Diagnosis, Outcome, and Management of Chylous Ascites Following Pediatric Liver Transplantation

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Data on postoperative chylous ascites (CA) after pediatric liver transplantation (LT) are scarce. This retrospective study was conducted to identify the incidence, risk factors, management, and outcomes of postoperative CA in a large single-center pediatric LT cohort (2000-2016). The study cohort comprised 317 LTs (153 living donors and 164 deceased donors) in 310 recipients with a median age of 2.7 years. The incidence of CA was 5.4% (n = 17), diagnosed after a median time of 10 days after LT. Compared with chylomicron detection in peritoneal fluid (the gold standard), a triglyceride cutoff value of 187 mg/dL in peritoneal fluid showed insufficient sensitivity (31%) for CA diagnosis. In univariate logistic regression analyses, ascites before LT, younger age, and lower weight, height, and height-for-age *z* score at LT were associated with CA. Symptomatic management of CA included peritoneal drain (100%) and diuretics (76%). Therapeutic interventions included very low-fat or medium-chain triglyceride–rich diets (94%) and intravenous octreotide (6%), leading to CA resolution in all patients. CA was associated with prolonged hospital length of stay (LOS; 40 days in the CA group versus 24 days in the non-CA group; $P = 0.001$) but not with reduced patient or graft survival rates after a median follow-up time of 14 years. In conclusion, CA in the pediatric LT recipient is a relatively uncommon complication associated with increased hospital LOS and morbidity. Measurement of chylomicrons is recommended in patients with ascites that is more severe or persistent than expected. Dietary interventions are effective in most patients.

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Liver transplantation (LT) is an established curative treatment for children suffering from end-stage liver disease, liver malignancies, and selected metabolic disorders. Prevention of postoperative morbidity and early directed therapy of immediate posttransplant complications improves patient outcomes and survival. Postoperative chylous ascites (CA), defined as the accumulation of lipid-rich lymph (chyle) in the peritoneal cavity, has rarely been studied in pediatric LT recipients.[1-3] Continuous leakage of chyle into the peritoneal cavity can lead to the loss of lymphocytes, essential proteins, lipids, vitamins, electrolytes, and water.[4] The resultant clinical consequences beyond the physical accumulation of ascites include dehydration, electrolyte imbalance, malnutrition, lymphopenia, and increased susceptibility to infection.[4,5]

The diagnosis of postoperative CA in the pediatric LT recipient can be challenging. First, its clinical presentation may mimic other posttransplant complications, such as hepatic arterial and portal venous

Abbreviations: A1AT, alpha-1-antitrypsin deficiency; CA, chylous ascites; CI, confidence interval; CMV, cytomegalovirus; EFA, essential fatty acid; IQR, interquartile range; LOS, length of stay; LT, liver transplantation; MCT, medium-chain triglyceride; NPO, nil per os; OR, odds ratio; PFIC, progressive familial intrahepatic cholestasis; POD, postoperative day; TPN, total parenteral nutrition; VAC, vacuum-assisted closure.

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abnormalities, hepatic venous outflow obstruction, small-for-size syndrome, and abdominal infection.[3,6] Second, diagnostic criteria for CA are not well established. Detection of chylomicrons by lipoprotein electrophoresis in peritoneal fluid is considered the gold standard diagnostic test.[7] When not available, a triglyceride cutoff of 110 mg/dL (1.25 mmol/L) in peritoneal fluid was recommended in the pediatric LT literature.[8] The latter recommendations were not supported by high-quality evidence, and it is recognized that triglyceride quantification in peritoneal fluid may not be a reliable diagnostic criterion for CA in fasting patients, particularly in the postoperative state.[9] Recently, these gaps in the literature were addressed, and it is now accepted that a triglyceride cutoff value of 187 mg/dL (2.13 mmol/L) establishes the diagnosis of CA.[10]

