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Ascites after orthotopic liver transplantation in children

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Abstract: Ascites is a poorly understood postoperative complication of orthotopic liver transplantation (OLT). It is associated with additional morbidity and can prolong hospitalization considerably. The incidence, the factors predictive of occurrence and the etiology of this complication are not known. The charts of 118 patients with 138 OLT were analyzed according to the following criteria: ascites lasting longer than the first 10 postoperative days, assessed by loss of ascitic fluid through drainage tubes, surgical wounds or paracentesis, with a peak volume of ≥10 mL/kg/day. Patients were divided into three groups: Group 1, no ascites; Group 2, ascites associated with postoperative complications, including chylus ascites; and Group 3, ascites not associated with postoperative complications. Postoperative ascites occurred in 43 of 138 OLT (31.2%). Patients with biliary atresia, preoperative portal hypertension, postoperative pleural effusion or at retransplantation had ascites significantly more often. In 32 of 138 (23.2%) OLT, ascites was associated with postoperative complications, including thrombosis, abdominal infections, intestinal perforation, biliary leak, pancreatitis, and chylus ascites. In 11 of 138 (7.9%) OLT, ascites was the only postoperative complication (group 3). Group three patients were significantly older, and had lower preoperative platelet counts and preoperative ascites more often than group 1 patients. The primary liver diseases were mainly cystic fibrosis of the pancreas, congenital hepatic fibrosis, and North American Indian childhood cirrhosis. The serum-ascites albumin gradient suggested a hepatic origin of ascites. Postoperative ascites is associated with the duration and degree of preoperative portal hypertension. We speculate that the mechanism involved includes a disproportion between venous blood volume and liver uptake capacity of the donor organ.

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Key words: pediatric – portal hypertension – congenital liver disease – complications – albumin

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Ascites is a frequent but poorly understood complication that occurs during the early postoperative period of OLT. It usually disappears spontaneously after a few days, and its production is believed to depend on the presence and degree of the hyperdynamic circulation, sodium levels, malnutrition, portopulmonary hypertension or cardiac dysfunction (1, 2). Nevertheless, ascites production is associated with postoperative complications, such as thrombosis or stenosis at vascular anastomoses (3, 4) or abdominal infections. Moreover, some patients develop ascites of large volume and long duration for no apparent reason. The latter type of ascites prolongs hospitalization, increases the risk of complications, such as cardiac insufficiency, renal dysfunction, thrombotic incidents, and abdominal infections (5). Ascites has been shown to occur in 7% of adult patients after OLT (6).

The current study investigates the incidence and characteristics of ascites of long duration after OLT in pediatric patients and describes

Abbreviations: AIH, autoimmune hepatitis; ASAR, albumin serum-ascites ratio; CF, cystic fibrosis of the pancreas; CHF, congenital hepatic fibrosis; CI, confidence interval; CMV, cytomegalovirus; NAIC, North American Indian childhood cirrhosis; OLT, orthotopic liver transplantation; OR, odds ratio; PFIC, progressive familial intrahepatic cholestasis. SAAG, serum-ascites albumin gradient.

some typical characteristics of those at risk for ascites formation.

Patients and methods

We reviewed the records of patients undergoing OLT at Hôpital Sainte-Justine between 1986 and 2002. Totally, 131 patients had 152 OLT. After excluding those with early postoperative mortality (first 10 days, 13 deaths and 14 OLT), the charts of 118 patients with 138 OLT were analyzed according to the following criteria: ascites lasting longer than the first 10 postoperative days, assessed by loss of ascitic fluid through drainage tubes, surgical wounds or paracentesis, with a peak volume ≥10 mL/kg/day. Patients with ascites seen by echography only, but without drainage, were not considered. Abdominal Doppler echography was undertaken daily during the first 7 postoperative days, and after that for specific indications. Patients were divided into three groups: Group 1, no ascites, by the afore-mentioned definition; Group 2, ascites associated with postoperative complications, including chylus ascites; and Group 3, ascites not associated with postoperative complications.

