

Chylous Ascites After Liver Transplantation: Incidence and Risk Factors

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In this study, we evaluated the diagnosis, epidemiology, risk factors, and treatment of chylous ascites developing after liver transplantation (LT). Between 2002 and 2011, LT was performed 693 times in 631 patients at our clinic. One-hundred fifteen of these patients were excluded for reasons such as retransplantation, early postoperative mortality, and insufficient data. Chylous ascites developed after LT (mean \pm SD = 8.0 \pm 3.2 days, range = 5-17 days) in 24 of the 516 patients included in this study. Using univariate and multivariate analyses, we examined whether the following were risk factors for developing chylous ascites: age, sex, body mass index, graft-to-recipient weight ratio, Model for End-Stage Liver Disease score, vena cava cross-clamping time, total operation time, Child-Pugh classification, sodium level, portal vein thrombosis or ascites before transplantation, donor type, albumin level, and perihepatic dissection technique [LigaSure vessel sealing system (LVSS) versus conventional suture ligation]. According to a univariate analysis, a low albumin level ($P = 0.04$), the presence of ascites before transplantation ($P = 0.03$), and the use of LVSS for perihepatic dissection ($P < 0.01$) were risk factors for developing chylous ascites. According to a multivariate Cox proportional hazards model, the presence of pretransplant ascites [$P = 0.04$, hazard ratio (HR) = 2.8, 95% confidence interval (CI) = 1.1-13.5] and the use of LVSS for perihepatic dissection ($P = 0.01$, HR = 5.4, 95% CI = 1.5-34.4) were independent risk factors. In conclusion, the presence of preoperative ascites and the use of LVSS for perihepatic dissection are independent risk factors for the formation of chylous ascites. To our knowledge, this study is the most extensive examination of the development of chylous ascites. Nevertheless, our results should be supported by new prospective trials. *Liver Transpl* 18:1046-1052, 2012. © 2012 AASLD.

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Chylous ascites is defined as the accumulation of a milky or creamy, triglyceride-rich peritoneal fluid due to the presence of intestinal lymph in the abdominal cavity.¹⁻⁴ The pathogenesis of chylous ascites formation is related to a failure of the lymphatic system, which can occur after trauma or because of an obstruction or congenital factors. The most common causes of chylous ascites are congenital lymphatic

abnormalities, various infections, malignant neoplasms, blunt abdominal trauma, cirrhosis, and abdominal surgeries such as aortic aneurysm repair, inferior vena cava resection, distal splenorenal shunting, and liver transplantation (LT).^{1,3,4-7} Chylous ascites after LT is an extremely rare complication, and there have been only a few reported cases.^{6,8} Two mechanisms, separately or in combination, may

Abbreviations: CI, confidence interval; CSL, conventional suture ligation; DDLT, deceased donor liver transplantation; HR, hazard ratio; LDLT, living donor liver transplantation; LT, liver transplantation; LVSS, LigaSure vessel sealing system; TPN, total parenteral nutrition.

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affect the formation of chylous ascites after transplantation. One is an increase in lymph production in the hepatic and splanchnic areas secondary to cirrhosis. The excess lymphatic fluid in the intestinal lymphatic drainage system enters the abdominal cavity directly. The second mechanism involves an injury to the lymphatic system in the periportal and retrohepatic areas during hepatic resection and inadequate ligation of injured lymphatic vessels.^{6,9,10} The drainage of chylous ascites is observed shortly after abdominal surgery and usually immediately after the initiation of oral nutrition.^{6,9} The diagnosis is based on the presence of milky and creamy ascites with a triglyceride ratio > 1.0, a cholesterol ratio < 1.0, and a leukocyte count ≥ 300 cells/mm³ and/or a predominance of lymphocytes. The optimal management of chylous ascites is controversial. Conservative treatment, which is recommended for most patients, involves paracentesis, a diet based on medium-chain triglycerides, total parenteral nutrition (TPN), and somatostatin. A surgical approach is recommended only when conservative treatment fails.^{2,7} In this article, we explore the risk factors for developing chylous ascites after LT.