The optimal management of postoperative CA is unclear. One study in pediatric LT recipients reported that dietary therapy is effective[11]; another advocated for the use of pharmacologic therapy as a first-line therapy in high-volume output chylous leakage (>20 mL/kg/day).[8] Our study aimed to determine the incidence, risk factors, treatment, and outcomes of postoperative CA in a large pediatric LT cohort using the detection of chylomicrons as the gold standard diagnostic test and the newly established triglyceride cutoff.[10]

Patients and Methods

STUDY POPULATION

The patient population in this case-control study included all children younger than 18 years old who underwent LT surgery at the Hospital for Sick Children, Toronto, Ontario, Canada, between January 2000 and December 2016. Infants and children receiving any organ in addition to liver, those transplanted at an outside institution, and those with CA diagnosed prior to LT were excluded. LT recipients experiencing graft loss or death within 30 days following LT were also excluded.

Data were retrieved from a prospectively populated electronic database and retrospective chart review. Data captured for all patients for risk analysis and outcome reporting included demographics; primary diagnosis; pretransplant comorbidities; surgical data; perioperative morbidity, such as vascular thrombosis and biliary complications; longterm outcomes, such as retransplantation; and death. Height and weight z scores were calculated using the World Health Organization growth standards.[12] Additional data were collected for all patients with CA and included information regarding diagnosis, management, and outcome of CA. All study patients followed institutional protocols for immunosuppression and received either standard induction with corticosteroids and a calcineurin inhibitor (tacrolimus) or a renal/neurotoxic-sparing protocol with corticosteroids and either antithymocyte globulin or 2 doses of basiliximab (Simulect; Novartis, Basel, Switzerland) with a delayed introduction of calcineurin inhibitor agents.[13] The study received approval from the institutional research ethics board at the Hospital for Sick Children.

DEFINITIONS AND OUTCOME MEASURES

Ascites prior to LT was defined as the presence of a moderate-to-large amount of peritoneal fluid on abdominal ultrasound or history of diuretic use for the treatment of ascites prior to LT. Posttransplant CA was defined as the presence of chylomicrons or a triglyceride value ≥187 mg/dL (≥2.13 mmol/L)[10] in the peritoneal fluid within 60 days from LT in the absence of a positive peritoneal fluid culture. Triglyceride values of ≥148 and <187 mg/dL (≥1.69 and <2.13 mmol/L) in the peritoneal fluid were considered equivocal for the diagnosis of CA.[10] Posttransplantation hospital length of stay (LOS) was defined as the interval between the day of transplantation and the day of first discharge from the hospital.

STATISTICAL ANALYSIS

Data were expressed as means and standard deviation or as median and interquartile range (IQR)

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when a nonnormal distribution of data was identified. Student *t* test was used for continuous variables. A nonparametric test (Mann-Whitney U test) was used for continuous variables when an abnormal distribution was identified. A chi-square test or Fisher's exact test was employed for categorical variables. The incidence of CA was calculated together with 95% confidence interval (CI) limits. Patient survival rates were estimated with the Kaplan-Meier method and compared with the log-rank test. Logistic regression univariate analyses were performed to explore the association between CA and variables with clinical significance. Statistical analyses were performed with SPSS, version 20.0 (SPSS Inc., Chicago, IL). A *P* value of <0.05 was considered statistically significant.

Results

PATIENT CHARACTERISTICS

The study cohort assembly is summarized in Fig. 1. The final study cohort comprised 317 isolated LTs performed in 310 pediatric patients with a median age at LT of 2.8 years (IQR, 0.8-10.3 years) and a median weight at LT of 13.8 kg (IQR, 8.0-29.8 kg). The median follow-up time of the whole cohort was 14.0 years (IQR, 11.0-15.0 years). There were 48.3% living donor and 51.7% deceased donor (24.6% whole liver, 14.2% reduced, and 12.9% split) grafts. Indications for LT included biliary atresia (38.2%), Alagille syndrome (4.7%), other cholestatic liver diseases (10.4%), metabolic diseases (17.4%), acute liver failure (14.5%), malignancy (7.3%), and other (7.6%).