The characteristics of ascites were evaluated by albumin concentration, lymphocyte count, SAAG (7) and ASAR (8). Routine patient evaluation before OLT consisted of assessment of immune status, of renal function by the Tc-99m-diethylenetriamine pentaacetic acid single injection technique, of cardiac function by cardiac Doppler echocardiography, and of nutritional and developmental status. Portal hypertension was defined as the presence of splenomegaly, small liver or hepatomegaly with or without ascites, and abnormal portal venous flow as detected by Doppler sonography, failure to thrive, and hypoalbuminemia with or without the presence of esophageal varices or ascites. Graft size was not assessed routinely during OLT.

The study was approved by the Research Ethics Committee of Hôpital Sainte-Justine. Based on the categorical or continuous nature of the variables, the likelihood ratio chi-squared test or Mann–Whitney *U*-test was used for preliminary group comparisons, with an alpha level set at 0.15. Conditional logistic regression was then applied to model factors potentially associated with ascites occurrence. Variables with $p < 0.15$ at univariate analysis were subjected to multivariate analysis by logistic regression.

Results

Group 1 consisted of 95 (69.8%) patients without ascites. In all, 43 of 138 (31.2%) OLT were complicated by ascites formation, of whom 32 (23.2%) developed simultaneous postoperative complications (group 2), while 11 (7.9%) had no other complication (group 3).

Primary diseases and ascites

The most frequent indications for OLT were nodular transformation of liver parenchyma in tyrosinemia, liver failure in biliary atresia, and retransplantation for chronic rejection or hepatic artery or portal vein thrombosis after OLT (Table 1). For statistical evaluation, the indications for OLT were combined in the following four groups: tyrosinemia, biliary atresia, retransplantation, and others. Biliary atresia was the disease most frequently associated with postoperative ascites of long duration (16 of 39 vs. four of 36 for tyrosinemia, four of 20 for retransplantation, and 19 of 53 for others, $p = 0.049$, chi-squared test).

Comparison of groups without (group 1) and with (groups 2 and 3) postoperative ascites of long duration

Table 2 reports the results of univariate analysis of factors associated with postoperative ascites production other than primary liver disease. No association with postoperative ascites was found for the following variables (not shown in Table 2): recipient or donor CMV and Epstein–Barr virus infection status, patient or donor blood groups or non-identical blood groups, donor sex, and age. The donor–recipient weight ratio was not predictive of ascites formation (reduced graft: $p = 0.89$; or cadaveric whole organ: $p = 0.11$).

Table 1. Ascites post-transplantation (in comparison with preoperative ascites) and indications for OLT

Indications for OLT	Group 1	Group 2	Group 3	Total (% postoperative ascites)
Tyrosinemia	22 (3)	4 (0)		26 (15.3)
Biliary atresia	23 (16)	14 (7)	2 (2)	39 (41)
Retransplantation	16 (1)	4 (0)		20 (20)
Fulminant hepatitis	6 (1)	1 (1)		7 (14.3)
PFIC	5 (1)	1 (1)		6 (16.7)
NAIC	5 (2)	0	3 (1)	8 (37.5)
Alagille syndrome	4 (1)	2 (0)		6 (33.3)
Glycogenosis type IV	3 (0)	0		3
CF	2 (0)	0	3 (2)	5 (60)
Hepatoblastoma	2 (0)	0		2
Hypercholesterinemia	1 (0)	0		1
Primary oxalosis	1 (0)	0		1
Wilson's disease	1 (1)	0		1
α1-AT deficiency	1 (1)	0		1
PSC	1 (1)	1 (1)		2 (50)
AIH	1 (0)	1 (0)	1 (1)	3 (66.7)
COACH syndrome	1 (0)	0		1
CHF	0	1 (1)	2 (1)	3 (100)
Langerhans cell histiocytosis	0	2 (1)		2 (100)
3β-HSD deficiency	0	1 (0)		1 (100)
Total	95	32	11	138 (45.3)

Group 1, no postoperative ascites (preoperative ascites); Group 2, postoperative ascites and complications (preoperative ascites); Group 3, postoperative ascites without complications (preoperative ascites).
OLT, orthotopic liver transplantation; PFIC, progressive familial intrahepatic cholestasis; NAIC, North American Indian childhood cirrhosis; CF, cystic fibrosis of the pancreas; α1-AT, α1-antitrypsine deficiency; PSC, primary sclerosing cholangitis; AIH, autoimmune hepatitis; COACH syndrome, hypoplasia of the carebellar vermis, oligophrenia, congenital ataxia, coloboma, and hepatic fibrosis; CHF, congenital hepatic fibrosis; 3β-HSD deficiency, 3β-hydroxy-delta-C27-steroid dehydrogenase/isomerase deficiency.