PATIENTS AND METHODS

Study Design and Background

Between March 2002 and December 2011, LT was performed 693 times in 631 patients at the Liver Transplantation Center of Inonu University. Demographic, clinical, pathological, radiological, and biochemical data for the patients were assessed retrospectively. Three factors were deemed to be exclusion criteria capable of altering the purpose and homogeneity of this study:

1. The first 75 LT procedures performed at our clinic were excluded from the study because their data were not sufficiently clear for comparison with the other cases.
2. Thirty-nine retransplants performed in 35 patients were excluded because different dissection methods were used during retransplantation.
3. Sixty-three patients who died within the first 4 days after LT for other reasons were excluded because chylous ascites typically forms 4 to 5 days after transplantation.

As a result, 516 patients who underwent LT met the inclusion criteria. We compared the demographic and clinical factors influencing the development of chylous ascites in 24 patients in whom chylous ascites formed postoperatively and in 492 patients who did not develop chylous ascites. The primary objective of this study was to identify the risk factors for the formation of chylous ascites after LT. The secondary objective was to share our experience with treating patients with chylous ascites.

Surgical Techniques

All transplantation procedures included in this study were performed by the same experienced surgical

TABLE 1. Basic Characteristics of Chylous Ascites

Color	Milky and creamy
Triglyceride ratio	>1.0
Cholesterol ratio	<1.0
Leukocyte count	≥ 300 cells/mm ³ and/or a predominance of lymphocytes
Culture	Mostly negative
Cytology	Mostly negative

team. Most of the methods that we used for both living donor liver transplantation (LDLT) and deceased donor liver transplantation (DDLT) are comparable to those in the literature. We used total vena cava clamping in the majority of our patients before liver implantation, and we dissected the pericaval and aortocaval windows to facilitate clamping. We think that the sealing techniques used in the dissection of this region are important to the development of chylous ascites because of the lymphatic drainage system of the abdominal region. When we started performing transplantation, we used electrocoagulation and the conventional suture ligation (CSL) technique to dissect the periportal, retrohepatic, and pericaval regions; for the last 3 years, however, we have been using the LigaSure vessel sealing system (LVSS; Valleylab, Boulder, CO) to dissect these regions. In almost all DDLT cases, a cavocaval anastomosis was made with the piggyback technique. Some modifications were made on the back table to enable a large hepatic vein anastomosis in LDLT. For this purpose, we formed a funnel-shaped model with a wide orifice by wrapping saphenous or iliac venous grafts preserved with a cryopreservation technique around the orifice of the graft hepatic vein. Because of this method, we have almost never experienced problems with hepatic drainage at our clinic.

Definition of Chylous Ascites

Chylous ascites was suspected because of milky or creamy peritoneal fluid drainage that began after oral intake or was found via ultrasonography for abdominal distension after drain removal, and it was diagnosed on the basis of the triglyceride, cholesterol, leukocyte, and lymphocyte contents of the liquid. In the laboratory diagnosis of chylous ascites, a drained liquid/serum triglyceride ratio > 1.0 and a cholesterol ratio < 1.0 were considered important⁵ (Table 1 and Fig. 1).

Conservative Management of Chylous Ascites

The initial conservative treatment consisted of the cessation of the oral diet, TPN (≤ 30 kcal/kg/day), and a crystalloid supplement for ascites drainage with or without somatostatin. Somatostatin (250 μ g/hour for adult patients and 125 μ g/hour for pediatric patients) was given until the drainage turned serous. If the amount and quality of the drainage did not change after the oral diet was resumed, the drains were removed. If there was any recurrence, the same

treatment was given. In some cases, emergency laparotomy, paracentesis, or catheter drainage was the initial treatment for liquid drainage.