INCIDENCE OF CA AND RISK ANALYSES

A total of 17 patients with postoperative CA were identified, giving an incidence of 5.4% (95% CI, 2.9%-7.9%). Table 1 summarizes the demographic and disease-specific data of the LT recipients with and without CA. Age, weight, and height at LT were significantly lower in LT recipients with CA compared with the non-CA group ($P < 0.005$). The groups were comparable with respect to sex, underlying diagnosis, and liver graft type. Pediatric LT recipients with CA had a significantly longer hospital LOS (CA group versus non-CA group; median [IQR], 40.0 [28.8-51.2] versus 24.0 [16.0-38.8] days; $P = 0.001$).

The incidence of postoperative hepatic artery thrombosis and portal vein thrombosis within the first 3 months after LT showed no statistical difference between the CA and the non-CA group. Two cases of bile leak were diagnosed and treated through laparotomy prior to CA diagnosis. Although a trend toward higher bile leaks was noted in the CA group, this did not reach statistical significance (CA group versus non-CA group; 11.8% versus 3.3%; $P = 0.20$). The incidence of CA per each of the 5 surgeons operating during the study period was similar ($P = 0.55$).

The 1-, 3-, and 5-year actuarial patient survival for patients without CA was 99%, 94%, and 93%, respectively, compared with 93%, 84%, and 84%, respectively, for patients with CA ($P = 0.26$; Fig. 2A). The 1-, 3-, and 5-year actuarial graft survival (Fig. 2B) for patients without CA was 98%, 93%, and 91%, respectively, compared with 93%, 84%, and 84%, respectively, for patients with CA ($P = 0.56$).

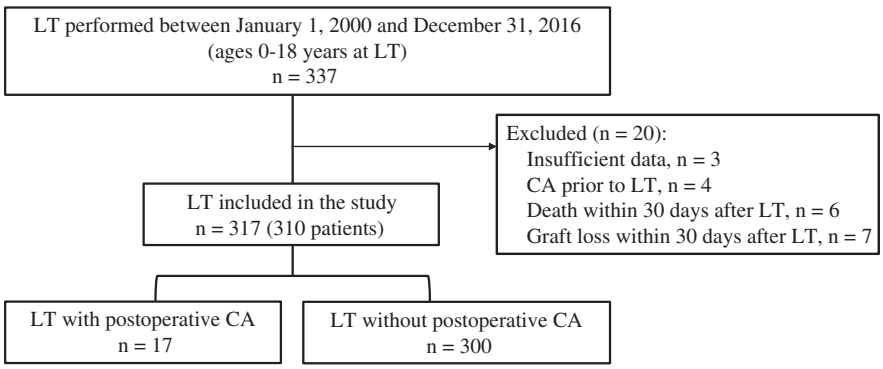

FIG. 1. A flowchart of the study cohort assembly.

TABLE 1. Patient Characteristics and Surgical Parameters of Patients With and Without Postoperative CA

	Postoperative CA (n = 17)	No Postoperative CA (n = 300)	*P* Value
Age at LT, years	0.8 (0.4-3.1)	2.9 (0.8-10.7)	0.002*
Sex, male	58.8	50.3	0.49
Underlying diagnosis			0.61
Biliary atresia	29.4	38.7	
Other cholestasis	23.5	14.7	
Acute liver failure	23.5	14.0	
Metabolic disease	17.6	17.3	
Other	6.0	15.3	
Weight at LT, kg	8.2 (6.0-10.4)	14.6 (8.2-31.9)	0.001*
Weight-for-age *z* score at LT	–1.4 ± 1.9	–0.6 ± 1.5	0.05
Height at LT, cm	66.6 (60.2-77.5)	91.0 (70.0-134.6)	0.001*
Height-for-age *z* score at LT	–1.7 ± 2.0	–0.7 ± 1.4	0.007*
Platelets, ×1000/mm^3	152.0 (59.5-190.5)	146.5 (81.2-242)	0.51
Ascites before LT	76.5	32.7	0.001*
Esophageal varices and/or variceal bleeding before LT	23.5	18.7	0.62
Abdominal surgery before LT	29.4	40.7	0.36
Re-LT	0	6.0	0.29
Graft type			0.21
Living donor	70.6	47.0	
Split	11.8	13.0	
Reduced	11.8	14.3	
Whole	5.8	25.7	
Aortic conduit reconstruction	0	7.0	0.26
Surgical drain in situ after LT	70.6	59.3	0.36
Abdominal wall closure			0.57
Closed	76.5	89.3	
Mesh placement	5.9	3.3	
VAC device	11.8	7.0	