Table 2. Demographic statistics and comparison of patients listed per group with or without ascites (univariate analysis)

	Group 1 (n = 95; 68.8%)	Groups 2 and 3 (n = 43; 31.2%)	p-value
Median of quantitative variables (range)			
Pre-OLT			
Age at OLT (months)	35 (1–226)	46 (5–264)	0.447
Z-score for weight (22)	−1.03 (−5.7–3.7)	−0.75 (−6.9–2.5)	0.783
Z-score for height (22)	−1.13 (−6.55–2.97)	−0.96 (−6–2.38)	0.938
PELD	14 (−14–47)	13 (−9–49)	0.829
GFR (mL/min/1.73 m^2)	101 (27–241)	120.5 (45–324)	0.216
Platelets ($\times 10^9$/L)	141 (22–614)	152 (22–466)	0.288
At OLT			
Cold ischemia (min)	480 (120–945)	480 (46–1070)	0.784
Post-OLT			
Peak ALT U/L (days 1–3)	864 (116–12 600)	677 (159–5460)	0.189
Peak AST U/L (days 1–3)	1396 (203–37 800)	1065 (198–9490)	0.125
Serum creatinine* day 15	1n (1–3.2)	1n (1–2.1)	0.12
Qualitative variables, n (%)			
Pre-OLT			
Gender	44f/51m (f = 53.7%)	27f/16m (f = 62.8%)	0.06
Portal hypertension	61 (64%)	40 (93%)	**0.002**
Ascites	30 (31.6%)	20 (46%)	0.144
At OLT			
Full graft	47 (49.5%)	14 (32.1%)	0.07
Segments 2 + 3 or 2, 3 and 4	48 (51.5%)	29 (67.9%)	0.07
Portoportal anastomosis	91 (95.8%)	37 (86%)	**0.024**
Post-OLT			
Pleural effusion	29 (30%)	28 (65%)	**0.001**
Rejection	52 (54.7%)	26 (60.5%)	0.491
Retransplantation†	7 (6%)	11 (25.6%)	**0.04**

Bold values, significant difference.
Group 1, no ascites; Groups 2 and 3, ascites.
PELD, pediatric end-stage liver disease score; GFR, glomerular filtration rate; ALT, alanine aminotransferase; AST, aspartate aminotransferase.
*Serum creatinine shown as an upper limit of normal for age.
†Three patients died at retransplantation during the first 10 post-op days.

Graft arterial anastomoses were performed with the donor hepatic artery on 40 occasions in group 1, and 19 times in groups 2 and 3. They were produced with a jump-graft connected to the infrarenal aorta 18 and seven times, respectively, or with the donor hepatic artery connected directly to the host infrarenal or suprarenal aorta or celiac trunk 33 and 15 times, respectively. No difference was observed in ascites formation according to the type of arterial anastomosis ($p = 0.45$, chi-squared test). Direct portal vein anastomosis was created in 91 patients from group 1, and 37 patients from groups 2 and 3. Jump-graft interposition or anastomosis to the mesenteric vein was required in four and six patients, respectively. Complicated portal-venous anastomoses were more frequent in the group with ascites ($p = 0.024$, chi-squared test). Direct bile duct anastomoses were undertaken in 25 patients from group 1, and in 10 patients from groups 2 and 3 ($p = 0.68$); the remainder underwent choledocho-jejunal anastomoses. The piggy back approach to venous anastomoses was undertaken in 13 and four patients, respectively ($p = 0.79$, chi-squared test); the remainder had end-to-end cavo-caval implantation. Type of biliary or venous anastomosis was not associated with postoperative ascites.