Immunosuppressive Treatment Models

All LT patients were given 2 doses of corticosteroids during surgery in the hepatic and nonhepatic phases. For the first 6 months after the operation, steroids were given in amounts recommended for adults and children. Additional immunosuppressive therapy was begun when increases in liver function test results



Figure 1. Appearance of chylous ascites.

were observed after approximately 2 to 3 days. For adults, tacrolimus and mycophenolate were added to the steroid therapy, whereas children were given cyclosporine in addition to steroids. All cases with a histopathological diagnosis of hepatocellular carcinoma were given routine low-dose sirolimus therapy, which began when the creatinine level exceeded 1.5 mg/dL. Cases with an increased creatinine level during tacrolimus treatment were switched to sirolimus.

Statistical Analysis

Statistical analyses were performed with SPSS 13.0 (SPSS, Chicago, IL). Data are presented as means and standard deviations for continuous variables and as frequencies for categorical variables. The statistical significance of differences between groups was examined with univariate analyses, and variables with a significant association ($P < 0.05$) were then subjected to multivariate analyses with a Cox proportional hazards model. All statistical tests were 2-sided with a significance level of 0.05.

RESULTS

Comparison of Patients With Chylous Ascites and Patients Without Chylous Ascites

Data for 516 patients, including 353 males and 163 females (age = 40.4 ± 17.3 years, range = 1-72

TABLE 2. Comparison of Variables for Patients With Chylous Ascites and Patients Without Chylous Ascites According to a Univariate Analysis

Characteristic	Postoperative Chylous Ascites		P Value
	Present (n = 24)	Absent (n = 492)	
Age (years)*	41.2 ± 12.5 (15-62)	40.3 ± 17.4 (1-72)	0.8
Sex (n)			
Male	17	336	0.8
Female	7	156	
Body mass index (kg/m ²) [†]	24.7 ± 1.1	24.6 ± 0.2	0.9
Graft-to-recipient weight ratio [†]	1.27 ± 0.09	1.30 ± 0.03	0.9
Model for End-Stage Liver Disease score [†]	20.3 ± 2.2	18.4 ± 0.4	0.4
Child-Pugh score [†]	9.8 ± 0.4	9.5 ± 0.1	0.6
Albumin level (g/dL) [†]	2.7 ± 0.1	3.0 ± 0.1	0.04
Sodium level (mEq/L) [†]	133.8 ± 1.1	135 ± 0.3	0.3
Pretransplant ascites (n)			
Present	20	307	0.03
Absent	4	185	
Pretransplant portal vein thrombosis (n)			
Present	1	26	0.8
Absent	23	466	
Transplant type (n)			
LDLT	20	413	0.9
DDLT	4	79	
Perihepatic dissection (n)			
LVSS	21	291	0.01
CSL	3	201	

NOTE: Bolded values are significant.

*The data are presented as means and standard deviations (with ranges in parentheses).

[†]The data are presented as means and standard deviations.

TABLE 3. Determination of Independent Risk Factors With a Multivariate Cox Proportional Hazards Analysis

Parameter	HR	95% CI	P Value
Albumin level (g/dL)	0.7	0.4-1.3	0.3
Preoperative ascites: present versus absent	2.8	1.1-13.5	0.04
Perihepatic dissection: LVSS versus CSL	5.4	1.5-34.4	0.01

years), were examined retrospectively for a comparison of patients with chylous ascites (n = 24) and patients without chylous ascites (n = 492). In univariate analyses, both groups were similar with respect to the following: age, sex, body mass index, graft weight, graft-to-recipient weight ratio, Model for End-Stage Liver Disease score, vena cava cross-clamping time (minutes), total operation time for the recipient hepatectomy and implantation process (hours), Child-Pugh classification, total bilirubin level, blood sodium level, presence of pretransplant portal vein thrombosis, and donation type (living versus deceased). However, a low preoperative albumin level ($P = 0.04$), the presence of pretransplant ascites ($P = 0.03$), and the use of LVSS for perihepatic dissection ($P < 0.01$) were noted to be risk factors for the formation of chylous ascites. The Cox proportional hazards model revealed that the presence of pretransplant ascites [$P = 0.04$, hazard ratio (HR) = 2.8, 95% confidence interval (CI) = 1.1-13.5] and the use of LVSS for perihepatic dissection ($P = 0.01$, HR = 5.4, 95% CI = 1.5-34.4) were independent risk factors. The results of the statistical analysis are summarized in Tables 2 and 3.