NOTE: Data are shown as mean ± standard deviation, median (IQR), or %.
*Significant *P* values.

In univariate analyses, ascites before transplantation was identified as a significant risk factor for the development of CA after LT with an odds ratio (OR) of 6.69. Young age at transplantation (OR = 1.26 per year), lower weight at transplantation (OR = 1.10 per kg), lower height at transplantation (OR = 1.04 per cm), and lower height-for-age *z* score at LT (OR = 1.59; as a surrogate marker of nutritional status) were also associated with the development of CA (Table 2).

CHARACTERISTICS OF CA

Median time to diagnosis of CA was 10.0 days (IQR, 8.0-16.5 days) after LT surgery and 5.0 days (IQR, 3.0-12.0 days) after postoperative start of enteral feeding. There were 7/17 patients who did not have surgical drains in place at the time of diagnosis, and fluid testing results were obtained by paracentesis (n = 5) or a vacuum-assisted closure (VAC) device (n = 2). Also, 7 patients developed pleural effusion that required drainage, and 2 of the patients had chyle detected in pleural fluid. Table 3 provides details on the 17 patients who developed postoperative CA within 60 days after LT.

Diagnosis of CA was biochemically evident through chylomicron detection in 11 patients, triglycerides ≥187 mg/dL (≥2.13 mmol/L) in 1 patient not tested for chylomicrons, and both chylomicron detection and triglycerides ≥187 mg/dL (≥2.13 mmol/L) in the remaining 5 patients. Triglyceride levels in peritoneal fluid were measured in all 17 patients with CA. Triglyceride values were above the diagnostic cutoff value in 6/17 patients, within the equivocal range for CA diagnosis in 4/17 patients, and below the equivocal range in 7/17 patients. White cell count in peritoneal fluid was >500 cells/mm^3 in 10 of 14 cases analyzed. Median cell count was 740 cells/mm^3 (IQR, 396-1507 cells/mm^3), all with lymphocyte predominance (lymphocyte percentage >80%). All patients had negative peritoneal fluid cultures at the time of CA diagnosis.

MANAGEMENT AND OUTCOME OF CA

A peritoneal drain was used in all patients with CA for symptomatic management of ascites. Median duration of CA from diagnosis to the time of drain removal was 15.0 days (IQR, 11.5-22.5 days), with 2 patients draining for 30 days or more. Drain reinsertion after removal was not required in any of the patients. In total, 13/17 patients received diuretics.

Dietary modifications were used in 15/17 children and included a very low-fat diet for 2 patients who were able to take solid food by mouth, low-fat formula (Tolerex) in 1 patient, and medium-chain triglyceride (MCT)–rich formula (Portagen; Mead Johnson Nutrition, Evansville, USA) in 13 patients. Diet modifications were maintained for a median of 49.0 days (IQR, 27.0-72.0 days). Total parenteral nutrition (TPN) and nil per os (NPO)