Renal dysfunction (9) (serum creatinine above the upper limit for age) occurred in four patients with postoperative ascites, and in 10 patients without postoperative ascites [four of 43 (9.3%) vs. 10 of 95 (10.5%); $p = 0.82$).

Biopsy proven graft rejections occurred with equal frequency: 52 of 95 (54.7%, group 1) vs. 18 of 32 (56%, group 2), and five of 11 (45.5%, group 3) (p = ns). In group 2, 10 of 18 treatments were administered during, and eight after, the resolution of ascites. In six of the latter eight, treatment with OKT3 monoclonal antibody or antithymocyte globulin was required for corticosteroid-resistant rejection. In group 3, the five patients with rejection were treated successfully with high-dose solumedrol alone; two of them were treated when the ascites had already resolved, one without biopsy because of ascites, and two on postoperative days 5 and 8, respectively.

Multivariate analysis revealed the already noted association of preoperative portal hypertension (OR 6.21, 95% CI 1.58–24.42), postoperative pleural effusion (OR 5.56, 95% CI 2.3–13.4) and postoperative ascites, plus additional reoperation during the first 3 postoperative days (OR 5.43, 95% CI 1.13–26.44). Preoperative ascites (OR 1.12, 95% CI 0.46–2.73), and liver function tests (aspartate aminotransferase: OR 1.0, 95% CI 0.71–1.01, and alanine aminotransferase: OR 1.02, 95% CI 0.73–1.04) were not associated with postoperative ascites. Female gender (OR 2.0, 95% CI 0.97–4.3) was associated with a greater risk for postoperative ascites.

Comparison of patients with ascites of long duration associated (group 2) or not (group 3) with postoperative complications

The following complications were associated with ascites in group 2 patients: hepatic artery thrombosis and hepatic necrosis (n = 2), portal vein thrombosis (n = 7), biliary leak (n = 6), abdominal infection and/or intestinal perforation (n = 13), pancreatitis (n = 3), and chylus ascites (n = 1). Most complications occurred in transplanted or biliary atresia patients (n = 14, Table 1). Postoperative complications were

recorded in 45 of 95 (47%) patients of group 1 vs. 32 of 43 (74%) in group 2 ($p = 0.003$). Table 3 reports the results of univariate analysis of factors associated with postoperative ascites production in groups 2 and 3. The only variable with a statistically significant difference between the two groups with ascites was platelet count ($p = 0.044$). Median maximal volume per day was 1200 mL (360–4624 mL), and 3000 mL (640–6000 mL), respectively ($p = 0.016$). Renal dysfunction [serum creatinine above the upper limit for age (9)] was more frequent in group 3 than in group 2 (two of 11, 18.2% vs. two of 32, 6.25%). Logistic regression could not detect additional risk factors for ascites of long duration.

Comparison of patients with ascites not associated with postoperative complications (group 3, n = 11) and patients without postoperative ascites (group 1, n = 95)

Ascites production in group 2 may have been induced by variable mechanisms. To evaluate the difference between patients with ascites not associated with complications and those without postoperative ascites, we compared Groups 1 and 3, using the same variables as before. The differences remained the same except for: (i) patients from group 3 (108 months, range 6–210 months) who were significantly older than those from group 1 (35 months, range 1–226 months; $p = 0039$). In group 3, 10 of 11 patients had preoperative surgery for portal hypertension [shunt operation (three with CHF) or repeated sclerotherapy for gastrointestinal bleeding (three with CF, two with NAIC, two with biliary atresia, and one with AIH)]. Two CF patients had postoperative pulmonary hypertension and/or tricuspidal regurgitation. The remaining group 3 patients presented no signs of preexistent or persistent portopulmonary hypertension (10); (ii) preoperative ascites occurred significantly more frequently in group 3 (eight of 11, 73%) than in group 1 (42 of 95, 44%; $p = 0.023$); (iii) platelet count was significantly lower in group 3 (median 70×10^9/L, range 30–288 $\times 10^9$/L) than in group 1 (median 152×10^9/L, range 22–614 $\times 10^9$/L; $p = 0.023$). No difference was found for graft size (full graft in 47 of 96 vs. six of 11 patients; $p = 0.3$).