Clinical Characteristics of Patients With Chylous Ascites

Chylous ascites formed after transplantation (mean \pm SD = 8.0 ± 3.2 days; range = 5-17 days) in 24 patients, including 17 males and 7 females (age range = 15-62 years). Twenty of these patients underwent LDLT, and 4 underwent DDLT. The diagnosis of chylous ascites was confirmed with the criteria listed in Table 1. The mean values for the triglyceride ratio, cholesterol ratio, ascitic fluid leukocyte count, and ascitic fluid lymphocytes were 1.8 ± 1.16 (range = 1.1-7.23), 0.2 ± 0.09 (range = 0.05-0.55), 494 ± 210.6 cells/mm³ (range = 80-850 cells/mm³), and 86% \pm 9.3% (range = 53%-95%), respectively. Routine cultures revealed no bacterial overgrowth. At the time of the diagnosis, 22 patients were using steroids and tacrolimus as their immunosuppressive treatment, whereas 2 were using steroids and cyclosporine. The patient with a diagnosis of hepatocellular carcinoma was switched to sirolimus therapy. In 8 patients, abdominal distension after drain removal was the initial sign of the formation of chylous ascites. Ultrasonography revealed free liquid in the abdomen and percutaneous drainage of a whitish liquid. One adult patient

TABLE 4. Clinical and Demographic Characteristics of 24 Patients With Chylous Ascites

Characteristic	Value
Original disease (n)	
Hepatitis B virus	14
Hepatitis B virus and hepatitis D virus	4
Hepatitis C virus	2
Budd-Chiari syndrome	1
Hepatocellular carcinoma	1
Toxicity	1
Cryptogenic	1
Hospital stay (days)*	42.5 ± 30.8 (10-135)
Triglyceride ratio*	1.8 ± 1.16 (1.1-7.23)
Cholesterol ratio*	0.2 ± 0.09 (0.05-0.55)
Onset of chylous ascites after transplantation (days)*	8.0 ± 3.2 (5-17)
Somatosatin infusion (days)*	10.0 ± 3.3 (4-16)
Daily amount of drainage (mL)*	5509 ± 3179 (1200-11,200)
Ascitic fluid leukocyte count (cells/mm ³)*	494 ± 210.6 (80-850)
Ascitic fluid lymphocytes (%)*	86 ± 9.3 (53-95)
Coexisting complications (n)	
Biloma	3
Chylothorax	1
Preoperative ascitic fluid in patients with chylous ascites (n)	
Moderate	16
Massive	4

*The data are presented as means and standard deviations (with ranges in parentheses).

and 1 pediatric patient underwent emergency laparotomy on postoperative days 15 and 28, respectively, because percutaneous catheters could not be placed. Conservative management was the initial treatment in all patients, except for the 2 patients in whom percutaneous drains could not be placed. Oral intake was reinitiated upon the conversion of chylous characteristics to serous characteristics and drainage of <100 mL/day. After 5 days of follow-up with no significant complications, the drains were removed, and the patients were considered cured.

Three patients had bilomas on cut surfaces as coexisting complications, and these were treated with percutaneous drainage. Chylous ascites recurred in 5 patients and was treated in the same conservative manner. One patient had dyspnea on postoperative day 20 because of a right pleural effusion and was treated with a chest tube and closed underwater drainage. Because the liquid had chylous characteristics, the patient was diagnosed with coexisting chylous ascites and chylothorax. This patient healed with no further complications. Five patients died for reasons unrelated to the formation of chylous

ascites: 1 patient with a small-for-size liver had late sepsis, 1 patient had a primary marginal nonfunctioning liver from a cadaveric donor, 1 patient had a hepatic vein obstruction with late sepsis, 1 patient had *Acinetobacter* sepsis, and 1 patient had multibacterial sepsis. Data on the patients with chylous ascites are summarized in Table 4.