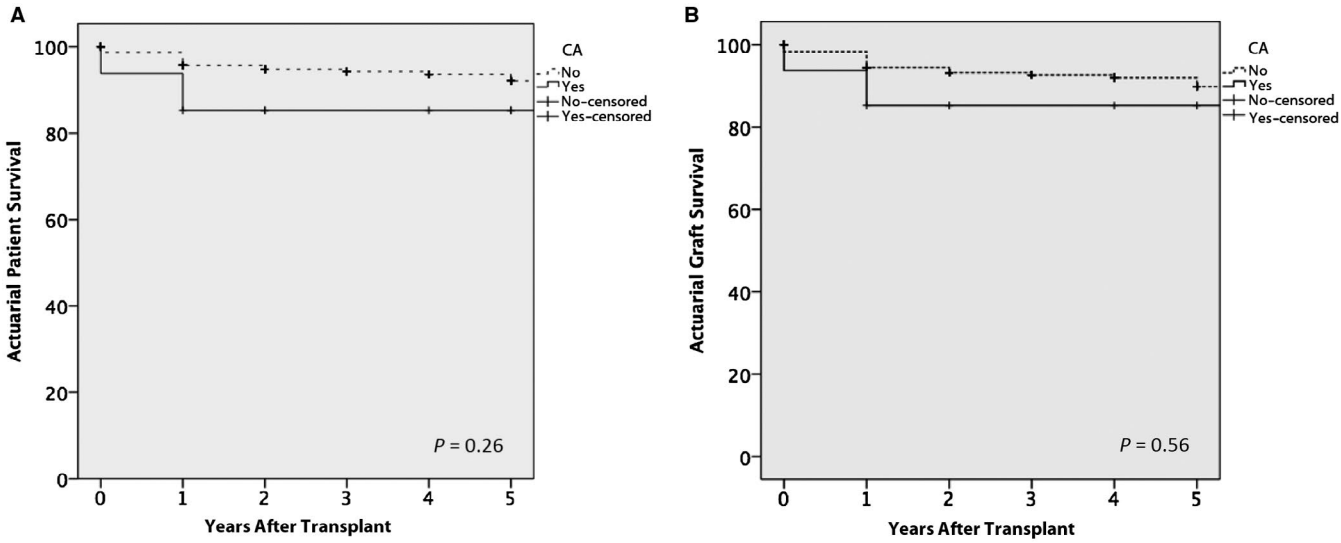

FIG. 2. The posttransplant survival outcomes. (A) Patient and (B) graft survival rates in patients with or without CA.

TABLE 2. Summary of Univariate Logistic Regression Models Evaluating the Effect of Independent Variables on the Development of CA

	OR	95% CI	*P* Value
Age (younger) at LT			
Overall (per year)	1.26	1.04-1.54	0.02*
≤1 year	2.90	1.07-7.84	0.04*
Weight (lower) at LT			
Overall (per kg)	1.10	1.01-1.19	0.02*
≤10 kg	6.04	1.92-18.97	0.002*
Weight-for-age *z* score (lower) at LT	1.38	0.99-1.91	0.05
Height (lower) at LT (per cm)	1.04	1.01-1.06	0.01*
Height-for-age *z* score (lower) at LT	1.59	1.13-2.24	0.01*
Platelet count (lower) at LT (per platelets x1000/mm^3)	1.00	1.00-1.01	0.37
Ascites before LT	6.69	2.12-21.07	0.001*
Surgical drain in situ after LT	1.63	0.56-4.74	0.37

*Significant *P* values.

was used in 4 patients for a median of 7.5 days (IQR, 4.5-9.8 days). Octreotide for 8 days at a maximum dose of 4 μg/kg/hour was added to the low-fat formula (Tolerex; Société des Produits Nestlé S.A., Vevey, Switzerland) in 1 patient for whom nutritional therapy failed to reduce chylous leakage volumes (Table 3).