Table 3. Characteristics of patients with and without postoperative complications associated with ascites production (univariate analysis)

	Group 2 (n = 32; 23.2%)	Group 3 (n = 11; 7.9%)	p-value
Quantitative variables, median (range)			
Pre-OLT			
Age at OLT (months)	36 (5–264)	108 (6–210)	0.199
Z-score for weight	−0.76 (−6.97–1.89)	−0.68 (−3.8–2.5)	0.7
Z-score for height	−1.2 (−6.0–2.38)	−0.67 (−3.42–0.78)	0.3
PELD	13 (−9.0–43)	15 (−1–49)	0.42
GFR (mL/min/1.73 m^2)	117.5 (45–206)	125 (54–203)	0.2
Platelets ($\times 10^9$/L)	161 (22–466)	70 (30–288)	**0.044**
At OLT			
Cold ischemia (min)	510 (270–1070)	450 (295–750)	0.42
Post-OLT			
Peak ALT U/L (days 1–3)	654 (159–5460)	885 (168–5425)	0.37
Peak AST U/L (days 1–3)	1,112 (198–9399)	979 (367–9490)	0.63
Qualitative variables, n (%)			
Pre-OLT			
Gender	20f/12m (f = 62.5%)	7f/4m (f = 63.6%)	0.95
Portal hypertension	29 (90.6%)	11 (100%)	0.17
Ascites	12 (37.5%)	8 (72%)	**0.047**
At OLT			
Full graft	9 (28%)	6 (54.5%)	0.12
Segments 2 and 3 or 2, 3 and 4	23 (72%)	5 (45.5%)	0.12
Portoportal anastomosis	27 (84%)	10 (90.9%)	0.58
Post-OLT			
Pleural effusion	21 (65%)	10 (90.9%)	0.75
Rejection	21 (65.6%)	6 (54.5%)	0.24

Bold values, significant difference.
Group 2, ascites associated with complications; Group 3, ascites without complication.
PELD, pediatric end-stage liver disease score; GFR, glomerular filtration rate; ALT, alanine aminotransferase; AST, aspartate aminotransferase.

Characteristics of ascites

Median ascites duration was 26 days (interquartile range 17), median ascites volume per day was 603 mL (interquartile range 815.7 mL), and median ascites volume/kg body weight/day was 37 mL (interquartile range 46.5 mL). The ascites lymphocyte count in percent of total leukocytes was 88% (3–93) in group 2, and 88% (50–99) in group 3 ($p = 0.3$). The median ascites albumin concentration was found to be high in most patients: 22 g/L (range 13–43 g/L) in group 2, and 20 g/L (range 8–43 g/L) in group 3 ($p = 0.64$).

Median SAAG was 15 g/L (range 4–29 g/L, interquartile range 15 g/L) in group 3, and 14 g/L (range 3–32 g/L, interquartile range 8.5 g/L) in group 2 ($p = 0.636$). Six patients had SAAG > 20 g/L: three patients from group 3 with CHF, biliary atresia, NAIC, respectively, and three from group 2 with tyrosinemia, biliary leak and abdominal bleeding, PFIC, anasarca, abdominal bleeding and death on day 27, and AIH, renal insufficiency, CMV, and *Candida* infection, respectively.

Median ASAR was 0.55 (range 0.16–0.81) in group 3, and 0.56 (range 0.2–0.9) in group 2 ($p = 0.813$). Combined low ASAR, SAAG, and ascites albumin concentrations were seen in nine

patients with ascites in association with pancreatitis, intestinal perforation, and biliary leak, each in three patients.

Discussion

The current study reports an overall 31.2% incidence of ascites after liver transplantation, which is comparable with the French incidence rate of 25.2% (11). In adults, using the same definitions and inclusion criteria, the incidence of ascites post-OLT was found to be 7% (6). As described previously, this higher incidence of ascites in pediatric OLT may be related to a higher postoperative complication rate in young children with reduced liver grafts (12) and in reduced graft recipients in general (13–15). Patients with postoperative ascites have, therefore, been subdivided into those with ascites associated with postoperative complications and those with ascites not associated with postoperative complications. The incidence of ascites of long duration not associated with postoperative complications was 7.9%, which is comparable with that in adult patients with OLT and postoperative ascites not associated with further complications (6).