DISCUSSION

Chylous ascites, an uncommon disease caused by an obstruction, rupture, or unsealing of lymph nodes or lymphatic ducts, is defined as the accumulation of chyle in the peritoneal or retroperitoneal cavities.⁷ The causes of chylous ascites can be classified as spontaneous and traumatic or congenital and acquired.^{1,4} Although the most common causes of chylous ascites in children are congenital lymphangiectasia and lymphatic hypoplasia, the most common causes in adults are malignant diseases, abdominal or thoracic surgery, abdominal trauma, infectious diseases, and liver cirrhosis. Two-thirds of the cases in Western societies occur with liver cirrhosis and malignancies, whereas infectious diseases give rise to the majority of the cases in developing countries.

The incidence of chylous ascites after abdominal surgery ranges from 0.17% to 1.1%. After retroperitoneal, esophageal, gastric, or cytoreductive surgery, the postoperative incidence is even higher (7.4%).^{7,11,12} The reported incidence of chylous ascites after LT varies from 0.6% to 1.6%.^{6,9} We had an incidence of 4.7% in our series.

Although ascites is a common feature of liver cirrhosis, chylous ascites is found in only 0.5% to 1% of patients with cirrhosis.¹² This discrepancy might be related to the fact that not all patients with ascites are treated by paracentesis, so chylous ascites remains undetected. The pathophysiological mechanism of chylous ascites in patients with liver cirrhosis is thought to be increased intra-abdominal pressure combined with degenerative changes in the splanchnic lymph vessels, and this leads to the intra-abdominal leakage of lymph fluid.¹² In our series, pretransplant ascites was detected in 327 of 516 cases, and only 0.061% had postoperative chylous ascites. This number is higher than reported values. Moreover, 83.3% of the cases with chylous ascites had preoperative ascites. These results indicate that the presence of pretransplant ascites is an independent risk factor for the development of chylous ascites.

The formation of chylous ascites after LT is relatively rare. There are several mechanisms that may explain its occurrence. First, there is increased lymphatic fluid production in the liver and splanchnic areas in patients with liver cirrhosis. This increase generally exceeds the capacity of the intestinal lymphatic drainage system.^{6,9,10,13} Second, the retrohepatic lymphatic vessels and lymphatic vessels in the porta hepatis are damaged during resection of the native liver.^{6,9} The final mechanism is the presence of pretransplant ascites.

Lymphatic channel injury during the dissection of the pericaval, periportal, and aortocaval windows and the resulting formation of chylous ascites are important issues for transplant surgeons because the fistulization of postoperative chylous fluid into the abdominal cavity due to inadequate or careless ligation (sealing) of lymphatic channels during the dissection of this region is unavoidable. This stresses the importance of ligation methods. Just as for other abdominal procedures, the main sealing techniques used for LT are CSL, electrocoagulation, hemostatic clipping, bipolar diathermy, and electrothermal bipolar vessel sealing.

LVSS was designed as an alternative to clips or ligatures for sealing vessels ≤ 7 mm in diameter. Comparative studies have proven that LVSS is as safe, feasible, and beneficial as other vessel closure tools and techniques, including the plasma trisector, the surgical clip, the harmonic scalpel, and conventional hemostasis.¹⁴⁻¹⁶ An increasing number of recent reports have described the use of LVSS not only for the sealing of vessels but also for the dissection/ transection of various soft and parenchymal tissues (eg, liver resection, pancreatectomy, pulmonary resection, and hysterectomy). Technological advances have reduced operating times and bleeding and have improved overall surgeon and patient comfort. At our clinic, LVSS has been used routinely for recipient hepatectomy since 2009.

Considering our results, we have determined that although LVSS reduces the duration of the dissection, its use is a risk factor for the development of chylous ascites. Therefore, we believe that the use of the suture ligation technique instead of LVSS is appropriate in areas with a high lymphatic system density during surgery with high mortality and morbidity rates (eg, LT). However, the retrospective design of our study limits our ability to make stronger recommendations. Further randomized prospective trials comparing the use of LVSS to the use of CSL in patients for whom orthotopic LT is planned are necessary.