Biopsy-proven liver allograft acute cellular rejection, defined as the rejection activity index according to the Banff schema ≥3 (range, 0-9), was diagnosed in 4/17 patients within the first 90 postoperative days (PODs). Severe lymphopenia (<0.5 × 10^9/L) was noted in 7/17 LT recipients with CA within 3 months after LT. Lymphopenia resolved in all patients except in 1 patient, for whom resolution could not be confirmed because of the transfer to another institution and death. There were 4/17 children with postoperative CA who received treatment for 5 documented infections following the diagnosis of CA and within 3 months after LT: cytomegalovirus (CMV) viremia (n = 1), urinary tract infection (*Enterococcus* spp.; n = 1), peritonitis while the peritoneal drain remained in situ (*Enterobacter* spp.; n = 1), upper respiratory tract infection (parainfluenza type 3; n = 1), and pneumonia (coronavirus; n = 1).

All children in the CA group except 1, who was transferred to another institution to receive palliative care, were discharged home after a median of 40.0 days (IQR, 28.8-51.2 days). Within 30 days of discharge, 2/16 patients were rehospitalized: 1 for pneumonia and 1 for biopsy-proven liver allograft acute cellular rejection. Also, 2 deaths occurred in the CA group due to posttransplant recurrence of hepatoblastoma and hemophagocytic lymphohistiocytosis at 4 and 6 months after LT, respectively.

Discussion

The findings of this large pediatric cohort reveal that postoperative CA is an uncommon complication in

TABLE 3. Clinical Course of Children With CA After LT

Demographics at LT				Diagnosis of CA			Management			Outcomes <3 Months After LT		
					Peritoneal Fluid Findings							
Patient Number	Sex	Age and Weight	Primary Diagnosis	Time of Diagnosis After LT, days	Chylomicron Detection	Triglyceride, mmol/L	Peritoneal Drain Duration, days	Treatment	Diuretics	Postoperative Complications	Infection	LOS, days
1	Male	0.2 years 6.4 kg	Neonatal liver failure	12	+	7.73	20	Portagen, 11 days	+	Bile leak and relaparotomy POD 4		33
2	Female	3 years 23.2 kg	Hepatoblastoma	8	Not tested	3.34	13	NPO/TPN, 4 days	+			*
3	Male	7 years 27.4 kg	Acute liver failure	15	+	1.79	16	NPO/TPN, 2 days Low-fat diet, 27 days	+	Bile leak and relaparotomy POD 4		46
4	Female	1 year 8.9 kg	Biliary atresia	19	+	2.77	13	Portagen, 12 days	–			31
5	Male	0.7 years 6.5 kg	Acute liver failure	10	+	2.14	5	Portagen, 90 days	–			123
6	Female	0.4 years 6.2 kg	Biliary atresia	7	+	0.86	15		+			28
7	Male	3 years 11.8 kg	Alagille syndrome	13	+	0.90	16	Portagen, 29 days	–			25
8	Male	0.2 years 4.5 kg	Neonatal liver failure	21	+	1.08	25	Portagen, 121 days	+		CMV viremia and urinary tract infection (*Enterococcus* spp.)	53
9	Male	1 year 8.2 kg	PFIC 1	18	+	1.10	35	Portagen, 61 days	+		Peritonitis (*Enterobacter* spp.)	87
10	Male	0.3 years 5.5 kg	Biliary atresia	8	+	1.40	15	Portagen, 86 days	+		Upper respiratory tract infection (parainfluenza type 3)	27
11	Female	0.4 years 5.8 kg	Biliary atresia	8	+	0.63	26	NPO/TPN, 9 days Portagen, 13 days	+			43
12	Male	0.8 years 8.4 kg	Biliary atresia	7	+	2.02	3	NPO/TPN, 6 days Portagen, 40 days	–			28

