renal vein was clamped very close to the vena cava. The extrahepatic portal vein did not dilate (arrows), and portal flow was absent after clamping the left renal vein. **c** The SFV was anastomosed to the distal left renal vein as an interpositional graft. **d** The graft's left portal vein was anastomosed to the donor's SFV. **e** The SFV graft after reperfusion of the anastomosis (arrows).
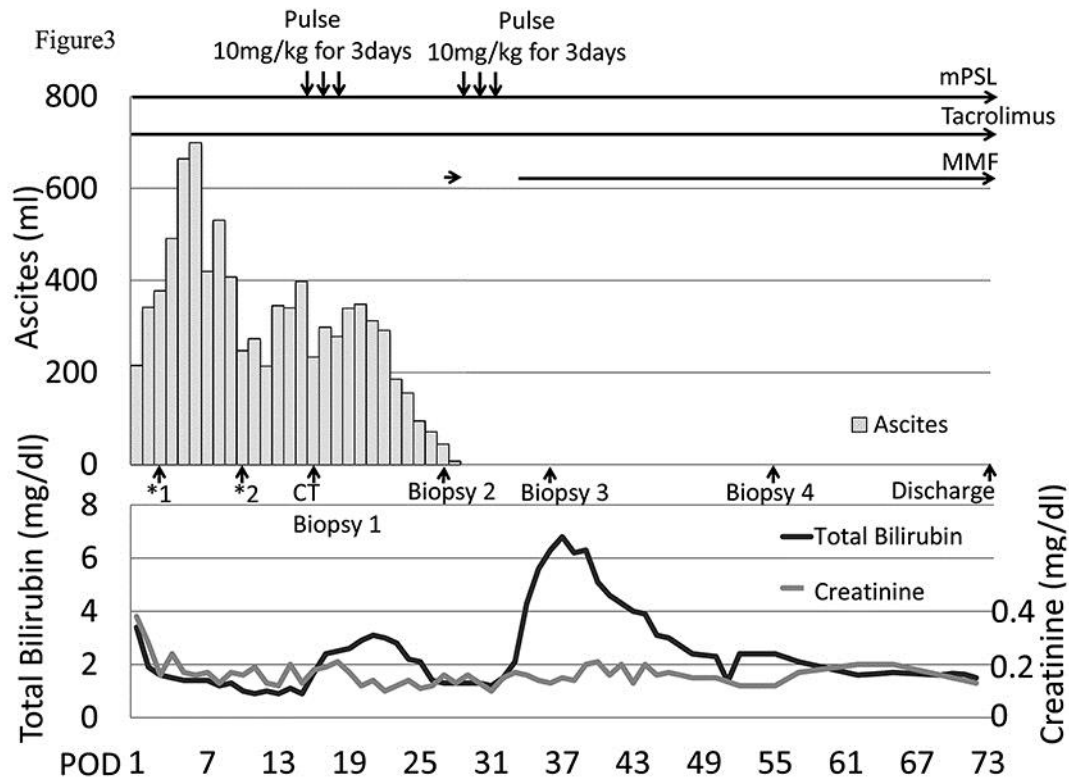
591

Ogasawara et al.: Renoportal Anastomosis in Left Lateral Lobe Living Donor Liver Transplantation: A Pediatric Case

Fig. 3. Posttransplant course. Ascites increased and became chylous after the start of milk intake (*1). The ascites decreased after the switch from milk to medium-chain triglyceride milk (*2). Biopsy 1 revealed severe acute cellular rejection (ACR). The rejection activity index (RAI) was 8 (P3, B2, V3). Biopsy 2 revealed an improvement of ACR and the RAI decreased to 3 (P1, B1, V1). Biopsy 3 showed further improvement in ACR and the RAI decreased to 2 (P0, B1, V1). Biopsy 3 also showed that hyperbilirubinemia was due to liver parenchymal damage, which was caused by ACR. Biopsy 4 revealed that ACR and parenchymal damage had disappeared. CT, computed tomography; mPSL, methylprednisolone; MMF, mycophenolate mofetil.

592

Ogasawara et al.: Renoportal Anastomosis in Left Lateral Lobe Living Donor Liver
Transplantation: A Pediatric Case

Table 1. Laboratory data on admission

	Measured value	Reference value
White blood cell count, /μL	9,000	4,000–9,000
Red blood cell count, ×10^4/μL	340	376–500
Hemoglobin, g/dL	9.5	12.0–16.0
Hematocrit, %	29.9	33.5–45.0
Platelet count, ×10^4/μL	28.6	15.0–35.0
Total protein, g/dL	6.1	6.7–8.1
Albumin, g/dL	2.9	3.8–5.3
Total bilirubin, mg/dL	1.4	0.2–1.0
Aspartate aminotransferase, IU/L	90	8–38
Alanine transaminase, IU/L	50	4–43
Alkaline phosphatase, IU/L	2,591	115–359
γ-Glutamyl transpeptidase, IU/L	463	10–47
Cholinesterase, IU/L	152	217–491
Blood urea nitrogen, mg/dL	5	8.0–20.0
Creatinine, mg/dL	0.13	0.32–0.84
Sodium, mEq/L	136	136–145
Potassium, mEq/L	4.2	3.5–5.1
Chloride, mEq/L	103	98–107
Prothrombin time, %	102.3	≥70.1
Active partial thromboplastin time, s	41.9	29.6–40.8
International normalized ratio	0.99	≤1.15
Ammonia, μg/dL	85	12–66