Small-for-size grafts are associated with persistent elevations of the portal vein pressure after LDLT, and portal hypertension is one of the causes of chylous ascites.⁶ Although there was no significant relationship between the graft weight and the graft-to-recipient weight ratio in our case series, 1 small-for-size case developed chylous ascites.

Literature searches of the PubMed, Google Scholar, and MEDLINE databases with the keywords *chylous ascites* and *liver transplantation* in different combinations revealed only 6 case reports of chylous ascites after LT.^{6,8-10,17,18} None of these case reports adequately described the mechanism of formation or management strategies. Hence, the diagnostic criteria, epidemiology, onset features, and treatment of chylous ascites after LT remain unclear. To our knowledge, ours is the first study of the risk factors for chylous ascites after LT.

The diagnosis of chylous ascites is confirmed by an analysis of the ascitic fluid, which is possible only if

the diagnosis is suspected. The chief characteristics of chylous effusions include a milky appearance, the separation of a creamy layer on standing, a lack of an odor, an alkaline nature, a specific gravity > 1.012 , bacteriostatic properties, a total protein concentration of 3%, the staining of fat globules with Sudan red, and a fat concentration of approximately 0.4% to 4%. The triglyceride level is an important diagnostic tool, and the concentration in chylous ascites is typically 2 to 8 times the concentration in plasma. Other tests with the highest yields of diagnostic information are computed tomography, lymphangiography, lymphoscintigraphy, laparoscopy, and laparotomy.^{1,4}

The optimal approach to patients with postoperative chylous ascites remains controversial. Various treatments to decrease the rate of chyle formation, including repeated therapeutic paracentesis or drainage, diuretics, somatostatin, a low-fat diet with high-protein supplementation, medium-chain triglycerides, and TPN, have been described.^{2-4,7,8,19} Paracentesis and drainage not only are diagnostic but also are therapeutic in the management of chylous ascites. Despite several complications, repeated paracentesis or catheter drainage is commonly included in conservative treatment regimens to relieve abdominal distention. An enteral dietary intervention to decrease lymph flow in the major lymphatic tracts and to facilitate the closure of chylous fistulas involves a diet that is rich in protein and low in fat and medium-chain triglycerides.^{1,7,20} TPN can be used to achieve complete bowel rest and might allow the resolution of chylous ascites. Successful treatment with octreotide or somatostatin has been reported.^{3,8,21} The exact mechanisms underlying the actions of somatostatin are not understood, although it has been shown to decrease the intestinal absorption of fats, lower triglyceride concentrations in the thoracic duct, and attenuate lymph flow in the major channels. It also decreases gastrointestinal secretions and motor activity in the gastrointestinal tract.^{3,9,22}

Fasting combined with TPN can decrease lymph flow in the thoracic duct dramatically from 220 to 1 mL/kg/hour.^{2,7,23} In addition, TPN restores nutritional deficits and balances the metabolic impairments imposed by longstanding chylous ascites and repeated paracentesis. Therefore, fasting and TPN are essential in the conservative management of chylous ascites⁷; in combination with somatostatin, these should constitute the first-line therapy for chylous ascites caused by various disorders and should be started as soon as possible.^{7,9} Surgical treatment may be considered for cases resistant to conservative therapy for more than 1 month or for patients who show a temporary initial response with decreased ascites but ultimately are not cured by more than 2 months of conservative therapy.^{2-4,9,17,24}

In conclusion, the presence of pretransplant ascites and the use of LVSS for dissection are independent risk factors for the formation of chylous ascites. Therefore, it is important to ligate the lymphatic vessel network with great care while the periportal, retro-

hepatic, and pericaval regions are being dissected. The sequence of nonoperative approaches in cases with chylous ascites depends on the experience of individual clinics. We use TPN and fasting combined with somatostatin in cases with chylous ascites after LT. We adjust the duration of the treatment according to each patient's clinical status and the characteristics of the chylous drainage.

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