TABLE 3. *Continued*

Demographics at LT				Diagnosis of CA			Management			Outcomes <3 Months After LT		
					Peritoneal Fluid Findings							
Patient Number	Sex	Age and Weight	Primary Diagnosis	Time of Diagnosis After LT, days	Chylomicron Detection	Triglyceride, mmol/L	Peritoneal Drain Duration, days	Treatment	Diuretics	Postoperative Complications	Infection	LOS, days
13	Female	2 years 8.9 kg	PFIC 1	8	+	3.51	45	NPO/TPN, 10 days Portagen, 11 days Tolerex, 38 days Octreotide, 8 days (added to Tolerex)	+			63
14	Female	5 years 20.9 kg	A1AT	23	+	1.77	12	Low-fat diet, 56 days	+			37
15	Male	0.7 years 6.2 kg	A1AT	8	+	3.14	13	Portagen, 67 days	+			36
16	Female	0.2 years 4 kg	Neonatal liver failure	12	+	1.94	11	Portagen, 37 days	+		Viral pneumonia (coronavirus)	45
17	Male	0.9 years 8.5 kg	Alagille syndrome	8	+	1.42	7	Portagen, 72 days	+			46

*Patient 2 was transferred to another institution for palliative care after primary disease recurrence (death occurred 4 months after LT).

children after LT, leads to a prolonged hospital LOS and immediate postoperative morbidity, but does not impact longterm graft and patient survival. In all of our cases, early recognition of CA and symptomatic management of ascites using a peritoneal drain and dietary interventions (MCT-rich formula/very low-fat diet/NPO and TPN if there is no response to formula) led to the resolution of CA, with only 1 patient receiving pharmacologic therapy.

The incidence of CA in our study population (5.4%) is similar to values reported in other mixed pediatric and adult studies (6.3%) and adult studies (4.7%).[8,14] Yet, a recent study describing a series of 120 pediatric LTs reported a CA incidence of 24%.[3] The reason for the incidence discrepancy is unclear and probably relates to different practices around surgical drain insertion and peritoneal fluid testing. The rate of surgical drain insertion was lower in our study (60.1%) compared with that reported by Marseglia et al.[3] (87.5%). In addition, only 15% of our study patients were investigated for chylomicrons in the peritoneal fluid, raising the possibility that only symptomatic CA was diagnosed in our and other cohorts with similar incidence.

In our cohort, the development of ascites prior to transplantation, a sign of decompensated cirrhosis, was associated with a higher risk for the development of CA. Lymphangiogenesis has been described in the context of cirrhosis, and it has been speculated that it may help accommodate increased lymphatic flows and elevated portal pressures.[15,16] It is possible that the increased lymphatic circulation in patients with cirrhosis contributes to an increased risk of traumatic CA during LT; however, evidence supporting this hypothesis is lacking. Our findings are in keeping with those reported by Yilmaz et al.,[14] who also identified development of ascites prior to LT as an independent risk factor for CA. Nonetheless, a smaller study reported that children and young adult LT recipients with postoperative CA had similar rates of ascites on pretransplant radiological imaging as those without CA.[8] The main difference between this study and ours lies in the timing of the identification of ascites (anytime preoperatively in the latter), without being clear whether LT recipients in the first study had previously developed ascites.

Our analysis yielded additional risk factors for the development of postoperative CA, namely, age ≤1 year and weight ≤10 kg at time of transplantation. These findings may be related to the smaller caliber of vascular and lymphatic structures in younger children, putting small children at a greater risk for traumatic injury of the lymphatic vessels during surgery. Similarly, a lower height-for-age z score at LT, which generally implies failure to thrive and malnutrition, was also significantly associated with a higher risk of postoperative CA. These results support reports describing pretransplant malnutrition in children as a risk factor for posttransplant morbidity.[17] Given that the weight-for-age z score may underestimate the degree of undernutrition in children with chronic liver disease because of the effects of organomegaly and/or ascites, it is not surprising that no association was found between weight-for-age z score at LT and postoperative CA.

Concurrent with the initiation of enteral feeding, postoperative CA manifests primarily with persistent ascites or milky drainage through surgical drains in situ, often in the setting of otherwise adequate graft function. CA should be an integral part of the differential diagnosis of early ascites after transplant in addition to other common postoperative complications, such as hepatic arterial and portal venous abnormalities, hepatic venous outflow obstruction, small-for-size syndrome, and abdominal infection.[3,6] Suspicion of CA should prompt further investigation to confirm the diagnosis. Detection of chylomicrons in peritoneal fluid, obtained either through the peritoneal drain or through paracentesis, is considered the gold standard test for the diagnosis of CA.