We first compared pre-, peri-, and postoperative variables in all patients with and without ascites after OLT. Multivariate analysis confirmed that patients with pretransplant portal hypertension and those with postoperative pleural effusion or complicated portal venous anastomoses developed ascites significantly more often. We found no difference in preoperative nutritional status, pediatric end-stage liver disease score or postoperative graft function between those with and those without postoperative ascites. There was equally no difference in graft size or the donor–recipient weight ratio, and we could not confirm the higher incidence of ascites in reduced graft recipients found by other groups (13–15).

To exclude an impact of post-transplant complications on the development of ascites, patients who incurred ascites with or without associated postoperative complications were compared with each other and with those without ascites. It appeared that patients with ascites not associated with complications were significantly older at OLT, had preoperative ascites more often, had lower preoperative platelet counts than those without ascites, and had all required sclerotherapy or shunt operation prior to OLT. While this suggests that the degree and duration of portal hypertension may play a role in postoperative ascites formation, with the exception of platelet count and ascites, no differences were found in pretransplant portal hypertension or its complications between patients with ascites-associated complications and those without complications or those without ascites. It is likely that the lack of differences between the two groups with ascites resulted from the small size and heterogeneity of the group with complications, and that complications in some patients may have masked the importance of duration and the degree of preoperative portal hypertension for ascites formation. SAAG, ASAR, and ascites protein concentration were found to be elevated in most patients with postoperative ascites. Because Doppler sonography could not detect any hepatic venous outflow or portal vein problems, this type of ascites may be a consequence of a persisting collateral circulation and large splanchnic blood volume (16, 17).

Recent studies in adult OLT patients have reported persistently elevated portal flow until 2 yr post-transplantation and persistent splenomegaly (2, 18). Some groups have already postulated that inadequate accommodation of liver blood flow in reduced liver grafts is the mechanism of ascites formation (15). In living-donor liver transplantation, elevated early postoperative portal venous pressure was correlated with small-for-size grafts and the incidence of ascites (19). Generally, a long duration and high degree of preoperative portal hypertension are accompanied by significant splenomegaly and the formation of an extensive collateral circulation. Although we did not measure spleen size or the degree of collaterals before OLT, the need for repeated sclerotherapy and/or shunt operation and low platelet counts are indicative of elevated pressure in the portal venous system, usually with splenomegaly. It can, therefore, be hypothesized that ascites of long duration after OLT is the expression of a functional sinusoidal block resulting from a disproportion between portal venous blood volume and liver uptake capacity in patients with persistent splenomegaly and an enduring collateral circulation. This hypothesis remains to be verified. Furthermore, it is not yet known if pharmacological reduction of portal venous pressure with β-blocking agents could shorten the duration of this type of postoperative ascites.

In summary, our study reveals that ascites post-liver transplantation is multifactorial. Patients with pathologies leading to long-lasting portal hypertension with upper gastrointestinal bleeding and hypersplenism develop ascites of

long duration more frequently after OLT. This ascites is time limited and reflects elevated splanchnic or hepatic venous pressures, may be of hepatic origin and may result from persistently increased hepatic portal venous inflow because of splenomegaly and venous collaterals. It prolongs hospitalization duration considerably, carries risks of infection, cardiac or renal problems, and requires repeated paracentesis. Better diagnostic and therapeutic approaches to this type of ascites are therefore required. In our study, the most valuable methods for diagnosis were the measurement of serum and ascites albumin concentration, and the most effective therapeutic tool was paracentesis. Pre- and postoperative echographic measurements of spleen size and routine monitoring of portal and hepatic venous velocity and volume by Doppler echography (20, 21), as well as preoperative evaluation of graft volume could facilitate the recognition of patients at risk for ascites of long duration. It is not known if these patients could profit from consequent preoperative and postoperative pharmalogical treatment of portal hypertension.

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