Recently, a triglyceride cutoff of ≥187 mg/dL (≥2.13 mmol/L) in peritoneal fluid was suggested as an additional diagnostic tool for CA.[10] In our study, 16/17 and 30/300 LT recipients in the CA and non-CA groups, respectively, were investigated for both chylomicrons and triglycerides in the peritoneal fluid. We found the recently proposed triglyceride cutoff to be insufficiently sensitive (31.25%) as a diagnostic tool. These findings suggest that the triglyceride cutoff may not be a reliable criterion to exclude the diagnosis of CA in fasting or partially fed pediatric LT recipients. Maldonado et al. reported similar findings when determining the biochemical parameters of chylous pleural fluid in patients with chylothorax, particularly in those fasting in the postoperative state.[9] Peritoneal fluid should also be sent for cell count (lymphocyte predominance) and culture (negative).

There is a lack of consensus regarding the optimal management of postoperative CA, and treatment

options range from nutritional interventions to pharmacologic treatments and surgical procedures.[1,8,11,18-20] In contrast to what other groups have reported, in our series, we relied on symptomatic management of the ascites with diuretics and/or insertion of a peritoneal drain and nutritional interventions to manage CA.[1,8,18] Large peritoneal drain losses were generally replaced with 5% intravenous albumin (ratio of 1:1 or less), and drains were clamped when fluid output <5 mL/kg/day was reached. In our series, abdominal infection (bacterial peritonitis) occurred in only 1 patient and was easily managed.

Nutritional interventions were used in 16 patients and included dietary fat modifications in 11 children and dietary fat modifications plus a period of fasting and parenteral nutrition in 5 patients for whom dietary fat modification failed to diminish peritoneal drain losses. Patients following fat-free or MCT-rich diets as the only fat source for any length of time may need to supplement essential fatty acids (EFAs) and fat-soluble vitamins.[21] Therefore, given the potential for EFA deficiency in patients following dietary fat modifications, we implemented empiric supplementation with vegetable oil in 2015.[21] In contrast to recommendations from other groups, pharmacologic therapy with the somatostatin analogue octreotide was used in only 1 patient for whom nutritional strategies failed to reduce chylous leakage volumes. Considering that octreotide markedly reduces splanchnic blood flow and also has the potential to cause liver injury,[22] we believe nutritional interventions are a safer treatment option than octreotide.

The limitations and biases of this study relate to the retrospective nature of data collection, which can be incomplete in some respects, such as interpreting clinical judgment and the rationale for clinical care decisions. Given the low incidence of CA reported in our study, prospective controlled studies to determine efficacy of the therapeutic options for the management of CA would be difficult to conduct in children. Strengths of our study include the use of the gold standard diagnostic test (chylomicron detection) and a relatively large pediatric sample size, with a wide range of pediatric ages, underlying diagnoses, and surgical techniques.

In summary, we present a comprehensive analysis of the incidence, risk factors, outcomes, and management strategies of postoperative CA in the pediatric LT recipients. Although CA after LT is relatively uncommon, it was associated with prolonged hospital LOS and immediate postoperative morbidity. CA should be sought in children with increased peritoneal drain losses or ascites after LT, particularly in the younger population with a history of ascites and/or malnutrition before LT. Chylomicron detection in peritoneal fluid appears to be a more sensitive test that overran arbitrary triglyceride cutoff values in children undergoing LT. Symptomatic management of CA with peritoneal drains and dietary interventions are highly effective first-line treatment modalities in the majority of patients. Routine use of octreotide is not recommended or required. Early recognition and treatment of this unusual complication will shorten hospital LOS and reduce the degree of postoperative morbidity.

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